The underrated role of the "move system" in determining saccade latency

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Abstract: The Findlay & Walker target article emphasizes the role of the target-nonspecific "fixate" system while downplaying the role of the target-specific "move" system in determining saccade latency. We agree that disengagement of the fixate system is responsible for the target-nonspecific latency reduction associated with the gap effect. However, high target predictability and extensive training at a target location can also result in latency reductions, the culmination of this being express saccades. The target-specificity associated with the latter forms of latency reduction implicate a mechanism involving the move system. Recently discovered neurophysiological correlates underlying these behavioural phenomena reside in the superior colliculus.

Findlay & Walker's (F&W's) target article proposes a plausible five-level model of saccade generation that can account for a variety of oculomotor phenomena, including the gap effect and express saccades. The authors argue that the spatial selection of the upcoming saccade occurs in the WHERE pathway and make the "strong prediction" (sect. 4.4.1, para. 1) that the effects of such target selection on latencies will be small because the triggering stage is located in the entirely nonspatial WHEN pathway. Although F&W stress that this is a functional rather than a physiological model, for the phenomena of the gap effect and express saccades they speculate that these may result from the push-pull mechanism between the "fixate" (When pathway of level 2) and "move" (Where pathway of level 2) systems at the level of the superior colliculus (SC). In this scheme, the fixate system contains the triggering stage and thus determines saccade latency. Here we suggest that the contribution of the move system has been underrated in its role in the determination of saccade latency.

F&W base their claim that the WHERE pathway has little effect on saccade latency on the premise that "a number of studies have failed to find any effect of the number of potential target locations on saccade latencies (Heywood & Churcher 1980; Megaw & Armstrong 1973; Saslow 1967b)" (sect. 4.4.1, para. 2). In contrast, we have found that the majority of the literature supports quite the opposite notion that the number or predictability of potential target locations affects saccade latency (Abrams & Jonides 1988; Bartz 1962; Basso & Wurtz 1996; Carpenter & Williams 1995; Dorris & Munoz 1998; Hackman 1940; Klein & Pontefract 1994; Michard et al. 1974; Paré & Munoz 1996; Reuter-Lorenz & Fendrich 1992). These studies show that prior knowledge of where a target will be presented has a consistent effect on saccade latency in a manner that cannot be accounted for by the proposed spatially nonspecific fixate system. This attribute of the saccadic system has been implemented in a number of latency models in which saccades are elicited when a threshold level of activation in the spatially specific move system is surpassed (Carpenter & Williams 1995; Fischer et al. 1995; Hanes & Schall 1996; Kopecz 1995; Kopecz & Schoner 1995; see Pacut 1977 for review of threshold models).

The influence of the fixate system on saccade latencies is exemplified in the phenomenon known as the gap effect (sect. 4.1.1). When an initial fixation point is removed prior to the presentation of an eccentric target, saccade latencies are reduced compared to the condition in which the fixation point is not removed prior to target presentation. The gap effect occurs for saccades directed to any target location indicating that it is mediated by a spatially nonspecific mechanism unrelated to the move system (Kingstone & Klein 1993b; Walker et al. 1995). This view is bolstered by the physiological findings that both fixation-related neurons in the SC (Dorris & Munoz 1995) and frontal eye fields (Dias & Bruce 1994) show modulations in their activity in relation to fixation point disappearance in the gap paradigm.

The influence of the spatially specific move system on saccade latencies is exemplified in the reduction observed with the increased target predictability mentioned above and the phenomenon of express saccades (target article sect. 4.1.3). Although the target article heavily implicates a spatially extended fixate system in the generation of express saccades, two lines of evidence point to the importance of the move system in this phenomenon. First, as alluded to in the target article, express saccades are triggered only by suddenly appearing targets in the periphery. Express saccades are not generated to constantly present peripheral targets (Boch & Fischer 1986; Edelman & Keller 1996; Rohrer & Sparks 1993), nor are they directed away from targets as in the antisaccade paradigm (Everling et al. 1998; Fischer & Weber 1992). This view is supported by physiological evidence that suggests that the normally separate target- and saccade-aligned bursts of SC saccade-related neurons are fused into one larger burst equally aligned on both target appearance and the saccade during express saccades (Dorris et al. 1997; Edelman & Keller 1996).

Second, unlike the gap effect in which latencies are reduced to saccades directed to all targets in the visual field, the generation of express saccades is spatially specific. The percentage of express saccades directed toward a target location is dependent on both target predictability (Paré & Munoz 1996; Sommer 1997; however, see Rohrer & Sparks 1993) and the level of training to that target location (Fischer et al. 1984; Paré & Munoz 1996; Rohrer & Sparks 1993). A simple disengagement in the fixate system cannot explain this spatial specificity. Furthermore, F&W suggest that express saccades can be triggered by target presentation when the fixate system is disengaged during the gap paradigm. Again this cannot account for the spatial selectivity of express saccades because all areas of the move system should be disinhibited equally through the nonspecific disengagement of the fixate system. Although SC fixation neurons decrease their activity during the gap paradigm, thereby disinhibiting the move system as suggested, their activity does not show a greater decrease prior to the generation of express saccades compared to regular latency saccades (Dorris et al. 1997). However, a proportion of SC saccade-related neurons display early, low-frequency activity prior to the generation eye movements (Munoz & Wurtz 1995). This early activity has been shown to be related to the selection of targets from a number of possible stimuli (Basso & Wurtz 1998; Glimcher & Sparks 1992) and the probability of generating a saccade into the response field of a neuron (Dorris & Munoz 1998). Unlike SC fix-

ation neurons, this buildup activity of saccade-related neurons is inversely related to saccade latency (Dorris et al. 1997; Dorris & Munoz 1998; Everling et al. 1999) and is significantly higher prior to express than regular latency saccades (Dorris et al. 1997).

Taken together, the evidence suggests that removal of the fixation point during the gap paradigm can reduce latencies of saccades directed to targets in all regions of the visual field through disengagement of the fixate system. In addition, there are the equally important, and often underrated, effects of spatially specific events such as target number and predictability, which can also influence saccade latency. Only when both disengagement of the fixate system and advanced preparation of oculomotor goals in the move system occur together can the initial target-aligned response of SC saccade-related neurons surpass saccadic threshold resulting in saccades of express latency.