

# Saccadic Reaction Time in the Monkey: Advanced Preparation of Oculomotor Programs is Primarily Responsible for Express Saccade Occurrence

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## SUMMARY AND CONCLUSIONS

1. The introduction of a period of darkness between the disappearance of an initial fixation target and the appearance of a peripheral saccade target produces a general reduction in saccadic reaction time (SRT)—known as the *gap effect*—and often very short latency *express* saccades. To account for these phenomena, premotor processes may be facilitated by release of visual fixation and advanced preparation of saccadic programs. The experiments described in this paper were designed to test the relevance of the *ocular fixation disengagement* and *oculomotor preparation* hypotheses by identifying the influence of different factors on SRTs and the occurrence of express saccades in the monkey.

2. The SRTs of two monkeys were measured in two behavioral paradigms. A peripheral saccade target appeared at the time of disappearance of a central fixation target in the no-gap task, whereas a 200-ms period of no stimuli was interposed between the fixation target disappearance and the saccade target appearance in the gap task. The distribution of SRTs in these tasks was generally bimodal; the first and second mode was composed of express and regular saccades, respectively. We measured the mean SRT, mean regular saccade latency, mean express saccade latency, and percentage of express saccades in both tasks. We also estimated the gap effect, i.e., the difference between the SRTs in no-gap trial and the SRTs in gap trials.

3. Once the animals were trained to make saccades to a single target location and produce express saccades, SRTs in both no-gap and gap trials displayed a broad tuning with respect to the spatial location of the trained target when the target location was varied randomly in a block of trials. Express saccades were made only to a restricted region of the visual field surrounding the trained target location. A gap effect was present for nearly all target locations tested, irrespective of express saccade occurrence. Finally, the probability of generating an express saccade at the trained target location decreased with the introduction of uncertainty about target location.

4. The occurrence of express saccades increased with the duration of the visual and nonvisual (gap) fixation that the animal was required to maintain before the onset of a saccade target. The gap duration was effective in reducing the mean SRT for gaps  $\leq 300$  ms, and it was more influential than comparable variation in the visual fixation duration.

5. The occurrence of express saccades made to targets of identical eccentricity increased when the initial eye fixation position was shifted eccentric in a direction opposite to the saccade direction. Concomitantly, mean SRT decreased by  $\sim 2$  ms for each 1-deg change in initial eye fixation position.

6. The occurrence of express saccades depended upon contextual factors, i.e., on both the behavioral task (no-gap or gap) and the latency of the saccade that the monkey executed to the same

target in the preceding trial. The highest percentage of express saccades was observed after an express saccade in a no-gap trial, whereas the lowest percentage was obtained after a regular saccade in a gap trial.

7. These findings indicate that training-dependent express saccades are restricted to a specific spatial location dictated by the training target, and their incidence is facilitated by high predictability of target presentation, long-duration foreperiod, absence of visual fixation, eccentric initial eye position opposite to the saccade direction, and express saccade occurrence in the previous trial. The release of fixation afforded by the gap accounts for the general gap effect, but has only a modulatory influence on express saccade generation. We conclude that advanced motor preparation of saccadic programs generally reduces SRT and is primarily responsible for the occurrence of express saccades, which therefore may be caused mainly by neuronal changes restricted to a specific locus—coding for the trained movements—in a neural map of saccades.

## INTRODUCTION

The time a primate takes to initiate a saccadic eye movement in response to a visual stimulus depends upon a number of factors (see for review Becker 1989, 1991; Fischer and Weber 1993). Saccadic reaction time (SRT) experiments in the monkey have demonstrated that saccadic latency can form a bimodal distribution, with “express” and “regular” saccades composing the first and second mode, respectively (Fischer and Boch 1983; McPeck and Schiller 1994; Schiller et al. 1987; Sommer 1994). In both monkey and human subjects, express saccades have been elicited principally by using a behavioral paradigm in which a temporal “gap” is introduced between the disappearance of a central fixation point and the appearance of a peripheral saccade target: the gap paradigm. This paradigm produces, irrespective of the presence of express saccades, a reduction in SRT (the gap effect) with respect to other experimental paradigms lacking the gap period (Fendrich et al. 1991; Fischer 1987; Fischer and Boch 1983; Fischer and Ramsperger 1984; Iwasaki 1990; Kalesnykas and Hallet 1987; Kingstone and Klein 1993a,b; Mayfrank et al. 1986; Reulen 1984; Reuter-Lorenz et al. 1991; Rohrer and Sparks 1993; Ross and Ross 1980; Saslow 1967; Wenban-Smith and Findlay 1991). The major goal of the experiments described in this paper is to identify the influence of different factors on SRTs and the occurrence of express saccades in the monkey.

In contrast to the robust gap effect, express saccades are not necessarily generated in the gap paradigm, thereby sug-

gesting that these two phenomena are somewhat independent (Fischer et al. 1993; Kingstone and Klein 1993a; Wenban-Smith and Findlay 1991). Moreover, it has been reported that the occurrence of express saccades increases with training and that the training effect is spatially selective, i.e., once exposed to specific targets, monkeys make express saccades exclusively to these targets (Boch and Fischer 1986; Fischer et al. 1984). These observations imply that all oculomotor programs are facilitated by the release of visual fixation afforded by the disappearance of the fixation point and therefore produce the general reduction in SRT, but that only the set of oculomotor programs that has been "trained" undergoes potentiation and ultimately generates express saccades. The *disengagement of ocular fixation* hypothesis—the current foremost hypothesis—can account only for a nonspecific SRT reduction (Dorris and Munoz 1995; Fendrich et al. 1991; Kingstone and Klein 1993b; Klein 1993; Klein and Kingstone 1993; Klein et al. 1995; Munoz and Wurtz 1992, 1993b; Nozawa et al. 1995; Reuter-Lorenz et al. 1991; Sommer 1994; Tam and Ono 1994; Tam and Stelmach 1993). In contrast, a specific SRT reduction and express saccade generation may be explained by *oculomotor preparation* hypotheses that, to date, have been outlined only partially (Becker 1989; Kowler 1990; West and Harris 1993). The series of experiments described in this paper is designed to test the relevance of the ocular fixation disengagement and oculomotor preparation hypotheses on the occurrence of express saccades.

The first goal of the experiments consists of determining the extent of the spatial selectivity of express saccades directed to one trained target and examining how the occurrence of these express saccades is affected by training and the addition of other target locations randomized with the trained one (spatial uncertainty). The second goal is to evaluate whether the duration the animal fixates (visually or not) before target presentation influences SRTs and the occurrence of express saccades. A third goal consists of determining the effect of initial eye position on the distribution of SRTs, particularly on the occurrence of express saccades. The last aim of this paper is to investigate the relative influence of the preceding SRT performance and behavioral saccade task (gap or no-gap paradigm) on the SRT distribution and occurrence of express saccades.

In general, our data indicate that the motor command responsible for the saccade generation, not the release of ocular fixation alone, is modulated to permit the production of express saccades. This provides support for an oculomotor preparation hypothesis, in which topographically organized motor programs coding saccade metrics can be prepared partially before target onset.

A brief report of some of the results described in this paper has appeared in abstract form (Paré and Munoz 1995).

## METHODS

### *Animal preparation*

Two rhesus monkeys (*Macaca mulatta*) were trained to fixate and make saccades toward target light spots that were back-projected onto a tangent screen. Eye movements were monitored by the magnetic search coil technique (Fuchs and Robinson 1967), which had a resolution of 0.1 deg (CNC Engineering). Both ani-

mals were trained and used for data collection for this behavioral study and for neurophysiological experiments, which previously were undertaken in only one animal (*monkey 1*). Water intake was controlled with the animals receiving water reward during training and experiments. Fresh fruits were provided daily as well as ad libitum standard laboratory monkey chow. Animal weight, health status, and water intake were monitored closely under the supervision of University veterinarians. All animal care and experimental procedures were approved by the Queen's University Animal Care Committee and were in accordance with the Canadian Council on Animal Care policy on use of laboratory animals.

A single surgical procedure was carried out under aseptic conditions. Animals initially were given an injection of ketamine hydrochloride (10 mg/kg im) to provide restraint during the preparation of the surgical area and the insertion of an intravenous catheter. An injection of alphaxalone and alphadolone acetate (CT1341; Saffan, 0.5 ml/kg iv) then was given to provide relaxation during the insertion of a tracheal tube, and surgical levels of anesthesia subsequently were maintained using isoflurane (1–2%) inhaled through the endotracheal tube. Heart rate, respiratory rate, and body temperature were monitored. A preformed 19 mm diameter wire coil (3 turns; Cooner Wire) for the measurement of eye position was inserted subconjunctivally in one eye (Judge et al. 1980). The connector for the eye-coil leads and a stainless steel head-holding device to restrain the animal's head during the experiments were secured in place, embedded in a dental acrylic explant that was anchored firmly to the top of the exposed skull by several stainless steel bolts. To further stabilize the explant, veterinary super glue (Vetbond) was applied to the dry bare skull before the liquid dental acrylic. In both animals, the explant also included two stainless steel chambers for microelectrode recording experiments, which are not described here. At the end of the surgery, the animals received an intramuscular injection of antibiotics (penicillin) as a prophylactic measure against infection. These antibiotics were administered on a daily basis for 10 postoperative days. To alleviate any discomfort, animals also were given analgesic medication (buprenorphine hydrochloride 0.01 mg/kg; Flunixin Meglumine, Banamine 5 mg/kg) during the postsurgical period. Animals were given 1–2 wk for recovery before the start of behavioral training.

### *Behavioral paradigms and recording procedures*

Behavioral paradigms, visual displays, and data acquisition were controlled by a 80486 IBM-compatible computer running a UNIX-based real-time data acquisition system (REX) (Hays et al. 1982). Monkeys were seated in a primate chair (Crist Instruments) with their head restrained for the duration of the experiment. They faced a tangent screen positioned 86 cm in front of them and for which they had an unobstructed view of 70 × 70 deg (±35 deg in any direction from straight-ahead). Each behavioral trial was performed in total darkness and lasted ~2–3 s. The intertrial interval varied randomly between 1,000 and 1,500 ms. During the latter interval, the screen was illuminated diffusely (1.0 cd/m<sup>2</sup>); the light/dark cycle prevented the animal from becoming dark adapted. At the start of each trial, the background light was extinguished, and after a period of 250 ms, the task was initiated by the appearance on the screen of a target spot, referred to as the fixation point, which, after a delay period, was followed by a saccade target. The target spots, produced by light emitting diodes (LEDs, 2.0 cd/m<sup>2</sup>), were back projected onto the screen. The position of the visual targets was controlled by the computer via digital-to-analog converters controlling a x-y mirror galvanometer (General Scanning).

Animals were trained to perform two behavioral paradigms, the no-gap and gap task. In each task, only one visual target was ever present on the screen in a trial. However, the number of potential locations of the target in a given session was variable. In both

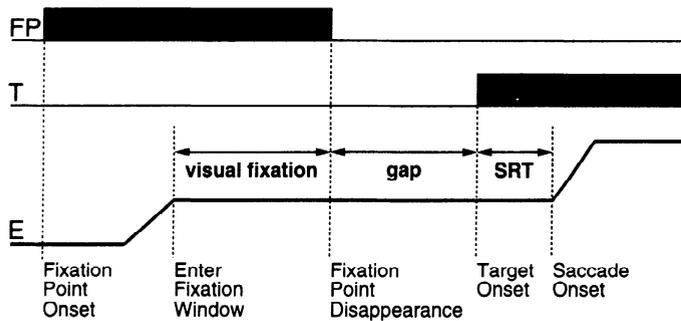


FIG. 1. Schematic representation of the behavioral paradigms used in this study on saccadic reaction time (SRT). All tasks began when monkey looked at fixation point (FP). Completion of tasks required execution of an eye (E) saccade to location of a visual target (T) presented either simultaneously with disappearance of fixation point (no-gap task) or after a gap period (gap task). For each visual stimulus, the solid bar indicates when stimulus is on. See METHODS for details.

tasks (Fig. 1), the monkey was required to look to the fixation point within 1,000 ms of its appearance. Once the eyes entered the computer-defined "window" (see below) centered on the fixation point, the fixation light remained on for a duration (visual fixation duration) that varied randomly from 500 to 800 ms, unless otherwise specified. At the end of this period, the fixation point was extinguished and, in the no-gap task, a peripheral target was illuminated simultaneously. In the gap task, the presentation of the peripheral target was preceded by a gap interval during which the monkey had to maintain steady (nonvisual) fixation on the now absent fixation point; the gap duration was kept constant at 200 ms, unless otherwise specified. The monkey was required to make a saccade to the new target within 500 ms of its onset and then maintain fixation upon it for 500 ms to correctly perform the task and receive a water reward.

The animals were given water through a sipping tube as a reward for saccades landing into the computer window surrounding the visual target. Target as well as fixation windows were square and measured between  $\pm 1.4$  to  $\pm 3$  deg around the target position. If the position of the eyes deviated out of the computer window during visual or nonvisual fixation period, the trial was aborted and no reward was delivered. No additional reward was given for short-latency saccades. Monkeys would typically complete between 600 and 1,200 trials in a 2- to 3-h experimental session taking place five to six times per week. They received water until satiation, after which they were returned to their home cage.

Before beginning each session, the gain and offset of the eye position signals were calibrated by adjusting their respective values while the animal looked at visual targets projected at known locations on the visual screen. During the sessions, the eye position signals were displayed on a storage oscilloscope that was triggered by the target onset.

In almost all sessions, no-gap and gap trials were interleaved randomly and had equal probability. The only two exceptions were the sessions in which we varied either the gap duration (all gap durations including no-gap trials had equal probability) or the initial eye fixation position (only gap trials were used). Throughout this study, only one type of experiment was performed by the animal in a single session. At the start of the sessions, both monkeys were familiar with the experimental setup and paradigms, because they had received initial training after the surgery (*monkey 1* was used additionally for neurophysiological experiments). The animals first were trained to maintain fixation of a central target spot (fixation task). Subsequently, they were required to make a saccade to a second eccentric target that appeared at the time the fixation target disappeared (no-gap task). Finally, the gap task was introduced. We never used the overlap task, in which the fixation

target remains present during target presentation. During this period of initial training, the target was randomized between two and eight locations. This is in contrast with the experiments described here in the sense that the first recording session was the first time that the animals were exposed to a block of trials with a single target location. Moreover, a period of rest ( $\geq 3$  wk) separated the initial training period from the recording sessions described in this report. The latter recording sessions started with the animals being trained to make saccades to a single target location located in the right visual hemifield. Target uncertainty then was introduced, first by varying the direction of the target and second by varying its amplitude. The effects of gap duration subsequently were investigated and followed by the study of initial eye position effects. The animals then were trained to make saccades to a single target location located in the left visual hemifield. The effects of initial eye position were investigated again, and in *monkey 2*, the target direction and amplitude as well as the gap duration variation were studied additionally.

#### Data collection and analysis

Horizontal and vertical eye position signals were digitized at 500 Hz and stored on a hard disk and analyzed off-line on a Sun Sparc 2 Workstation. Saccades were detected and marked using a computer program that identified the beginning and end of each saccade using velocity and acceleration threshold criteria and template correlation (described by Waitzman et al. 1991). For each recorded trial, saccade recognition was verified by visual inspection and, when needed, the saccade start and end times were marked manually using a 20 deg/s velocity crossing threshold. The SRT was measured as the interval from target onset to the beginning of the saccade (Fig. 1). Computer software measured and stored the following parameters: the time of occurrence of saccade start and end, movement duration, vectorial and component amplitudes, movement direction, vectorial and component peak velocities, initial and final eye positions, target location, and visual fixation and gap durations preceding target onset.

Analysis of SRTs was restricted to correct saccades initiated between 70 and 500 ms after target onset. The small percentage of saccades with latencies shorter than 70 ms were classified arbitrarily as anticipatory because they commonly exhibited lower peak velocities and larger targeting errors as reported in previous studies (Bronstein and Kennard 1987; Fischer and Weber 1993; Fischer et al. 1993; Kalesnykas and Hallett 1987; Smith and van Gisbergen 1989; Wenban-Smith and Findlay 1991). The saccades with latencies beyond 500 ms were presumably not triggered by the appearance of the visual target. We identified the first mode of the SRT distributions, collected mainly in the gap task, as containing express saccades (see Fig. 2). Based on all the SRT histograms obtained from the two animals studied here, we defined arbitrarily the latency of these movements to range from 70 to 120 ms. We named regular saccades those that compose the second mode of the SRT distributions, with latencies ranging from 120 to 500 ms but mostly concentrated from  $\sim 130$  to 200 ms. The mean of SRT distributions have been used extensively in previous studies to describe and analyze SRT data. However, because mean SRT values alone do not capture the multimodal nature of these distributions, we quantified our data using: percentage of express saccades; mean reaction time of all correct saccades (mean SRT); mean reaction time of regular saccades (when  $\geq 10$  saccades were produced); and mean reaction time of express saccades (when  $\geq 10$  saccades were produced). The magnitude of the gap effect for all responses was obtained by subtracting the mean SRT obtained in the gap trials from the mean SRT obtained in the no-gap trials. A gap effect for regular saccades only also was computed.

Because the assumption of normality of the distributions was not tenable, comparisons between the SRT distributions obtained

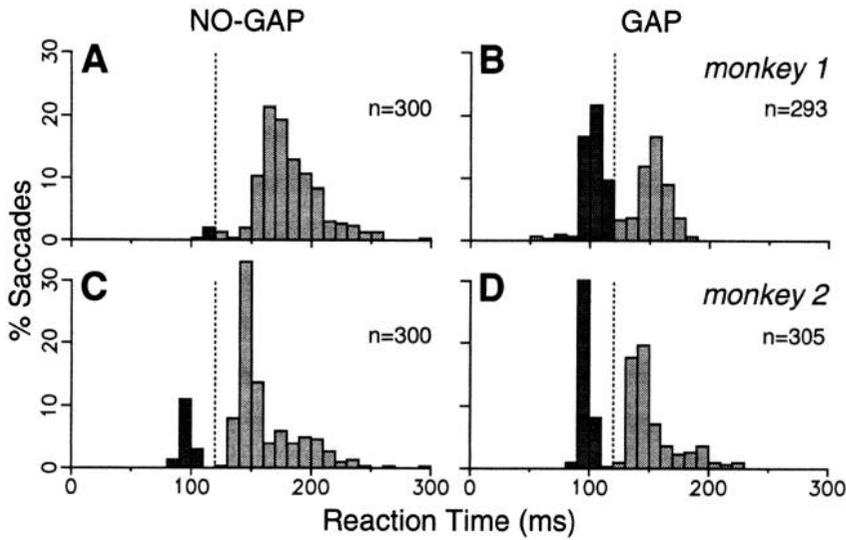


FIG. 2. Histograms of typical distributions of saccadic reaction time in no-gap (A and C) and gap (B and D) tasks. Results from *monkey 1* (3rd training session) making saccades to a right-up 10 deg target (A and B) and *monkey 2* (2nd training session) making saccades to a right 10 deg target (C and D). Binwidth is 10 ms. Vertical dotted lines indicate upper limit (120 ms) of range of express saccades.

in different conditions were performed using the distribution-free Rank Sum test of Mann-Whitney or the Kruskal-Wallis analysis of variance (ANOVA) on ranks followed by an all pairwise multiple comparison procedure (Dunn or Student-Newman-Keuls method). Percentages were compared with the  $\chi^2$  test.

RESULTS

Effects of training

The monkeys were first trained to make saccades toward one single target location, which was 10 deg eccentric from the fixation point situated at the straight-ahead position. For both monkeys, the target was located initially in the right hemifield (45 and 0 deg direction for *monkeys 1* and 2, respectively), and subsequently in the opposite left hemifield (225 and 180 deg direction for *monkeys 1* and 2, respectively). Figure 2 illustrates typical SRT distributions for both animals in the no-gap and gap paradigms when the target was located in the right hemifield. In the gap task (Fig. 2, B and D), the SRT distribution was often bimodal, with the first peak centered ~100 ms and the second ~150 ms. In the no-gap task (Fig. 2, A and C), the number of saccades in the first express mode was reduced significantly, therefore rendering the SRT distribution less clearly bimodal with most of the saccades grouped in the second regular mode.

The training effects on SRTs are quantitatively summarized in Fig. 3 for no-gap (filled symbols) and gap (empty symbols) trials. Even though the target location was very predictable in this condition, anticipatory responses were rarely produced: 0.48% (28/5836) in *monkey 1*, and 0.05% (4/7888) in *monkey 2*. When the target was located in the right hemifield, the animals produced a noticeable percentage of express saccades in the gap (41.7 and 36.5% for *monkeys 1* and 2, respectively) and no-gap task (13.4 and 12% for *monkeys 1* and 2, respectively) in the first session of training (Fig. 3, top). This percentage increased during training, except for no-gap saccades in *monkey 1*; the percentage of express saccades for these trials decreased to a minimal value (~3%). In contrast, when the target was

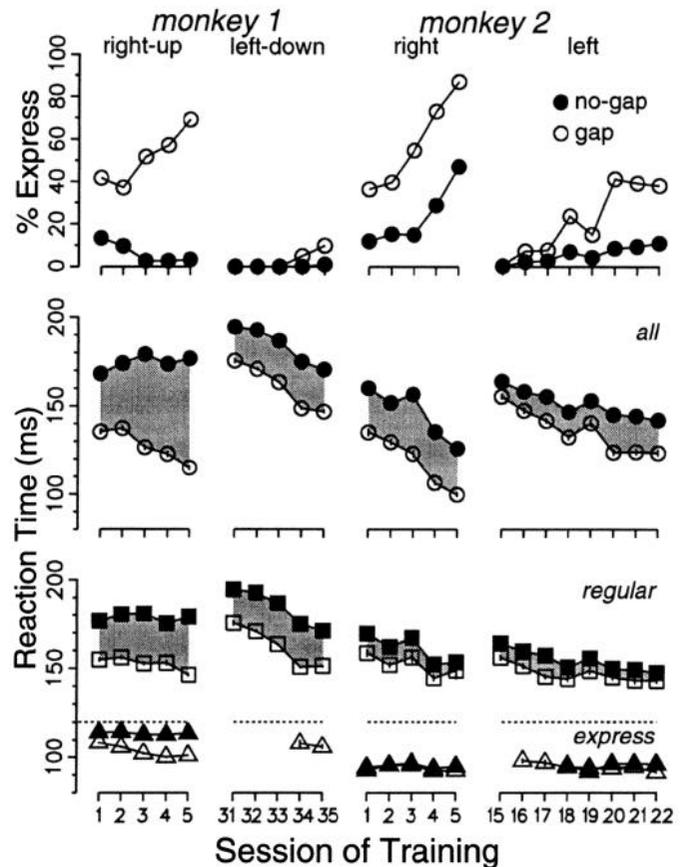


FIG. 3. Effects of training to a single target on saccadic reaction time. Top: percentage of express saccades; middle: mean reaction time of all saccades; bottom: mean reaction time of regular (squares) and express (triangles) saccades. Filled symbols are for no-gap trials, whereas empty symbols are for gap trials. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean and are smaller than symbol size. Each session of training contains a total of ~300 saccades in both no-gap and gap trials.

located in the left hemifield, the animals made no express saccades in the first session. After five sessions, the SRTs of *monkey 1* finally exhibited an express mode (10% in gap trials). For *monkey 2*, the percentage of express saccades increased gradually and after six sessions reached a value of ~40 and 10% in gap and no-gap trials, respectively. The mean SRT and the latency of regular saccades for both gap and no-gap trials generally decreased in a concomitant fashion during training (Fig. 3, *middle* and *bottom*). Consequently, the magnitude of the gap effect was roughly constant during training, particularly for regular saccades (Fig. 3, *shaded areas*). For *monkey 1*, making saccades to the right-up target, the mean SRT in no-gap trials appeared unmodified and therefore the magnitude of the gap effect for all saccades increased markedly. In *monkey 1*, the gap effect was  $34.9 \pm 15.1$  ms (mean  $\pm$  SD; range: 19.1–61.8) when considering all saccades and  $23.8 \pm 4.0$  ms (19.1–32.6) for regular saccades. In *monkey 2*, these figures were  $19.7 \pm 7.5$  ms (8.4–33.6) and  $7.8 \pm 2.5$  ms (4.4–11.7). Note that for each target and in each session there was always a gap effect, even though there were sometimes no express saccades produced.

#### *Influence of spatial factors*

After the training to a single target location, the spatial selectivity of express saccades was investigated by varying first the target direction while keeping the amplitude constant, and then the target eccentricity while keeping the direction constant.

**SPATIAL SELECTIVITY OF EXPRESS SACCADES.** In the direction variation experiments, the target was presented randomly at one of two, four, five, eight, or nine possible directions including the trained location. In all these experiments ( $n = 13$ ), *monkey 1* produced 0.06% (5/7985) anticipatory responses, and *monkey 2* produced 0.04% (1/2772). Figure 4A shows the results from *monkey 2* obtained in two consecutive sessions in which the target direction was varied randomly. The animal was trained previously to make express saccades to the right target location (direction 0 deg) and the saccade target appeared randomly at one of eight locations (45 deg apart, from  $-180$  to  $+180$  deg) during the first session, and at one of five locations (22.5 deg apart, from  $-45$  to  $+45$  deg) during the second session—due to overlap of target locations in the two sessions, there was a total of 10 target locations. Although no-gap and gap trials were interleaved during these sessions, only the results from the correct saccades produced in gap trials are shown in Fig. 4; the observations were qualitatively similar for no-gap trials (see below), except that the number of express saccades was reduced. Inspection of the SRTs at each target location reveals that saccades with express latencies were still produced despite target location uncertainty, but were directed only at the trained target and spatially adjacent targets located up to  $\pm 45$  deg from the latter (Fig. 4A).

Figure 5 illustrates the results obtained for both gap (empty symbols) and no-gap (filled symbols) trials in three direction variation experiments (right-up target for *monkey 1*; right and left target for *monkey 2*). In these experiments, anticipatory responses were produced only by *monkey 2*: 0.04% (1/2772). The percentage of express saccades (Fig.

5, *A, D*, and *G*), mean SRT (Fig. 5, *B, E*, and *H*), and regular saccade latency (Fig. 5, *C, F*, and *I*) displayed a broad tuning with respect to target direction, with considerable variability from one data set to another. In general, mean SRT and regular saccade latency reached a minimum at the trained target direction, and percentage of express saccades attained a maximum at the trained target direction; the occurrence of express saccades appeared to be correlated inversely with the latency of regular saccades (Fig. 5, *C, F*, and *I*). The mean SRTs (as well as regular saccade latencies) in gap trials showed a similar tuning as that observed in no-gap trials, which indicated a constant gap effect. Indeed, a positive gap effect was observed for almost all target locations, but it did not seem to be as strongly tuned for the trained target direction as were express saccades and SRTs (Fig. 5, *shaded areas*). In *monkey 1*, the gap effect had a mean of  $20.4 \pm 8.3$  ms (range: 10.4–32.5) when considering all saccades and  $17.3 \pm 5.1$  ms (10.4–23.5) for regular saccades. In *monkey 2*, these figures were  $9.8 \pm 5.9$  ms (0.8–20.7) and  $5.5 \pm 4.2$  ms ( $-3.5$ –12.3). Note that the spatial selectivity of express saccades was not due to the incapability of the animals to generate express saccades to the untrained targets, for these targets elicited express saccades when the monkey was provided with sufficient training. For instance, when *monkey 2* was trained to make express saccade to the 10 deg right target, it failed to make express saccades to the target located 10 deg left (Figs. 4A and 5D,  $\pm 180$  deg). However, after subsequent training to this latter target location, express saccades were produced (Fig. 5G).

In the amplitude variation experiments, we randomized six different target amplitudes: 3, 6, 10 (the trained target amplitude), 14, 20, and 30 deg. Figure 4B shows results from *monkey 2* obtained in a single session and from correct saccades made in the gap trials only. Inspection of the SRTs at each target location reveals that saccades with express latencies were directed mainly at the trained target. Their number decreased with increasing spatial disparity relative to the trained target amplitude; almost no express saccades were observed for the extreme target amplitudes, i.e., 3 and 30 deg.

Figure 6 illustrates the results obtained for both gap (empty symbols) and no-gap (filled symbols) trials in three amplitude variation experiments (right-up target for *monkey 1*; right and left target for *monkey 2*). In these experiments, anticipatory responses were produced only by *monkey 2*: 0.13% (2/1487). The amplitude tuning of percentage of express saccades (Fig. 6, *A, D*, and *G*), mean SRT (Fig. 6, *B, E*, and *H*), and regular saccade latency (Fig. 6, *C, F*, and *I*) was broad. Mean SRT and regular saccade latency reached a minimum for the trained target amplitude, and percentage of express saccades usually attained a maximum at the trained target amplitude; the occurrence of express saccades was associated with the regular saccades of shortest latencies (Fig. 6, *C, F*, and *I*). With respect to target amplitude, the variation of mean SRT (as well as regular saccade latency) in gap trials paralleled the one observed in no-gap trials. Consequently, a positive gap effect was present for each target location, but it did not appear tuned for the trained target amplitude (Fig. 6, *shaded areas*). In *monkey 1*, the gap effect had a mean of  $29.8 \pm 6.9$  ms (range: 21.7–37.9)

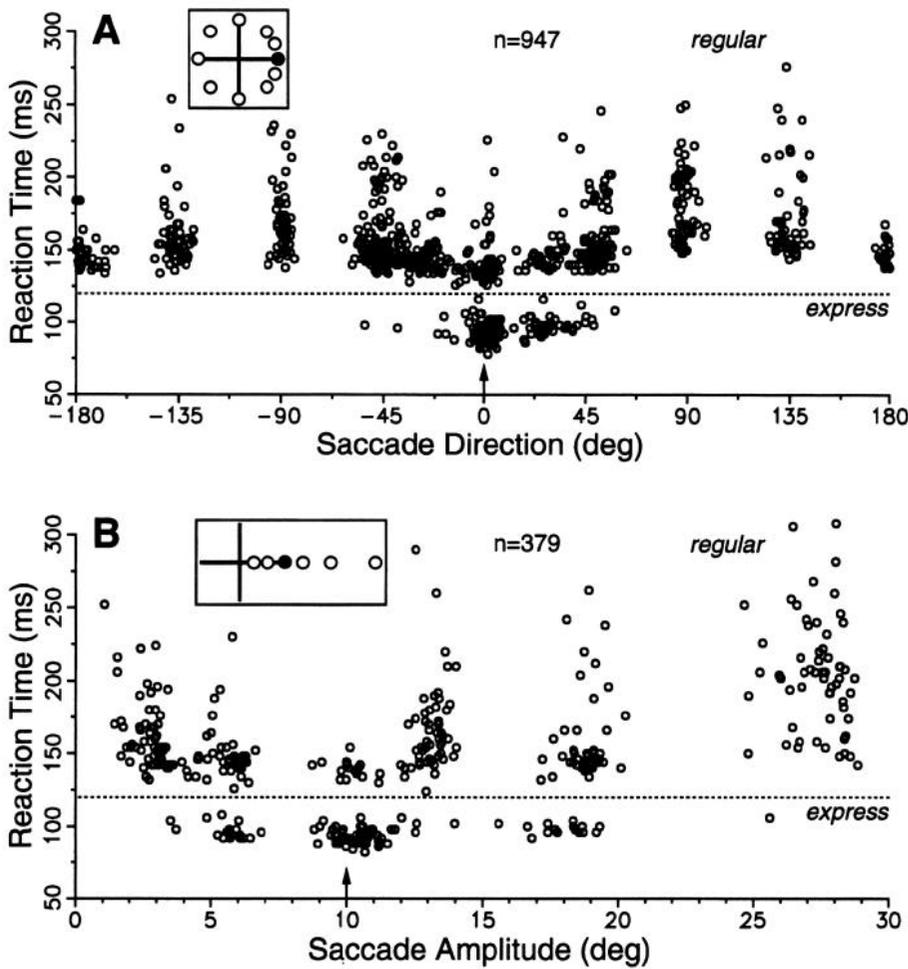


FIG. 4. Variation of saccadic reaction time with direction (A) and amplitude (B) of saccades made in response to targets of different direction (constant amplitude: 10 deg) or amplitude (constant direction: 0 deg). Each data point represent a correct saccade. Results are from *monkey 2* trained previously to make saccades to a right 10 deg target.  $\uparrow$ , target direction or amplitude for which animal has been trained previously. Horizontal dotted lines indicate upper limit of range of express saccades.

when considering all saccades and  $19.6 \pm 3.7$  ms (15.1–25.7) for regular saccades. In *monkey 2*, these figures were  $18.4 \pm 14.1$  ms (3.5–57.4) and  $13.6 \pm 15.5$  ms (1.4–56.1).

**EFFECTS OF SPATIAL UNCERTAINTY.** We investigated the change in percentage of express saccades and SRTs for saccades made to the trained target location when the spatial uncertainty about the target direction (constant amplitude: 10 deg) was increased for the first time after initial training. The data obtained in one experimental session when only one target position was used were compared with the session the following day, in which two (180 deg apart; *monkey 1*) or eight (45 deg apart; *monkey 2*) target positions were possible. Anticipatory responses were produced only by *monkey 1* for the one-target condition: 0.83% (5/603). When the trained target location was presented randomly with additional untrained locations, the percentage of express saccades directed to this target decreased significantly ( $\chi^2$ ,  $P < 0.001$ ; Fig. 7, A and D) and the SRTs increased significantly (Mann-Whitney rank sum test,  $P < 0.0001$ ; Fig. 7, B and E), except for the no-gap saccades in *monkey 1*. The regular saccade latency did not vary with the reduction of target predictability, except for the no-gap saccades in *monkey 2* which decreased significantly (Mann-Whitney rank sum test,  $P < 0.0001$ ). In both animals, express saccade

latency in gap trials increased significantly (Mann-Whitney rank sum test,  $P < 0.05$ ; Fig. 7, C and F).

*Influence of temporal factors*

To determine whether the duration of the period preceding target presentation was important to reduce SRTs and generate express saccades, we varied the duration of the visual and nonvisual (gap) fixation that the animal was required to maintain before target onset.

**EFFECTS OF VISUAL FIXATION DURATION.** To test the influence of the duration of visual fixation of the fixation point on the occurrence of express saccades and SRTs, we used three different durations of visual fixation before the fixation point disappeared: 200, 500, and 800 ms. A single trained target was presented and only one of the visual fixation durations was used in a session. A visual fixation duration of 500 ms was used on the first session, followed by a 800-ms duration in the second session, and then a final session with 200 ms as visual fixation duration. Figure 8 shows quantified results for both animals (right-up target for *monkey 1*; right target for *monkey 2*). In these experiments, *monkey 1* produced 0.22% (4/1805) anticipatory responses, and *monkey 2* produced 0.06% (1/1747). In both no-gap and gap trials, the percentage of express saccades increased

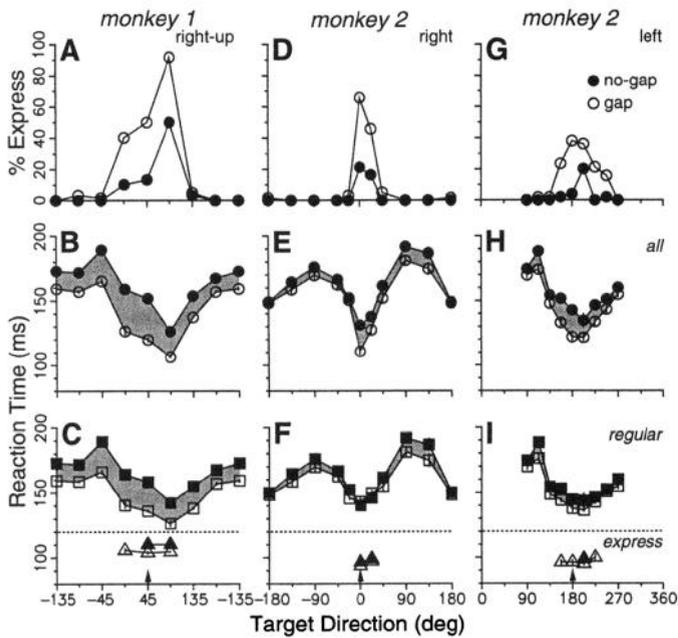


FIG. 5. Quantitative effect of target spatial location on saccadic reaction time when target direction was varied. Central target direction in each plot represents target direction for which the monkey has been trained to make (express) saccades previously. Data points from *monkey 1* making saccades to a right-up 10 deg target (A–C) and *monkey 2* making saccades to a right (D–F) or left (G–I) 10 deg target. Filled symbols are for no-gap trials, whereas empty symbols are for gap trials. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Each condition contains  $\geq 60$  saccades. All data sets were obtained from a single session of recording, except for *monkey 2* making saccades to right target, in which 2 sessions were used (see Fig. 4A). Percentage of express and saccadic reaction times from the 2nd session were normalized relative to results obtained at trained target location in 1st session. Only left hemifield (9 targets, 22.5 deg apart) was tested in the direction series of *monkey 2* making saccades to the left.

significantly with increasing visual fixation duration ( $\chi^2$ ,  $P < 0.005$ ; Fig. 8, A and D); in *monkey 1*, the difference between percentage of express saccades for 500 and 800 ms in the no-gap trials did not reach significance ( $\chi^2$ ,  $P = 0.07$ ). Concomitantly, the SRTs decreased significantly with increasing visual fixation duration (Kruskal-Wallis ANOVA on ranks,  $P < 0.0001$ ; Student-Newman-Keuls or Dunn method,  $P < 0.05$ ; Fig. 8, B and E). Note the particularly long-latency saccades in the no-gap trials with 200-ms visual fixation. The difference among the regular saccade latencies of each visual fixation duration was significant (Kruskal-Wallis ANOVA on ranks,  $P < 0.0005$ ; Fig. 8, C and F). Express saccade latencies were found to vary significantly only for the gap saccades in *monkey 2* (Kruskal-Wallis ANOVA on ranks,  $P < 0.0001$ ). In *monkey 1*, the gap effect had a mean of  $66.9 \pm 24.4$  ms (range: 52.6–95.1) when considering all saccades and  $36.8 \pm 23.2$  ms (18.3–62.9) for regular saccades. In *monkey 2*, these figures were  $44.0 \pm 12$  ms (31.8–55.8) and  $19.6 \pm 11.8$  ms (8.6–32.1).

**EFFECTS OF NONVISUAL FIXATION (GAP) DURATION.** We examined the SRT distribution to a single trained target when the gap duration was varied randomly from 200 to 600 ms, with incremental steps of 100 ms. All sessions included no-gap (or 0-ms gap) trials. Figure 9 summarizes the results

obtained in three experiments (right-up target for *monkey 1*; right and left target for *monkey 2*). In these experiments, *monkey 1* produced 0.55% (5/903) anticipatory responses, and *monkey 2* produced 0.16% (2/1234). From a small value in the no-gap task (8.7%), the percentage of express saccades in *monkey 1* increased with increasing gap duration to attain a maximum value (63.1%) in the 300-ms gap and then decreased significantly ( $\chi^2$ ,  $P < 0.05$ ) for longer gap durations (Fig. 9A). In *monkey 2*, the percentage of express saccades for the right target (Fig. 9D) was 41% in the no-gap task, and it reached 86.4% in the 300-ms gap and did not vary significantly from this level for longer gap durations. A similar observation was made for the left target (Fig. 9G), except that the percentage of express saccades was much lower: the maximum value was 15.8% in the 300-ms gap. For all experiments, the introduction of a gap increased significantly the percentage of express saccades ( $\chi^2$ ,  $P < 0.001$ ) with respect to no-gap trials (Fig. 9, A, D, and G), except for *monkey 2* making saccades to the left target in the 200-ms gap. In *monkey 1*, the 300-ms gap induced the shortest mean SRT, and the mean SRTs for longer gap durations were significantly longer (Fig. 9B). In *monkey 2*, mean SRT for both right and left target locations decreased with gap duration

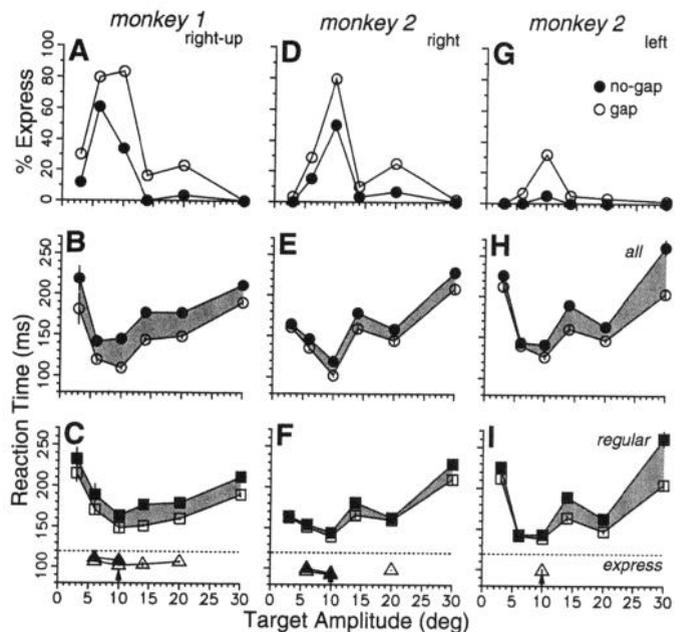


FIG. 6. Quantitative effect of target spatial location on saccadic reaction time when target amplitude was varied. Animals were trained previously to make express saccades to a target amplitude of 10 deg. Data points from *monkey 1* making saccades to a right-up 10 deg target (A–C) and *monkey 2* making saccades to a right (D–F) or left (G–I) 10 deg target. All data sets were obtained from a single session of recording. Filled symbols are for no-gap trials, whereas empty symbols are for gap trials. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Each condition contains  $\geq 60$  saccades.

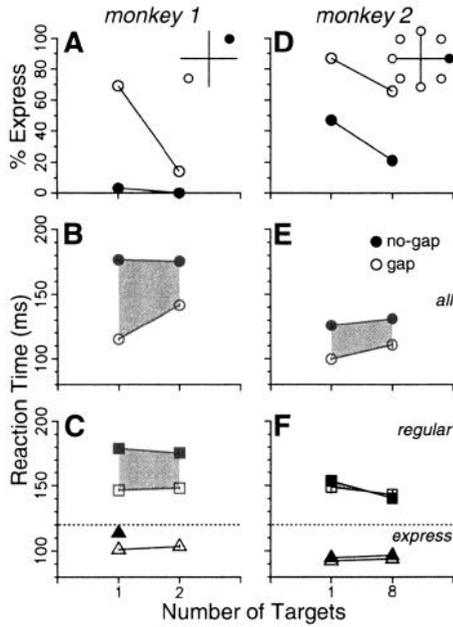


FIG. 7. Effects of uncertainty about target direction on saccadic reaction time. Percentage of express saccades (A and D) and mean saccadic reaction times (B, C, E, and F) observed in an experimental session in which a single target was presented (● in schematic) are contrasted against same parameters observed in a following experimental session in which the same saccade target was presented randomly with other target locations [○(s) in schematic]. Data points from *monkey 1* making saccades to a right-up 10 deg target (A–C) and *monkey 2* making saccades to a right 10 deg target (D–F). Filled symbols are for no-gap trials, whereas empty symbols are for gap trials. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Each condition contains from 68 to 303 saccades.

increasing from 0 to 300 ms and then asymptoted with further increase in gap durations (Fig. 9, E and H). There was a statistically significant difference among both the regular and express saccade latencies (Kruskal-Wallis ANOVA on ranks,  $P < 0.05$ ), except for the leftward express saccades of *monkey 2*. In general, the regular saccade latency was affected by gap duration to a lesser extent than mean SRT, thereby indicating that the gap-related modulation of the latter was due mainly to the changes in the percentage of express saccades. In *monkey 1*, the gap effect had a mean of  $37.7 \pm 8.9$  ms (range: 28.0–48.9) when considering all saccades and  $15.0 \pm 5.4$  ms (9.7–21.1) for regular saccades. In *monkey 2*, these figures were  $22.0 \pm 6.0$  ms (11.2–29.8) and  $8.4 \pm 4.0$  ms (1.1–12.2).

**GLOBAL EFFECTS OF PRECEDING FIXATION DURATION.** The separate demonstration of the effects of visual fixation and gap duration on SRTs (Figs. 8 and 9) suggests that both of these parameters contribute to express saccade generation. To evaluate quantitatively the relative influence of these two parameters on the occurrence of express saccade, we systematically varied both of them in a single experimental session. The target appeared at a single trained location and the visual fixation and gap durations were each selected randomly from four different values: respectively, 100, 200, 300, and 500 ms and 0, 100, 200, and 300 ms. The experiment was repeated in five sessions in each animal. Figure 10 illustrates typical results from *monkey 2*. For constant gap duration and pro-

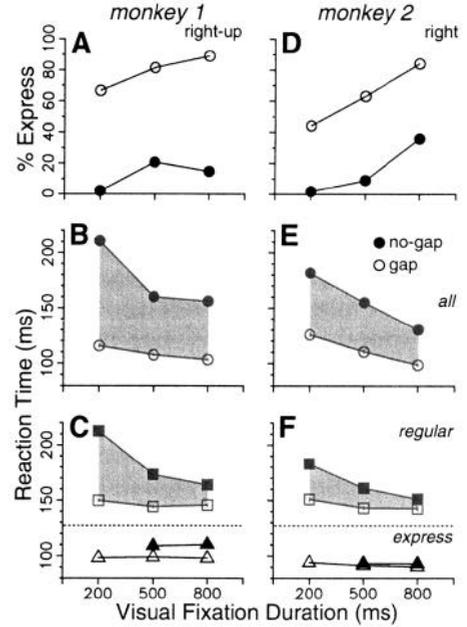


FIG. 8. Quantitative effect of visual fixation duration on saccadic reaction time. Data points from *monkey 1* making saccades to a right-up 10 deg target (A–C) and *monkey 2* making saccades to a right 10 deg target (D–F). Filled symbols are for no-gap trials, whereas empty symbols are for gap trials. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Data obtained from ~300 saccades in each condition.

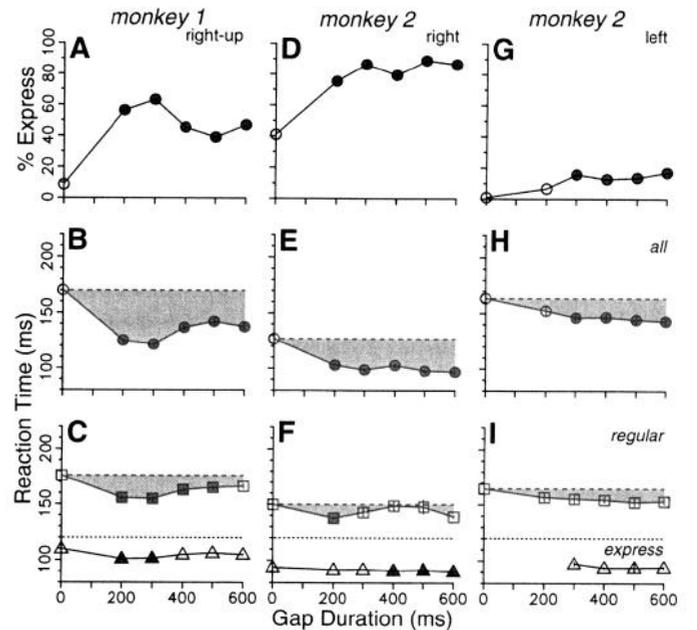


FIG. 9. Quantitative effect of gap duration on saccadic reaction time. Data points from *monkey 1* making saccades to a right-up 10 deg target (A–C) and *monkey 2* making saccades to a right (D–F) or left (G–I) 10 deg target. Gap effect is illustrated by shaded areas. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Empty symbol indicates lack of statistically significant difference ( $P < 0.05$ ) with no-gap trials. Number of saccades per condition varied from 72 to 187.

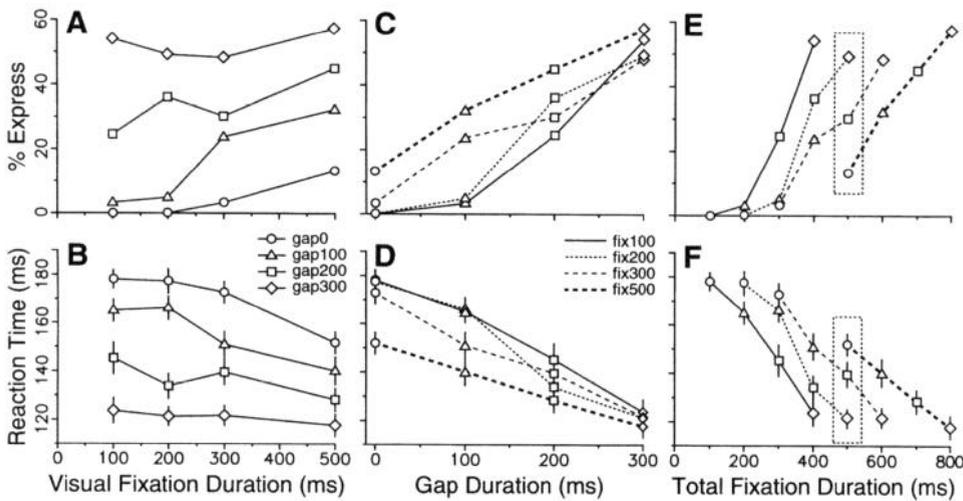


FIG. 10. Effects of preceding fixation duration on percentage of express saccades (A, C, and E) and mean saccadic reaction time (B, D, and F). Percentage of express saccades and mean saccadic reaction time are plotted in function of visual fixation duration (A and B), gap duration (C and D), and total fixation duration (visual fixation + gap duration) (E and F). Results from *monkey 2* making saccades to a right 10 deg target. Dotted box shows how percentage of express saccades (E) and mean saccadic reaction time (F) varied despite constant total fixation duration. Number of saccades per condition was  $\sim 60$ .

vided that the gap duration was  $< 200$  ms, the percentage of express saccades increased with increasing visual fixation duration ( $\chi^2$ ,  $P < 0.001$ ; Fig. 10A), while the SRTs decreased (Kruskal-Wallis ANOVA on ranks,  $P < 0.001$ ; Fig. 10B). For constant visual fixation duration, the percentage of express saccades also increased with increasing gap duration ( $\chi^2$ ,  $P < 0.0001$ ; Fig. 10C), while the SRTs decreased (Kruskal-Wallis ANOVA on ranks,  $P < 0.0001$ ; Fig. 10D). The gap-induced change in the percentage of express saccades (or mean SRT) diminished with increasing visual fixation duration, but it was always greater than the relation between percentage of express saccades (or mean SRT) and visual fixation duration (compare Fig. 10, C with A, D with B). As a consequence, the two temporal factors (visual fixation and gap durations) did not affect express saccade generation with the same magnitude. Mean SRT and express saccades prominence did not show a single relationship with the total fixation duration: visual fixation + gap (Fig. 10, E and F). For instance, the percentage of express saccades for a 500-ms visual fixation plus a 0-ms gap (13.3%, mean SRT = 151.9 ms) was much less than with a 300-ms visual fixation and a 200-ms gap (30.2%, mean SRT = 139.6 ms), which was also less than a 200-ms visual fixation and a 300-ms gap (49.2%, mean SRT = 121.4 ms; Fig. 10, E and F, dotted box). Thus gap duration was clearly a more potent factor than visual fixation duration to reduce SRT and generate express saccades.

#### Influence of initial eye fixation position

To test the influence of initial eye fixation position on the occurrence of express saccades and SRTs, the position of eye fixation was varied randomly and the animals were required to saccade to one of two possible targets presented randomly at 10 deg on either side of the fixation point: 0 and 180 deg direction in *monkey 2*; 45 and 225 deg in *monkey 1*. Three or five initial eye fixation positions were used, each spatially separated by either 10 or 20 deg. These initial eye positions were distributed along the same axis as the targets (Fig. 11) and only gap trials were used. The animal was trained previously to make express saccades to one retinal target location, or both. In these experiments,

*monkey 1* produced 0.17% (3/1797) anticipatory responses, and *monkey 2* produced 0.04% (1/2188). Figure 11 shows two sets of data obtained from both animals for saccades directed toward to the right hemifield. The salient effect of varying the initial eye fixation position upon the SRTs was of changing the relative frequency of the different modes. The percentage of express saccades decreased significantly ( $\chi^2$ ,  $P < 0.0001$ ) when the orbital position was shifted eccentric in a direction identical to the saccades. Concomitantly, the SRTs increased significantly (Kruskal-Wallis ANOVA on ranks,  $P < 0.0001$ ).

The quantified data from eight experimental sessions (4 in each animal) are summarized in Fig. 12. The percentage of express saccades increased with increasing initial eye fixation position in the direction opposite to the saccade direction (Fig. 12, A, D, G, and J). It varied from a minimum

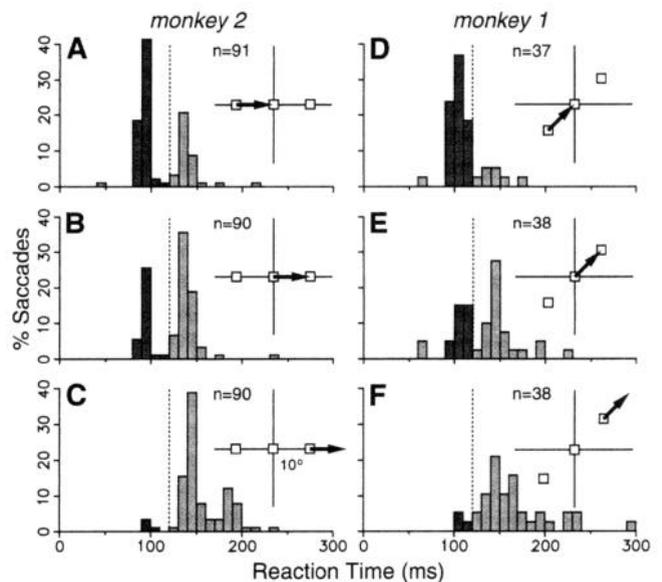


FIG. 11. Histograms of distribution of saccadic reaction time for saccades of identical metrics but initiated from different initial eye fixation position. Results from *monkey 2* (A–C) and *monkey 1* (D–F). Schematic in right-up portion of each histogram depicts saccades ( $\rightarrow$ ) made in each condition;  $\square$ , different initial eye fixation positions.

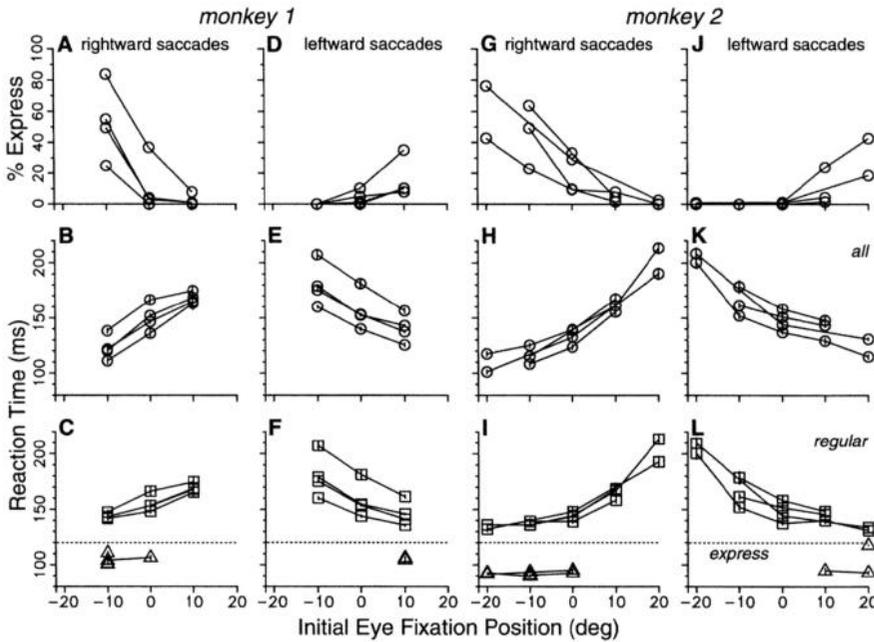


FIG. 12. Quantitative effect of initial eye fixation position on saccadic reaction time. Data from *monkey 1* (A–F) and *monkey 2* (G–L). Leftward and rightward shifts in initial eye fixation position relative to central position (abscissa) have negative and positive values, respectively. Horizontal dotted lines indicate upper limit of range of express saccades. Vertical bars represent standard error of mean. Each condition contains from 33 to 113 saccades.

value of 0% to a maximum of 81.6 and 41.3% for rightward and leftward saccades, respectively. In all sessions, the SRTs decreased with increasing initial eye fixation position in the direction opposite to the saccade direction (Kruskal-Wallis ANOVA on ranks,  $P < 0.0001$ ; Fig. 12, B, E, H, and K). The reduction in SRT appeared to be caused by a progressive reduction in regular saccade latency followed by the appearance of express saccades (Fig. 12, C, F, I, and L).

As a method of estimating quantitatively the change in SRTs across different orbital positions, the individual SRT data points for each session in each animal were fitted with a linear regression equation. The Pearson correlation coefficient of the regression lines ranged from 0.32 to 0.72, with a mean of 0.52 ( $F$ -test,  $P < 0.0001$ ). In *monkey 1*, the mean slope of the linear relationship between SRT and initial eye fixation position was 2.26 ms/deg (range: 1.83–2.61) and  $-2.00$  ms/deg ( $-1.74$  to  $-2.55$ ) for, respectively, rightward and leftward saccades. For *monkey 2*, these figures were 2.36 ms/deg (2.23–2.6) and  $-1.62$  ms/deg ( $-0.92$  to  $-2.03$ ). Thus for each 1-deg change in initial eye fixation position, there was about a 2-ms change in mean SRT. When testing the influence of initial eye fixation position on SRTs, we observed that saccades in the wrong direction were rare and, when they occurred, were initiated from eccentric eye positions and directed toward the primary position.

*Influence of preceding behavior*

To determine a contextual influence on express saccade generation, we analyzed our data with respect to the preceding SRT performance. Figure 13 shows SRT histograms of *monkey 2* for saccades to the right target (single target condition) that were preceded by either an express (Fig. 13, A and B; dotted line in E and F) or a regular saccade (Fig. 13, C and D; solid line in E and F) in the previous trial, regardless of whether this previous trial was a no-gap or a gap. After express saccades, the percentage of express sac-

cades was 29.8 and 75.2% for no-gap and gap trials, respectively. After regular saccades, the percentage of express saccades was significantly reduced to 14.7% for no-gap trials and 56.4% for gap trials ( $\chi^2$ ,  $P < 0.0001$ ; Fig. 13, C and D). After express saccades, the mean SRT was  $138.6 \pm 1.2$  (SE) ms and  $105.8 \pm 1.0$  ms for no-gap and gap trials, respectively. The mean SRT after regular saccades was  $157.7 \pm 1.4$  ms and  $118.7 \pm 0.9$  ms for no-gap and gap trials,

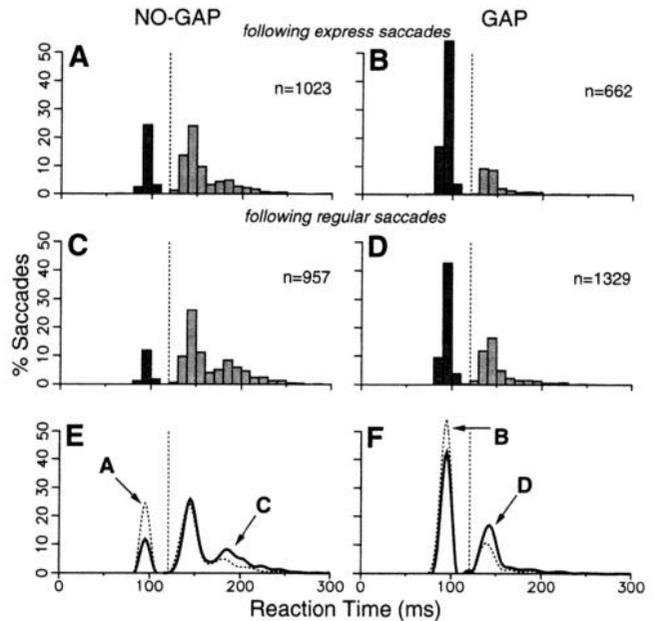


FIG. 13. Histograms of distribution of saccadic reaction time in the no-gap (A, C, and E) and gap (B, D, and F) tasks when preceding saccade was either an express saccade (A and B; dotted line in E and F) or a regular saccade (C and D; solid line in E and F) in the previous trial, regardless of whether this previous trial was a no-gap or a gap. Each histogram in A–D was fitted with a spline function (de Boor 1978), and respective no-gap and gap functions were superimposed in E and F to facilitate comparison.

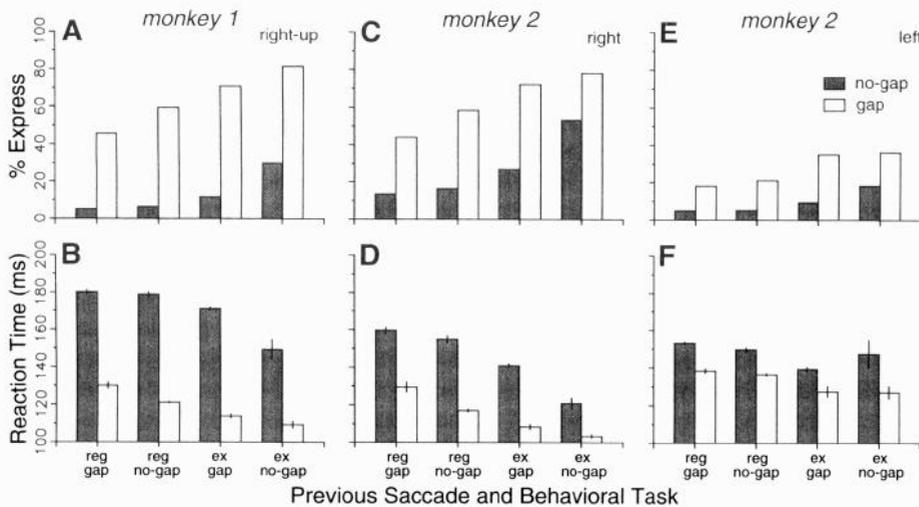


FIG. 14. Quantitative effect of preceding saccadic reaction time and behavioral task on percentage of express saccades (A, C, and E) and mean saccadic reaction time (B, D, and F). Data points from *monkey 1* making saccades to a right-up 10 deg target (A and B) and *monkey 2* making saccades to a right (C and D) or left (E and F) 10 deg target. No-gap and gap task results are shown in filled and empty columns, respectively. Results are shown for saccades following a regular saccade made in a gap trial (reg gap), a regular saccade made in a no-gap trial (reg no-gap), an express saccades made in a gap trial (ex gap), or an express saccades made in a no-gap a trial (ex no-gap). Vertical bars represent standard error of mean. Largest number of saccades in one condition was 1,483 and smallest was 33 (F, no-gap data of ex no-gap).

respectively. The respective SRT distributions were statistically different (Mann-Whitney rank sum test,  $P < 0.0001$ ). In summary, the saccades after express saccades had shorter latencies than those after longer-latency regular saccades.

The quantified data obtained in each animal are summarized in Fig. 14. These data were taken from sessions in which the animals generated saccades toward a single visual target. In these experiments, *monkey 1* produced 0.42% (32/7,641) anticipatory responses, and *monkey 2* produced 0.05% (5/9,635). In Fig. 14, the percentage of express saccades and mean SRT for both no-gap (filled columns) and gap (empty columns) trials are plotted with respect to the preceding SRT (express or regular) and behavioral task (either a no-gap or a gap; see abscissa). One can see that the percentage of express saccades in both no-gap and gap trials was the lowest when a regular saccade occurred in the preceding trial and the highest when the preceding saccade had an express latency (Fig. 14, A, C, and E). Furthermore, for each case in which the preceding trial contained, respectively, an express saccade or a regular saccade, the percentage of express saccades was always higher when the preceding saccade was made in a no-gap trial rather than in a gap trial. The distinct increase in the percentage of express saccades according to the preceding SRT performance and behavioral task led to the modification of the SRT distributions and to a systematic decrease in mean SRT (Fig. 14, B, D, and F).

## DISCUSSION

We have shown that the training effect on express saccade occurrence was spatially selective (see Figs. 4–6). With respect to the no-gap condition, the gap paradigm produced a gap effect for nearly all the targets tested, but express saccades were not always generated. Furthermore, the physical absence of the fixation point before target onset was not a prerequisite for the occurrence of express saccades. The latter depended on both the type (visual or nonvisual) and the duration of the period the animal had to maintain fixation prior to target onset (see Figs. 8–10). The percentage of express saccades increased when the orbital position was

shifted eccentric in a direction opposite to the saccades, such that movements toward central eye position were facilitated (see Figs. 11 and 12). The probability of making an express saccade was reduced by target location uncertainty (see Fig. 7) and also depended on both the behavioral task (no-gap or gap) and the latency of the saccade that the monkey executed in the preceding trial (see Figs. 13 and 14); the greatest percentage of express saccades was observed when an express saccade had occurred in a preceding no-gap trial, whereas the lowest percentage was obtained when a nonexpress saccade was made previously in a gap trial. Our observations are consistent with the suggestion that training-dependent express saccades are caused by neuronal changes restricted to a specific locus—coding for the trained movements—in a neural map of saccades. We first discuss our data with respect to other SRT studies in monkeys. Then we address the various hypotheses that have been proposed and suggest a new hypothesis wherein advanced preparation of saccadic programs, under the modulatory influence of the state of fixation, is primarily responsible for express saccade generation.

### Comparison with previous studies

**SPATIAL ATTRIBUTES OF TRAINING EFFECTS.** Fischer and colleagues (Boch and Fischer 1986; Fischer et al. 1984) first showed a spatially selective effect of training on express saccades using a detection paradigm, in which the monkey was neither required nor rewarded for making saccades to a visual target appearing in the peripheral visual field, but for detecting its dimming. Making a saccade to the target, however, facilitated the detection and was therefore customary. Once trained with a target located in one visual quadrant in this “no-saccade-necessary” task, monkeys generated express saccades in this quadrant exclusively; the percentage of express saccades decreased with the distance of the saccade end position from the target position. In spite of the differences between our respective experimental protocol, our results reproduced and extended those of Fischer’s group.

The spatial extent of the express saccade selectivity indi-

cates that the training to a single target location facilitates the initiation of a subset of saccades, whose metrics are not restricted to the trained direction and amplitude but relatively similar to them. Visual space and saccadic eye movements are represented in the brain by the activity of neurons distributed on topographical maps, and these neurons are activated by stimuli restricted, for a particular neuron, to a specific range of directions and eccentricities, collectively defined as the neuron's receptive field or movement field. Our results are consistent with the notion that a single visual stimulus causes the activation of an ensemble of neurons that also can be activated by other adjacent stimuli. If training produces local changes in the activity of the population of neurons coding for the trained saccades, a range of saccades with related metrics thus can be affected partially. Although we trained our monkeys with a single target location at a time, the spatial selectivity of the express saccade does not signify that express saccades cannot be made to more than one spatial area of the visual field: monkeys can make express saccades to different visual targets presented randomly at various locations (see Fig. 12) (Fischer et al. 1984; Rohrer and Sparks 1993; Schiller et al. 1987).

**PRECEDING FIXATION.** Previous studies have documented separately the effect of preceding visual or nonvisual fixation. In the gap paradigm, increasing percentage of express saccades has been shown to accompany increasing gap duration  $\leq 200$  ms (Schiller et al. 1987). Increasing percentage of express saccades (Sommer 1994) and decreasing SRTs (Hanes et al. 1992) with increasing visual fixation duration have been observed in overlap paradigms. Some of these observations also have been reported in humans (Braun and Breitmeyer 1988; Findlay 1981; Kalesnykas and Hallett 1987; Mayfrank et al. 1986; Saslow 1967; Tam and Ono 1994; Tam and Stelmach 1993). To our knowledge, our data are the first experimental demonstration that these two effects are different.

A simple explanation for the fixation duration effects is that fixation behavior is a dynamic process that diminishes in intensity as fixation (visual or not) progresses, and therefore the processes related to the preparation of possible saccades taking place during the fixation period become less inhibited. Accordingly, motor preparation signals for potential saccades could rise with a time course similar to the decay in fixation signals. Moreover, the difference in the magnitude of the effect in gap trials versus no-gap trials suggests that the decay and rise of, respectively, fixation and motor preparation during nonvisual fixation should be more important than during visual fixation. For gap durations  $> 300$  ms, however, the variation of mean SRT and percentage of express saccades was idiosyncratic (see Fig. 9). For these long gap durations, one can speculate that the change in fixation and motor preparation signals may saturate (*monkey 2*) or be altered (*monkey 1*), thereby indicating that the events underlying nonvisual fixation are temporally limited. We do not know whether there is an upper limit in visual fixation duration.

**INITIAL EYE FIXATION POSITION.** In the only other available study, Rohrer and Sparks (1993) claimed that the frequency of occurrence of express saccades does not vary with changes in initial eye position similar to those used in our

experiments. Worthy of note, these authors neither defined the latency range of express saccades nor quantified their results. Moreover, their data do seem to show a variation, at least in mean SRT. The remaining discrepancy between our respective data may be reconciled by the fact that, in their results, the percentage of express saccades at the most ipsilateral position was maximal and therefore could not be increased by having the initial eye position more contralateral. Note that our results do not contradict the conclusion—based mainly on another experiment—reached by Rohrer and Sparks (1993) that express saccades are programmed in relative, not spatial coordinates.

Initiating saccades from different orbital positions appears intimately related to the action of recentering the eyes in the orbit. Because targets can be located anywhere in the visual field and the oculomotor range is limited, it is crucial to keep the eyes roughly centered in the orbit to permit maximal orbital reserve at both the beginning and end of an impending saccade of undetermined direction. Hence, movements starting from an eccentric position are more likely to be centripetal than centrifugal. In natural viewing conditions—when the head is unrestrained—this situation may be compensated by the involvement of the head in the displacement of the visual axis (see Becker and Jürgens 1992). The recentering aspect of the eye position effect on saccade initiation bears some resemblance with the mechanism responsible for the initiation of the rapid resetting eye movements (quick phases) of the vestibuloocular reflex.

**CONTEXTUAL FACTORS.** The effect of preceding trial performance on SRTs and express saccade occurrence has never been explored previously in monkey. In a different type of experiment and in human subjects, Jüttner and Wolf (1992) demonstrated that express saccade probability depends on stimulus sequence and target occurrence uncertainty. Manipulating the probability of target presentation by inserting catch trials—trials in which the target does not appear after the gap and for which the subject must maintain fixation—into a block of gap trials, these authors showed that the express saccade occurrence decreases with increasing percentage of catch trials, i.e., increasing target presentation uncertainty (see also Carpenter and Williams 1995; Gordon 1967; Kingstone and Klein 1993a; Näätänen 1972). In addition, the saccades after catch trials were found to have longer latencies than those after saccade trials.

This modulation of SRT can be understood by a temporary inhibitory effect of catch trials on the subject preparation for the next trial. The state of readiness (or motor intention) of a subject to make a saccade may therefore determine the SRT and express saccade occurrence. In our experiments, the level of motor intention on a given trial may have exerted a direct influence on the SRT performance in the subsequent trial. The highest percentage of express saccades was obtained when the animal generated, in the preceding trial, an express saccade in the no-gap task. In this circumstance, the motor intention was presumably at its highest level because the animal usually did not make express saccades in this task. In contrast, for the condition in which the lowest percentage of express saccade was obtained, the motor intention most probably was depressed after a regular saccade was made in the gap task because the animal predominantly pro-

duced express saccades in this task. Our results are consistent with the suggestion of Schall and Hanes (1993) that SRT should be modulated in advance by the state of movement intention.

One explanation for the related effect of target location uncertainty (see Fig. 7) is that the intention of making a saccade to a given target is reduced when several saccades are possible. This reduction in motor intention may be generated by inhibitory influences originating from competing oculomotor programs required to foveate the additional targets. Accordingly, there should be a limit in the number of distinct oculomotor programs a subject can prepare in advance.

### *Previous hypotheses*

In this section, we review the hypotheses based on the large body of observations made in concerted SRT studies performed in both monkeys and humans. It initially was suggested that the reduction in SRT, as well as the occurrence of express saccades, was due to the disengagement of covert attention induced by the disappearance of the fixation point (Braun and Breitmeyer 1988; Fischer 1987; Fischer and Breitmeyer 1987; Fischer and Weber 1993; Mayfrank et al. 1986). Alternatively, the reduction in SRT has been attributed to a gap-related disengagement of ocular fixation that facilitates premotor processes (Dorris and Munoz 1995; Fendrich et al. 1991; Kingstone and Klein 1993b; Klein 1993; Klein and Kingstone 1993; Klein et al. 1995; Munoz and Wurtz 1992, 1993b; Nozawa et al. 1995; Reuter-Lorenz et al. 1991; Sommer 1994; Tam and Ono 1994; Tam and Stelmach 1993). In addition to this fixation disengagement, which appears to be specific to the oculomotor system, some authors have argued that a nonspecific response preparation (warning) signal, afforded by the disappearance of the fixation point, facilitates SRT reduction (Kingstone and Klein 1993b; Klein 1993; Klein and Kingstone 1993; Reuter-Lorenz et al. 1995; Ross and Ross 1980, 1981). The attentional disengagement role in the gap effect has been challenged (Fendrich et al. 1991; Kingstone and Klein 1993b; Klein et al. 1995; Reuter-Lorenz and Hughes 1993; Reuter-Lorenz et al. 1991; Tam 1993; Tam and Ono 1994; Tam and Stelmach 1993; Walker et al. 1995; West and Harris 1993). Nonetheless, the fixation release hypothesis is not sufficient to explain all the experimental data. First, the disappearance of the fixation point before the appearance of a peripheral saccade target in the gap paradigm produces, relative to the no-gap condition, a general reduction in SRT for almost all target locations (see Figs. 5 and 6). However, this paradigm does not necessarily produce express saccades; their occurrence often requires training (see Fig. 3). Second, this training effect is spatially selective (see Figs. 4–6). This spatial selectivity of express saccades cannot be easily accounted for by a mechanism for disengaging fixation. The function of the fixation system—to control the maintenance of the eyes in a stable position—makes it hardly selective to a restricted set of movement metrics. The modulation of express saccade occurrence by changes in initial eye fixation position specific to the direction of the saccades is also inconsistent with a simple fixation disengagement hypothesis (see Figs. 11 and 12).

The selective occurrence of express saccades may be ex-

plained, however, by a motor preparation hypothesis, in which topographically organized motor programs coding saccade metrics can be prepared partially before target presentation and reach decision level soon after target onset. This hypothesis was originally and independently suggested by Kowler (1990) and Becker (1989). Kowler (1990) hypothesized that SRT can be shortened by advanced preparation of the saccadic programs, i.e., partial computation. Accordingly, it is only when the program appropriate for the target has already been prepared that SRT is reduced, because the subject only has to retrieve it from a memory “buffer” and execute it. Otherwise, the program has to be computed entirely, hence the increase in SRT. A similar hypothesis has been proposed by West and Harris (1993). Starting from Fischer’s (1987) view on saccade generation) the 3 process loop: attention disengagement, decision making, and computation of movement metrics), Becker (1989) reasoned that SRT is reduced by the gap because attention then is disengaged, but that the additional reduction in SRT leading to express saccade production requires training and should be afforded by the completion of the decision process. To further account for the spatially selective training effects, Becker hypothesized that express saccades occur because training induces local changes on a “decision” map. In this two-dimensional map of decision channels, each of the latter is associated with a retinal locus and a saccade is initiated when it is maximally excited, and its level of activation is influenced by excitation and inhibition from neighboring and remote channels, respectively.

### *A new motor preparation hypothesis*

We propose a new motor preparation hypothesis wherein saccadic programs are topographically organized in a two-dimensional map that contains the computation of saccade metrics, motor preparation, and saccade decision. This neural map of saccades is the central constituent of our motor preparation hypothesis, which contains the following proposals. 1) The effects of training on the neural map of saccades produce local changes that increase the level of motor preparation for a subset of saccade neurons encoding the metrics of saccades appropriate to foveate the trained target locations and concomitantly reduce the SRT. With maximal preparation, express saccades are ultimately produced. 2) Oculomotor programs at trained sites can be prepared in advance of target onset and multiple programs can coexist on the neural map of saccades before saccade decision. 3) Because saccades are encoded by populations of neuronal units in a motor map (Lee et al. 1988; Munoz and Wurtz 1995b), the preparation of oculomotor programs is facilitated by topographically adjacent programs with overlapping population of neurons and impeded by distant programs with distinct populations. 4) Independently of training, all oculomotor programs are facilitated by the release of fixation, which therefore produces the general reduction in SRT. A fixation-mediated inhibitory influence on oculomotor programs is surmised to be maximal at the start of fixation and to decay thereafter during visual fixation; the decrease is accentuated during nonvisual fixation. The level of motor preparation rises with a time course similar to the decay in fixation. 5) The neural map of saccades has access to

information about the position of the eyes in the orbit such as the preparation of each oculomotor program is modulated by specific changes in initial eye position. For example, the level of preparation for rightward saccades increases as the starting eye position of the saccades is being shifted from extreme right positions to extreme left positions. The more centripetal the saccades are, the greater the level of oculomotor preparation. 6) The preparation of each oculomotor program in an experimental trial is influenced by the level of preparation attained in the recent past in reaching the same target, i.e., by the state of movement intention achieved before the actual trial. This contextual influence is restricted to the motor programming associated with the target for which the behavior was performed previously; it does not extend to other target locations (Paré and Munoz 1996a). 7) Because the training effect is related to the saccade metrics and not the spatial location of the target, the neural map of saccades is organized topographically in relative coordinates (Rohrer and Sparks 1993).

#### *Relation to physiological studies*

The integrity of the superior colliculus (SC) is critical for the generation of express saccades (Schiller and Lee 1994; Schiller et al. 1987). The SC saccade neurons, which discharge a burst of action potentials immediately before saccades, form an orderly oculomotor map with attributes of our postulated neural map of saccades (see for review Sparks and Hartwich-Young 1989). Several of these saccade neurons also exhibit neuronal activity changes that occur time-locked to the onset of visual signals. A subset of saccade neurons additionally displays anticipatory activity (Glimcher and Sparks 1992; Munoz and Wurtz 1995a), which has been postulated to be related to the preparation of making a saccade. Training to one target may activate selectively the population of saccade neurons encoding the motor command of the corresponding saccade. A local change in the SC preparatory activity therefore may explain the spatial selectivity of express saccades as well as the correspondence between their broad tuning (see Figs. 5 and 6) and the dimension of the active population of saccade neurons for the trained saccade (Munoz and Wurtz 1995b). The eye position effects on saccade initiation may be explained by the observation that the preparatory changes in neuronal activity of these SC saccade neurons are influenced by eye position (Paré and Munoz 1996b). Fixation neurons found at the rostral border of the collicular map exhibit a discharge pattern reciprocal to that of SC saccade neurons, i.e., they discharge during fixation and pause during saccades (Munoz and Wurtz 1993a). In accordance with the observations that visual fixation reduces both the probability of evoking saccades and the amplitude of saccades evoked by electrical stimulation of the SC (Sparks and Mays 1983; Paré et al. 1994), it has been suggested that the release of the SC fixation activity facilitates saccade initiation (Munoz and Wurtz 1993b; Paré and Guitton 1994). The modulation of this fixational inhibitory influence on saccade neurons in the gap paradigm may account for the general SRT reduction, the gap effect (Dorris and Munoz 1995).

The activity of the SC fixation and saccade neurons is influenced by inputs originating from higher centers (cortex,

basal ganglia, and thalamus) that act to suppress or facilitate saccade initiation. Fixation-saccade interactions similar to those observed in the SC also may take place in these neural structures. Additional inhibitory influences on SC saccade neurons may be provided by the substantia nigra pars reticulata (Hikosaka and Wurtz 1983). Specific gap-related increases in neuronal discharge have been recorded in the frontal eye field (Dias and Bruce 1994), and the regulation of saccade initiation certainly involves the contribution of both the frontal and supplementary eye fields (Schall 1991a,b; Schall et al. 1995). Signals related to movement intention may be found in the supplementary eye field (Schall 1991a). Last, the learning capability of cortical areas and basal ganglia may account for the neuronal changes accompanying the training period from which express saccades emerge.

In a typical gap trial, the sequence of neural events is postulated to be as follows. At the start of fixation of the visual fixation point, fixation activity is maximal and no saccade activity is present. With ongoing visual fixation behavior, fixation activity decays while preparatory saccade activity rises. During the gap, this decay and rise is accentuated (Dorris and Munoz 1995; Munoz and Wurtz 1995a). If the preparatory activity is high at target onset (presumably because of repeated presentation of the target), the target-locked activity changes can permit directly the SC saccade neurons to fire a strong enough burst that immediately leads to the production of a saccade with express latency (Dorris et al. 1995). In contrast, if the preparatory activity is lower—because advanced preparation is precluded, e.g., low target presentation probability—the target-locked activity changes fail to generate a motor command and more time is needed for the visual signal to be processed and the presaccadic burst to be formed. Further reductions in the level of preparatory activity leads to additional increases in regular saccade latencies. This scheme can account for the observed bimodality in primate SRTs, and it is consistent with the visuomotor hypothesis, which states that the “visual” and “motor” discharges of collicular saccade neurons generate, respectively, express and regular saccades (Edelman and Keller 1996; Rohrer and Sparks 1986; Sommer 1994). It also further extends the latter hypothesis by postulating that, for a given saccade program, the level of motor preparation (under the influence of various factors) determines SRT and thus the probability of an express saccade being initiated.

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