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Express saccades and visual attention

B. Fischer and H. Weber

Saccade latency in context: Regulation of gaze behavior by supplementary eye field

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Fischer & Weber (F&W) demonstrate that saccade latency reduces as the time and location of the target become more predictable. The conditions yielding the shortest latency saccades are exactly those in which the distinction between anticipatory and visually guided saccades blurs (Figs. 2 and 6).

These findings replicate numerous investigations of the effects of foreperiod on response time (Niemi & Näätänen 1981). Response time is reduced if prior warning is given, and the degree of reduction is proportional to the reliability and salience of the warning event. The improvement in performance is commonly conceived as a result of covert preparation of the movement before execution.

We argue that latency in a gap task can be understood in terms of preparatory processes. Constant 200 msec gap durations are commonly used (e.g., Fischer & Boch 1983). To control for the obvious predictability, gap durations are selected randomly from a specific range (e.g., Schiller et al. 1987; Wenban-Smith & Findlay 1991); a 20 msec variation is hardly sufficient, however (Fischer & Ramsperger 1984). Using delays from 0–300 msec, saccade latency declines and the incidence of express saccades increases (Schiller et al. 1987; Wenban-Smith & Findlay 1991).

We measured latency in a rhesus monkey performing a modified gap task. Unlike other studies, the fixation spot changed color rather than disappearing. A green fixation spot signaled go trials (80%); red signaled no-go, with the monkey rewarded for maintaining fixation after target presentation. No-go trials were included to prevent anticipatory saccades. The target could appear at one of 4 positions (10° ecc). In one condition, gap durations were sampled with equal likelihood from 0–300 msec. Our findings (Fig. 1) replicated Wenban-Smith and Findlay (1991) with no bimodal latency distributions following any gap delay even though mean latency declined significantly (linear regression $df = 550$, $t = 13.4$, $p < 0.001$). The higher latency of these express saccades is explained by the use of four target positions, no-go trials, and the continued presence of the fixation spot.

This latency reduction following longer delays can be explained in terms of the conditional probability of target presentation at different times. Because the probability of each gap is constant and limited if the target has not appeared following a given time, then the probability of its appearing in the next interval increases. Thus, the passage of time itself conveys information that can lead to enhanced readiness to initiate the movement.

This temporal predictability can be controlled by using gap delays with constant conditional probability. Such a "nonaging" distribution of foreperiods has an exponentially declining probability of successively longer times (Fig. 1). Manual response times become unchanged with nonaging foreperiods (e.g., Baumeister & Joubert 1969). Using nonaging gap delays we found less latency variation. In the first 150 msec latency declined for both aging and nonaging conditions. Whereas latency in the remaining 150 msec for the aging condition continued to decline significantly ($df = 299$, $t = -2.6$, $p < 0.01$), latency in the nonaging condition did not. Furthermore, express saccade latencies following nonaging gaps were longer than those following the uniformly sampled gaps ($df = 302$, $t = -6.3$, $p < 0.01$).

The temporal pattern that influences latency cannot be identified on a single trial; it must be sensed in the context of many trials. This fact, in combination with the extremely short latency of express saccades, suggests that the state from which an express saccade can be generated must be achieved *before the trial begins*. F&W review the evidence for inhibitory control over the SC from the substantia nigra, controlled via the caudate nucleus from the cortex. They illustrate caudate afferents from FEF but the oculomotor caudate also receives afferents from the supplementary eye field (SEF) rostral to the supplementary motor area (SMA; Parthasarathy et al. 1992).

Neurons in SEF discharge before voluntary saccades (Schall 1991a; Schlag & Schlag-Rey 1987) and saccades are evoked by intracortical microstimulation of SEF (Schall 1991b; Schlag & Schlag-Rey 1987). SEF projects to superior colliculus and the brainstem saccade generator (Huerta & Kaas 1990; Shook et al. 1990).

Many studies have indicated that SMA is involved in planning and regulating movement (Goldberg 1985). Lesions of SEF in humans specifically impair the generation of sequences of remembered saccades (Gaymard et al. 1990). Moreover, SMA neurons discharge specifically for movement sequences (Mushiake et al. 1991). Recordings in FEF and SEF also indicate that whereas neurons in FEF are linked to visual stimuli and saccade metrics, cells in SEF are modulated more according to movement intention (Schall 1991a; 1991b). We believe a useful hypothesis is that SEF is responsible for organizing saccadic behavior in the context of current task contingencies. Thus, when conditions merit, SEF, in concert with FEF, allows the superior colliculus to generate express saccades.

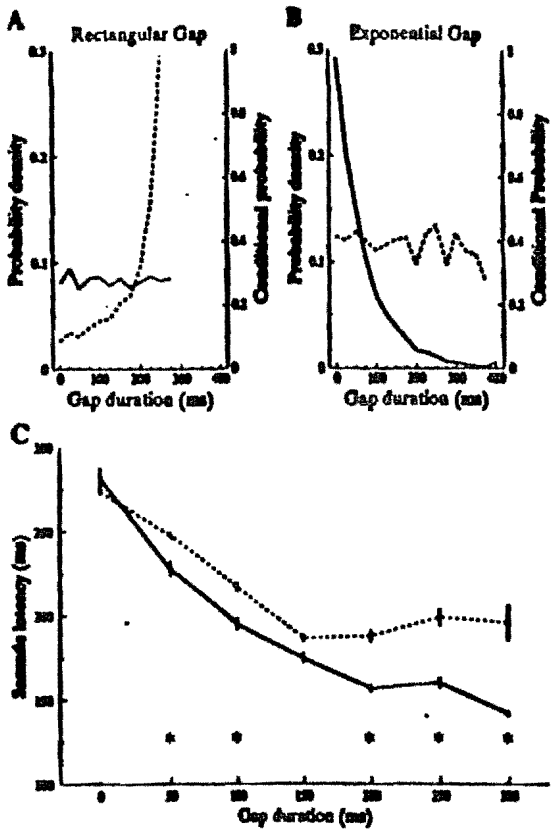

Figure 1 (Schall & Hanes). Saccadic performance with aging and nonaging gap durations. Gap delay was varied in two ways. A: Probability density (solid line) and conditional probability (dashed line) for gap delays sampled from a rectangular distribution ranging from 0–300 msec. With a constant probability density the conditional probability is initially low and increases for longer gaps. B: Probability density and conditional probability for gap delays sampled from a nonaging, exponential distribution. Note the constant conditional probability. C: Saccade latency as a function of aging (solid line) and nonaging (dashed line) gap delays. Vertical bars represent 1 standard error of the mean (SEM). Asterisks indicate delays at which the latency following nonaging gaps was significantly longer than the latency following aging gaps (*t* test, $p < 0.01$).