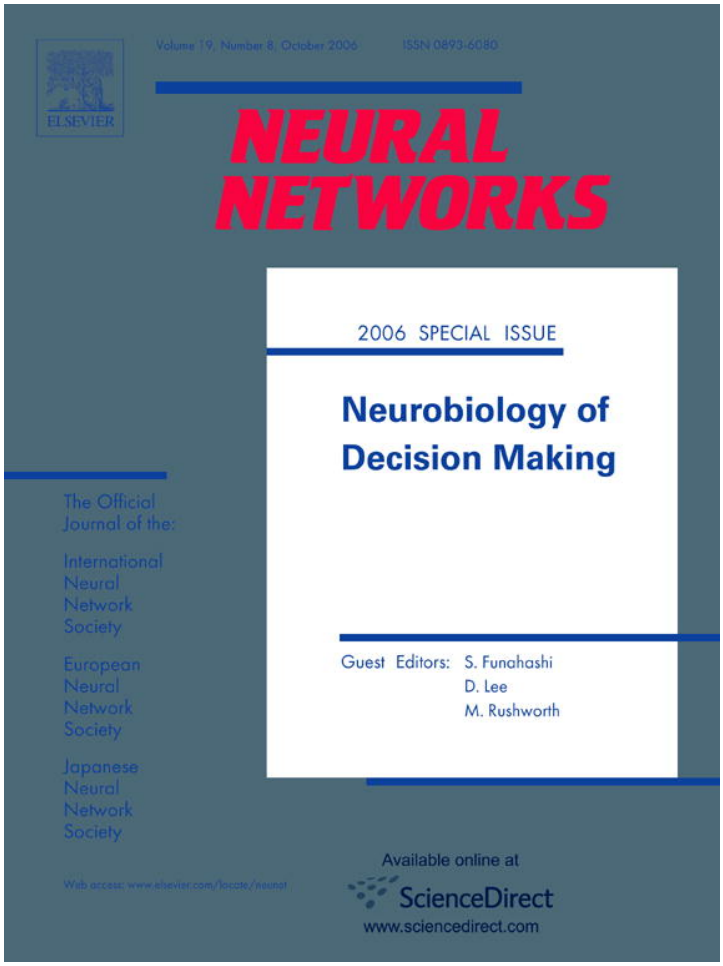

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The time course of saccadic decision making: Dynamic field theory

Claudia Wilimzig, Stefan Schneider, Gregor Schöner*

Institut für Neuroinformatik, Ruhr-University of Bochum, Germany

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Abstract

Making a saccadic eye movement involves two decisions, the decision to initiate the saccade and the selection of the visual target of the saccade. Here we provide a theoretical account for the time-courses of these two processes, whose instabilities are the basis of decision making. We show how the cross-over from spatial averaging for fast saccades to selection for slow saccades arises from the balance between excitatory and inhibitory processes. Initiating a saccade involves overcoming fixation, as can be observed in the countermanding paradigm, which we model accounting both for the temporal evolution of the suppression probability and its dependence on fixation activity. The interaction between the two forms of decision making is demonstrated by predicting how the cross-over from averaging to selection depends on the fixation stimulus in gap-step-overlap paradigms. We discuss how the activation dynamics of our model may be mapped onto neuronal structures including the motor map and the fixation cells in superior colliculus.

Keywords: Neural modelling; Selection; Saccades; Fixation

1. Introduction

Primates perform many tens of thousands of saccades per day, rapid eye movements to bring the saccadic goal onto the fovea. Saccadic movements are thus an important and ubiquitous prerequisite for the processing of visual information. The saccadic system is one of the best studied systems both at the neurophysiological and psychophysical level. For both reasons, saccadic eye movements are often used as a window into understanding the neural basis of sensori-motor decision making. Making a saccade necessarily involves decision making in two senses. First, among a set of potential visual targets, one must be selected as the next end-point of a saccade. Only in the laboratory is this process sometimes trivialized by reducing the visual array to a single visual item. In the real world, the visual array is rich and the decision of selecting one visual target must be stabilized against the influence of many distractors. Second, to initiate a saccade, the decision must be made to release the system from its previous state of fixation. Again, outside the laboratory this typically involves overcoming visual stimulation at the previous fixation sites. These two aspects of saccadic decision making have been referred to as the "when" and "where" processes (Findlay & Walker, 1999; van Gisbergen, Gielen, Cox, Bruijns, & Kleine Schaars, 1981).

Experimentally, the process of selection can be simplified into the form of double target paradigms with either two targets or one target and a distractor. Typically, if the targets are metrically close to each other, the nervous system does not select one of them but directs a saccade to an averaged spatial position, weighted by factors such as contrast, eccentricity, and probability and sometimes called the "center of gravity" of the two targets. By contrast, for metrically distant targets the nervous system always selects one of the two targets (Ottes, van Gisbergen, & Eggermont, 1984).

What is the neuronal basis of this selection process? One candidate is the superior colliculus, which provides a topographic map of saccadic end-points. A single, unambiguous target is represented by a relatively broad distribution of neurons that are activated in a graded way (for a review see Schall (2004b)). For two targets a bimodal distribution of activation can be found in the superior colliculus structures with – for sufficiently close targets – the center of gravity of the activation in the middle between the two targets (Basso and Wurtz (1998), review Schall (2004b)).

* Corresponding address: Ruhr-University of Bochum, Institut für Neuroinformatik, ND 04, 44780 Bochum, Germany. Fax: +49 234 32 14209.
E-mail address: Gregor.Schoener@neuroinformatik.ruhr-uni-bochum.de (G. Schöner).

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The first goal of this paper is to understand how saccadic decision making depends on the metric structure of the stimulus layout and subsequently how it relates to the respective neural representation in superior colliculus and related structures. Based on previous work by Kopecz and Schöner (1995), Schöner, Kopecz, and Erlhagen (1997) and analogous to work by Erlhagen and Schöner (2002) on arm movement planning we propose that parameters of saccadic movements are represented by dynamic fields, activation fields defined over continuous spaces such as the location of visual targets in retinal coordinates (see discussion of coordinate frames in Kopecz and Schöner (1995). Within these fields, input may induce localized distributions of activation, which are stabilized by local-excitatory and longer-range inhibitory interactions. We hypothesize that such localized peaks of activation reflect decisions and show that the transition from input-driven activation to localized peaks involves dynamic instabilities (from a bistable to a monostable state). Similarly, we hypothesize that the transition from averaging to selection is a second dynamic instability (from a monostable to a bistable state).

A signature of these instabilities are the *time-courses* of decision making processes. The shift from an averaging to a selection mode is driven not only by the metric distance between targets but also by the time available for processing. Experimental manipulations emphasizing accuracy rather than speed shift the balance from averaging to selection within the trial-to-trial variability (for monkey data, see Edelman and Keller (1998)). For sufficiently large distances, however, averaging cannot be observed even under speeded conditions – saccades always to go to one or the other target, and performance in the target–distractor paradigm may drop to chance level.

Here we argue that taking into account the inherent temporal dynamics of neural interaction provides an understanding of the time-course of decision making, the shift from early averaging to late inhibition. To show that, we extend an earlier Dynamic Field model (Kopecz & Schöner, 1995) to take into account the organization of neuronal activation into excitatory layers and inhibitory interneurons. This makes that inhibitory interaction becomes effective later than excitatory interaction. The resulting evolution of the state of the selection process matters, because movement plans are continuously fed into movement generation mechanisms. This postulate stands in contrast to classical information processing ideas of successive stages of processing which only transmit information when "done" with their computations (e.g. Miller (1988)), but is consistent with neurophysiological evidence for a continuous flow between stages (Bichot, Rao, & Schall, 2001; Miller, Riehle, & Requin, 1992).

Given this dynamic form of movement *planning*, how does the system transition into action? The decision to initiate a saccade is never against a entirely neutral background. Typically, a saccade is initiated through a transition from a fixation state, in which the fixation stimulus is foveated. That the visual structure supporting fixation behavior (the fixation signal) matters has been shown both at the neural (Munoz & Wurtz, 1993a, 1993b) and at the behavioral level (Ross and Ross (1980), for an overview see Kopecz (1995)). The competition between the fixation state and movement initiation can be studied in gap-step-overlap paradigms in which the time interval between the offset of the fixation stimulus and the onset of the movement target is varied. Because this leads to a variation of the latency of saccadic initiation – the later, the more temporal overlap between fixation and target signal – the time-course of movement planning can be probed in this paradigm as well.

We model the interaction of fixation and saccadic planning in a second Dynamic Field, the *initiation* field, which receives input from the selection system as well as directly from the sensory surface. The complete model is thus a two-level system, each level consisting of an excitatory and an inhibitory layer (Fig. 2). The decision to initiate a saccade occurs when the previously stable "fixation" peak in the initiation level, becomes unstable through competition with input from the selection field. This generates a new peak at the planned saccadic end-point. The saccadic movement that ensues, but is not modelled here, reinstates the fixation peak. Because multiple factors contribute to the instability that leads to movement initiation, this account differs importantly from accounts in which movement initiation is initiated when a threshold is reached (e.g., Ratcliff and Rouder (1998), for critical discussion see Schall (2004a)).

Varying one such factor, the time at which the fixation signal is extinguished, leads to an account for the gap-step-overlap effect. Countermanding can be understood as the influence of a second factor, the global level of activation in the selection field. The instruction to suppress a previously planned saccade is modelled as global inhibitory input to the selection system. In the initiation level; this shifts the balance of competition in favor of fixation. We show that whether or not a saccade can be suppressed depends on the strength of fixation activity. Because saccades with shorter than average latency are more likely to overcome the renewed fixation signal, failed-to-inhibit saccades are typically faster than regular saccades. On rare occasions, however, failed-to-inhibit saccades may be much slower than regular saccades. This comes from competition between the saccade-related and fixation related activation. Our model explains contradictory psychophysical evidence for both slower and faster failed-to-inhibit saccades (Özyurt, Colonius, & Arndt, 2003; Schall, 1995). We shall argue that the decisions involved in countermanding, often invoked to support threshold ideas, can be best understood in terms of dynamic instabilities.

Finally, we shall combine our understanding of the role of both metrics and time for saccadic decision making by simulating the fusion–selection transition in gap-step-overlap paradigms. We predict that the pattern of early fusion–late selection will be observed when early saccades are generated by a gap, late by an overlap fixation condition. If confirmed experimentally, this prediction would provide evidence that processes of selecting and of initiating saccades evolve in parallel and dynamically.

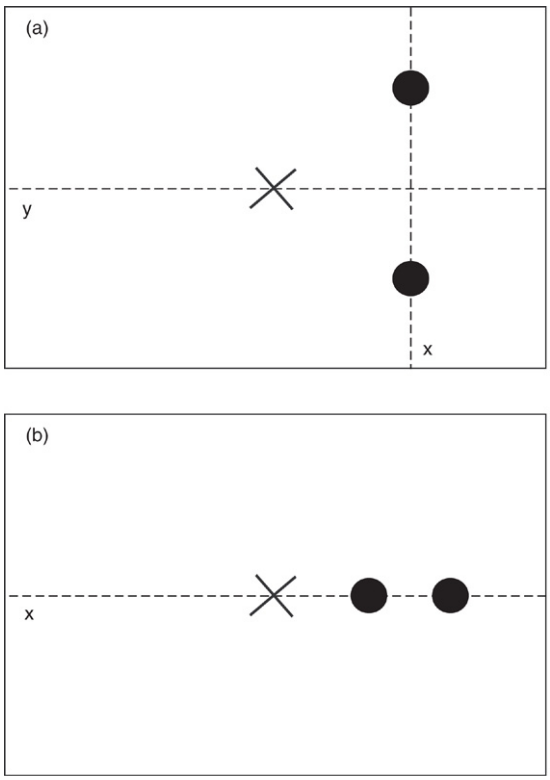

Fig. 1. Psychophysical set-up for double-target stimuli in direction (a) or eccentricity paradigms (b). In direction paradigms, the visual targets (black dots) lie on an imaginary line (vertical here), that is offset against the initial fixation point (cross). The whole arrangement may also be rotated by ±90°. We denote the dimension separating different visual targets by x, and the dimension separating the fixation position from the line of visual targets by y (see dashed lines). In the eccentricity paradigm, fixation signal and visual targets all lie on the same imaginary line (horizontal here). We denote the associated dimension by x.

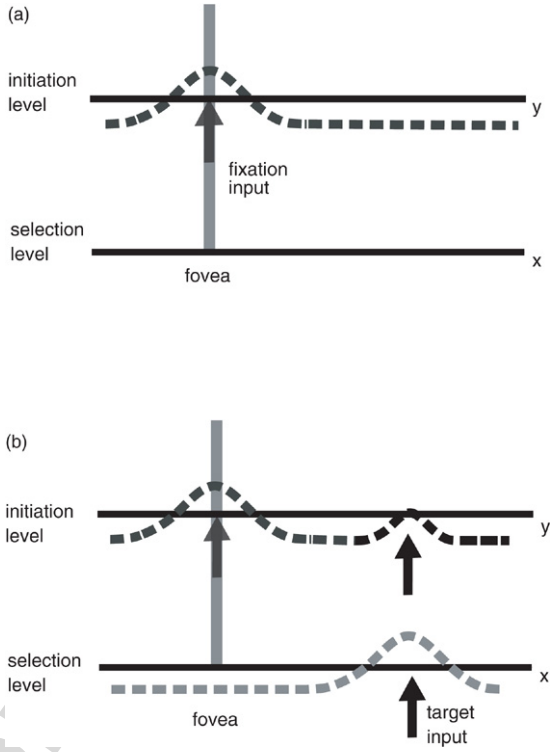

Fig. 2. The Dynamic Field Model of saccadic decision making consists of an initiation level and a selection level. During the fixation period, a single peak of activation in the initiation level at the foveal position reflects the active state of fixation. (a) In the absence of a visual target, activation is negative at the selection level, and the fixation peak remains stable. (b) At target onset, input to the selection level generates a self-stabilized activation peak there, which provides extra-foveal input to the initiation level, and competes with the fixation peak and ultimately wins, inducing a movement-related peak in that level.

2. Model

Information about upcoming movements is represented by distributions of population activation in cortical structures such as the frontal eye fields and subcortical structures such as the superior colliculus. When distributions of population activation are characterized by a strong overlap between information coded by neighboring neurons with similar tuning curves information processing in such neural networks can be described by continuous neural fields. This approximation was first proposed based on the anatomy of cortical areas by Amari (1972, 1977) and Wilson and Cowan (1973). The link to population coding has been established more recently (Bastian, Schöner, & Riehle, 2003; Erlhagen, Bastian, Jancke, Riehle, & Schöner, 1999; Jancke et al., 1999). We follow the mathematical formalization by Amari and Arbib (1977) and the conceptual framework of Dynamic Field Theory by Erlhagen and Schöner (2002), Kopecz and Schöner (1995) and Schöner et al. (1997), which we briefly review now by describing how the model of the selection system is constructed.

The first step is to define the metric dimensions that span the space of possible eye movements. These are clearly the two dimensions of visual space in retinal coordinates, representing possible saccadic end-points. To simplify the modelling, we exploit that typical paradigms probing saccadic decision making sample this space in specific ways. Fig. 1 illustrates the direction (top) and eccentricity (bottom) paradigms. In the first, the initial fixation lies off an imaginary line, along which two targets are presented. For selection, it is sufficient in this case to model representations along the dimension, x, separating different possible targets (vertical in the figure). For initiation, it is sufficient to model representations that separate initial fixation from the shared component of the two visual targets along a perpendicular dimension, y (horizontal in the figure). In eccentricity paradigms, initial fixation position and visual targets are all lined up, so the same linear dimension, x, can be used for both initiation and selection processes. To generalize this account to two dimensions of selection and initiation does not require any new mathematics, but is numerically considerably more costly (Erlhagen & Schöner, 2002; Wilimzig & Schöner, 2005).

An activation variable $u(x)$ is assigned to each site along this dimension. The level of activation $u(x)$ represents the degree to which this particular value is currently specified. High levels of activation drive neuronal processes down-stream from the activation field, low levels of activation do not. When, for instance, no saccadic end-point is specified in the absence of sensory information, the field is flat at negative levels $u(x) = \text{constant} < 0$ (Fig. 2(a)). A localized peak of

activation represents the planned saccadic end-point, specified by the location of the peak in the field (Fig. 2(b)). The activation field can therefore be viewed as a mean field description of neuronal activity based on the space code principle of neurophysiology, in which the location of neurons within a network determines what the neuron represents and its firing rate represents the extent to which the information is present (Erlhagen et al. (1999), textbook treatment Dayan and Abbott (2001)). In this picture, mapping activation patterns of the model onto actual neural activities in the central nervous system requires a normalization in which positive levels of activation correspond to high levels of firing in populations of neurons while negative levels of activation correspond to lower levels of firing than the spontaneous firing rate.

The activation field evolves continuously in time as described by a dynamical system:

$$\tau \dot{u}(x,t) = -u(x,t) + h + S(x,t) \ldots . \tag{1}$$

The rate of change, $\dot{u}(x,t)$ is proportional to $-u(x,t)$. The proportionality constant is the time scale, τ, of the relaxation process with which the field moves toward stable states. This models mechanisms for stability inherent in neuronal function at the cellular level, which the population level inherits (see discussion in Hock, Schöner, and Giese (2003) and Wilson (1999)). The parameter, h, acts as the resting level of the field, that is, the level to which the field relaxes without input or interaction. Unspecific factors, such as go- or stop-signals may be modelled by changing the level of this parameter (Erlhagen & Schöner, 2002). Specific information, by contrast, takes the form of localized inputs, $S(x,t)$, which model visual stimuli, $S_i(t)$, specifying saccadic target locations, x_i, here in the form of gaussians:

$$S(x,t) = \sum_i S_i(t) \exp\left\{-\frac{(x-x_i)^2}{2\sigma^2}\right\}. \tag{2}$$

Up to this point, the field is essentially a simple (if spatially continuous) neural network responding to positive input, $S(x,t)$, by building up activation at matching locations. Dynamic Field Theory postulates, by contrast, that neuronal interactions within the field (maybe mediated by recurrent loops through other structures), may dominate and effectively turn on or off input sensitivity. In contrast to previous models of saccadic programming (Kopecz & Schöner, 1995), we model interaction as occurring in a two-layer structure (Amari & Arbib, 1977). The first layer, $u(x,t)$, is projected excitatorily onto efferent structures and receives afferent localized input. The second layer, $v(t)$, projects inhibitorily onto the first layer, from which it receives excitatory input. This layer thus mediates inhibitory interaction within the excitatory layer (see Fig. 3). Unlike the approximate one-layer model used earlier (and introduced by Amari (1977)), this formulation is consistent with the fact that neurons in the central nervous systems have only one type of synaptic projections, either excitatory or inhibitory (sometime referred to as Dale's law, see Dayan and Abbott (2001)).

The spatial structure of interaction is described by local excitation, global inhibition. This means that excitatory interaction falls off with increasing distance between field sites:

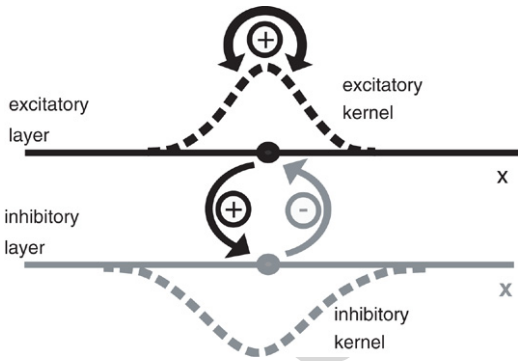

Fig. 3. Interaction as mediated by the two-layer structure. Excitatory interaction takes place within the excitatory layer. Positive activation there drives the inhibitory layer, which in turn inhibits the excitatory layer.

$$w_u(x-x') = k_u \exp\left\{-\frac{(x-x')^2}{2\sigma_u^2}\right\} \tag{3}$$

while the inhibitory layer receives homogeneous input from the excitatory layer, w_v = constant. As a result of this approximation, the inhibitory layer can be replaced by a single, inhibitory interneuron.

Only sufficiently activated field sites contribute to interaction. The sigmoidal threshold function

$$f_i[r] = \frac{a_i}{1+\exp[-\beta_i r/a_i]} \tag{4}$$

($i = u$ or v) makes the neuronal dynamics non-linear.

The dynamics of the selection system thus reads:

$$\begin{aligned}\tau_{u,\mathrm{sel}}\dot{u}_{\mathrm{sel}}(x,t) &= -u_{\mathrm{sel}}(x,t) \\ &\quad + \int w_u(x-x') f_u[u_{\mathrm{sel}}(x',t)]\mathrm{d}x' \\ &\quad - f_v[v_{\mathrm{sel}}(t)] + S_{\mathrm{tar}}(x,t) + h_{u,\mathrm{sel}} \\ \tau_v \dot{v}_{\mathrm{sel}}(t) &= -v_{\mathrm{sel}}(t) + \int w_v f_u[u_{\mathrm{sel}}(x',t)]\mathrm{d}x' + h_{v,\mathrm{sel}}. \end{aligned} \tag{5}$$

This system receives only extra-foveal input, $S_{\mathrm{tar}}(x,t)$, representing visual targets. In the absence of target information the field is therefore in a stable state of negative activation at the resting level (Fig. 2(a)). When a visual stimulus has induced a localized peak, stabilized by interaction, the selection system provides input to the initiation system (Fig. 2(b)). When go- or stop-signals shift the resting level up or down, such self-stabilized peaks may be enhanced or suppressed.

The dynamics of the initiation level is analogously developed for a one-dimensional cut, y, through visual space that separates the initial fixation location from the saccadic targets. For the direction paradigm, this dimension is identical to x, for the eccentricity paradigm it lies vertical to x (Fig. 1). The dynamics of the initiation level reads:

$$\begin{aligned}\tau_{u,\mathrm{ini}}\dot{u}_{\mathrm{ini}}(y,t) &= -u_{\mathrm{ini}}(y,t) \\ &\quad + \int w_u(y-y') f_u[u_{\mathrm{ini}}(y',t)]\mathrm{d}y' \\ &\quad - f_v[v_{\mathrm{ini}}(t)] + w_{\mathrm{ini,sel}} f_u[u_{\mathrm{ini}}(y,t)] \\ &\quad + S_{\mathrm{fix}}(y,t) + h_{u,\mathrm{ini}} \end{aligned}$$

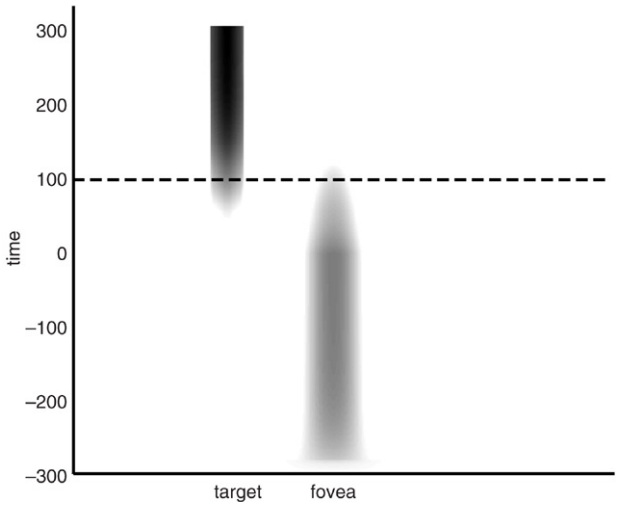

Fig. 4. Time course of activation in the initiation level. Positive activation is depicted in grey scales as a function of retinal position and time. At time = 0 the target is switched on and the fixation input is switched off. Movement initiation according to our criteria occurs at the time marked by the dashed line.

$$\tau_{v,\mathrm{ini}}\dot{v}_{\mathrm{ini}}(t) = -v_{\mathrm{ini}}(t) + \int w_{\mathrm{ini}} f_u[u_{\mathrm{ini}}(y',t)]\mathrm{d}y' + h_{v,\mathrm{ini}}. \quad (6)$$

This field receives only foveal input, $S_{\mathrm{fix}}(y,t)$, while visual structure at other locations does not directly generate input. Instead, extra-foveal input is provided from the selection field. In the presence of a fixation signal, there is typically a self-stabilized peak at the origin representing a fixation state (both panels of Fig. 2). When the selection field provides extra-foveal input, competition between activation at the fovea and at the specified location leads to the suppression of the fixation peak and the generation of a peak at the specified saccadic end-point (see Fig. 4). If we map positive levels of activation onto elevated firing rates and negative levels of activation onto lower than spontaneous firing rates, then this mechanism in the model matches neurophysiological results, which show that saccade initiation correlates with an increased discharge rate in saccade-related neurons and at the same time with a decreased discharge rate in fixation neurons (Dorris and Munoz (1998) and Dorris, Pare, and Munoz (1997), see review by Schall (2004a)).

Stochastic variability is represented in the model through fluctuations of the level of activation. These are caused by stochastic inputs, modelled in the simplest form as independent gaussian white noise at each field site (with zero mean $\langle\xi(x,t)\rangle = 0$ and variance, q: $\langle\xi(x,t)\xi(x',t')\rangle = q\delta(t-t')\delta(x-x')$. These approximate the influence of other neuronal processes, unrelated to the task as well as intrinsic neuronal variability. Spatially uncorrelated noise is the weakest possible stochastic perturbation. To model variance in the countermanding paradigm we introduce variability from trial-to-trial in the strength of fixation inputs, which models random variations of unspecific factors such as attention or pretrial effects.

Finally, we need to specify how activation patterns in the model drive saccadic eye movements. In earlier work, we showed how a self-stabilized peak of saccade-related activation may set a new stable state for the motor control system of the eyes (Kopecz & Schöner, 1995). Although the details were not realistic, the conceptual issue was that the transition from a peak-less state to a state with a self-stabilized peak may induce a related transition in the motor control system from a fixation state to a movement state. In reality, the motor control system has considerably more complex structure, including horizontal and vertical burst generators which are transiently activated (review, Lefèvre, Quaia, and Optican (1998), Robinson (1986)). Here we seek a way to simplify the problem by replacing the entire motor control system with a simple rule that determines the time of initiation of a saccade as well as its metrics. Saccade latency was determined as the time interval from stimulus presentation to the moment in time when the activation within the fixation peak

$$F(t) = \int_{-\sigma_{\mathrm{fix}}}^{\sigma_{\mathrm{fix}}} f_u[u_{\mathrm{ini}}(y',t)]\mathrm{d}y' \quad (7)$$

fell below a criterion level F_{thresh}. To this time we added 70 ms to account for an estimated 40 ms afferent and 30 ms efferent delay (e.g. Smit and van Gisbergen (1989)). The metrics of the saccades were characterized by the center of gravity of the activation distribution in the selection field:

$$x_c = \int_{R'} x' f_u[u(x')]\mathrm{d}x' \Big/ \int_{R'} f_u[u(x')]\mathrm{d}x'. \quad (8)$$

Thus, the read out of saccadic end-point is done within the selection level while the fixation level solves the release of the fixation activity and the building of a new activation peak at the location of the target within the coordinates of the fixation level. To decide whether movement cancellation was successful in countermanding trials we observed whether a peak was generated at the target site of the field by looking for positive activation there.

3. Results

3.1. Overcoming fixation and countermanding

In the model, a saccade is initiated when extra-foveal activation in the initiation level induced by input from the selection level inhibits the fixation peak. How much time this takes depends on the amount of foveal fixation activation, which in turn, depends on the fixation stimulus. This can be illustrated by simulating the gap-step-overlap paradigm (Fig. 5), in which the fixation signal is extinguished either before (gap), at the same time (step), or after (overlap) the visual target appears. The mean latency of saccade initiation increases from gap to step to overlap conditions, matching the experimentally established effect (panel (b) of the figure) and reproducing Kopecz's (1995) earlier modelling results. While Kopecz did not model variance, the stochastic inputs included in our model enables us to generate histograms of latencies (panel (a) of the figure) that can be compared to experimental assessments of variability (Gezeck & Timmer, 1998). In the model, the compact, sharp histograms in the gap and step condition are in contrast with the broader, noisier histogram in the

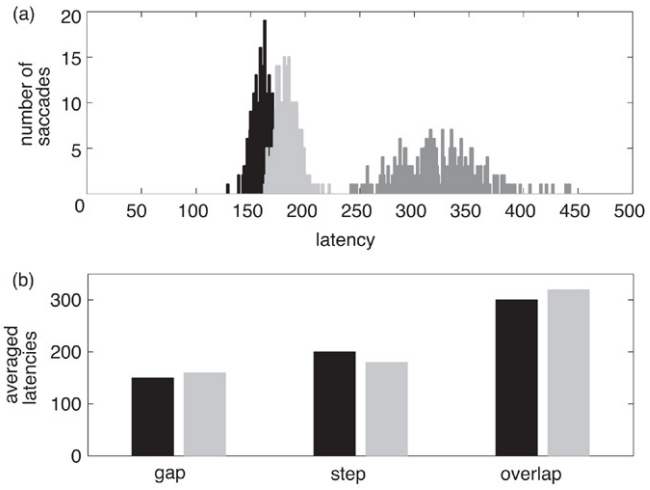

Fig. 5. Latencies for single target trials in the gap-step-overlap paradigm. (a) Histogram obtained from the model under gap (black), step (light grey), and overlap (dark grey) conditions. (b) Mean latencies predicted by the model (grey bars) are compared to averages from experimental studies (black bars) (Becker, 1989; Kopecz, 1995).

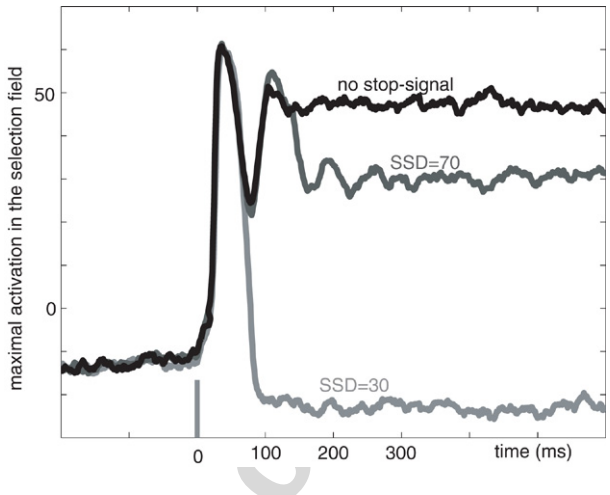

Fig. 6. Modelling the stopping process in the countermanding paradigm. The negative shift of the resting level suppresses activation in the selection field at the location of the visual target when SSD is long (70 ms), but not when SSD is short (30 ms). In either case, activation is lowered compared to trials without stop-signal. Trials are aligned by the time the visual target is presented (grey bar).

overlap condition. This matches the typical patterns found in experiment. Moreover, we were able to match the experimental range within which latencies vary (from 140–160 ms for gap saccades up to 300 ms for overlap saccades).

The countermanding paradigm (Hanes & Schall, 1995) provides access to the fine structure of the initiation processes. In this paradigm, typically a visual target is presented at the same time at which the fixation stimulus is extinguished. In a certain percentage of trials, the fixation stimulus reappears after a variable time delay, the *stop-signal delay* (SSD). This indicates to the participant that the saccade to the visual target must be suppressed. In the model, this instruction is captured by lowering the resting level, h_{sel}, of the selection field, which amounts to injecting global inhibition into the field. This may prevent the generation of a self-stabilized peak in that field and thus cancel the saccadic movement plan. Essentially, the field may be pushed below the detection instability, at which the target input becomes sufficient to induce a self-stabilized peak (see Bicho, Mallet, and Schöner (2000) for a discussion of these instabilities).

Whether or not the h-shift is sufficient for cancelling the movement plan depends on the timing of the stop-signal and on the amount of fixation activation. For small SSDs, the peak in the selection field has not grown enough to self-stabilize and the h-shift does succeed in cancelling the movement plan (see Fig. 6, SSD of 30 ms). For larger SSDs, the peak is capable of self-stabilizing, although at a lower level of activation (SSD of 70 ms in the figure). In experiment, either outcome occurs with some probability, which depends on the SSD. The stop function, that is, the probability that a saccade is generated even though a stop signal was presented, increases with increasing SSD (Fig. 7, Hanes & Schall, 1995). This probability can be estimated in the model by taking into account stochastic perturbations, here in the amplitude of fixation input. The match between model and experiment is excellent (Fig. 7).

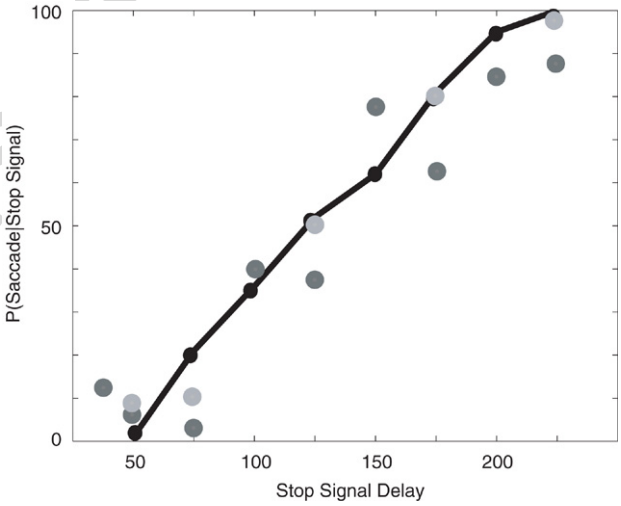

Fig. 7. Stop functions as observed experimentally (light and dark grey dots represent data from two monkeys as published in Hanes and Schall (1995)) and as predicted from the DFT model (black dots and black line). To fit the experimental results, the initial fixation strength and the strength of the countermanding signal were adjusted.

The role of the level of activation in the fixation peak is illustrated in Fig. 8. That level is varied in the simulations, by changing the strength of the visual fixation signal. Each column shows on top a simulation in which activation in the fixation peak (solid line) was just large enough to cancel the movement plan (dashed line) and on bottom a simulation in which fixation activation was just too small to cancel the movement plan. The cross-over between these regimes occurs at lower levels of fixation activation for small SSD (left column) than for large SSD (right column). To our knowledge, there is no direct experimental test of this account. One way such a test could be generated is by manipulating the attentional load. Because attention increases the activity of task-relevant neurons (Reynolds & Chelazzi, 2004; Treue, 2003), increasing

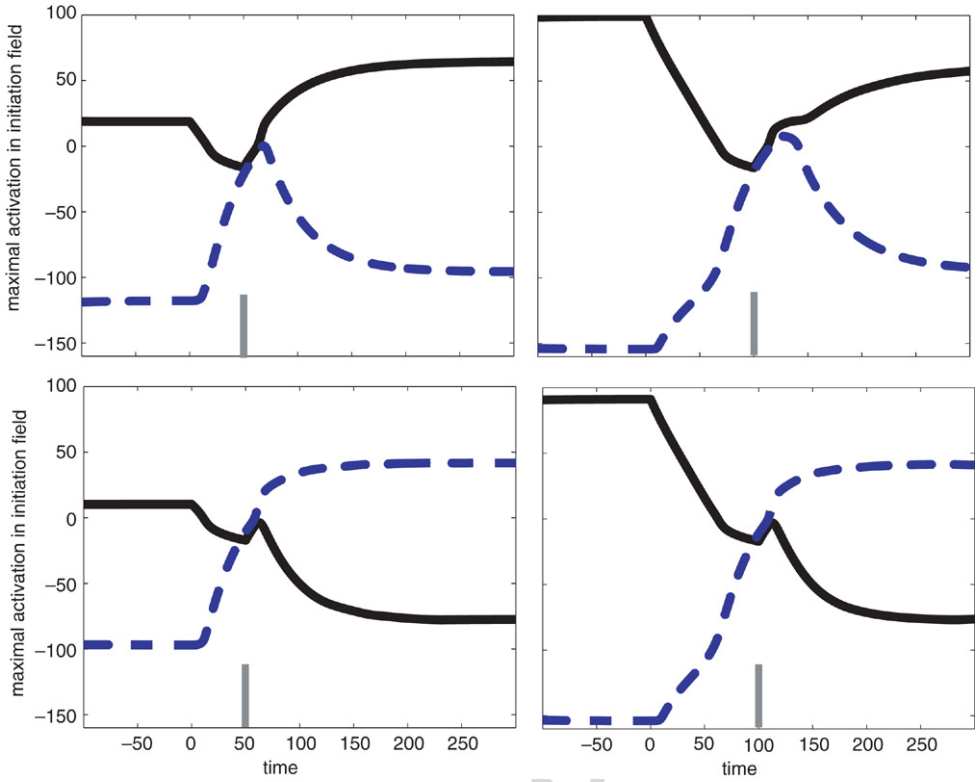

Fig. 8. The amount of fixation related activity (solid lines) determines whether a saccade can (top row, higher level of fixation related activity) or cannot (bottom row, lower level of fixation related activity) be inhibited at a given stop-signal delay (SSD) (short = 50 ms left column; long = 100 ms right column; see grey marker on time axis). The dashed lines is peak activation at the location of the planned movement target, the solid line is peak activation in the fixation peak. Different levels of fixation related activity were obtained by drawing the fixation strength, s_{fix}, from a distribution (gaussian, mean = 70, SD = 40) of which four examples were selected here for illustration.

the intention to the fixation site (e.g., by imposing a secondary task there) would lead to higher stopping probability.

The time courses of activation in Fig. 8 may be directly compared to neuronal data, again based on mapping high levels of activation to high firing rates, low levels of activation to lower than spontaneous firing rates. Note how in successful stop-trials (top), fixation activity first falls, but then recovers, while movement related activation first rises and then falls again. In failure trials (bottom), by contrast, activation representing movement plans continues to grow (with a little hiccup when the stop-signal hits), while fixation activity falls, then grows briefly, then falls again. Neurophysiological studies have indeed shown that when saccades are successfully cancelled, movement related neurons show an initial increase followed by a rapid decrease in firing rate. Fixation neurons may decrease their firing rate initially, then generate a rapid increase of firing rate. This was observed both for neurons in the frontal eye field (FEF) (Hanes, Patterson, & Schall, 1998) as well as in superior colliculus (Pare and Hanes (2003), for a review see Schall (2004a)).

Another signature of the competition between fixation activity and movement-related activity can be obtained when examining latencies in failed stop trials. There are two factors at play here. First, failure to inhibit a saccade is more likely for saccades that would have been faster than average had a stop signal not been given. In the model, such shorter latencies may arise due to weaker than average fixation signals, which puts the saccade promoting activation at a competitive advantage. Because the stop signal thus cuts off the slower part of the latency histogram, such failed-to-inhibit saccades have shorter latency than regular saccades. With increasing SSD, less of the histogram is cut off and the mean latency of the failed-to-inhibit saccades approaches that of regular saccades as illustrated in Fig. 9. For small SSDs, failed-to-inhibit saccades are rare because the saccade-related activation has not yet had time to build. This frequency increases with increasing SSD.

The second factor is that the renewed input to the fixation system provided by the stop-signal strengthens the fixation-related activation in the competition with the saccade-related activation. This tends to slow down the initiation of a saccade, leading to longer latency of failed-to-inhibit saccades. Only at very short SSDs does this inhibition have a noticeable effect on latency, because only then do go-signal and countermanding-signal effectively compete. At such short SSDs, on the other hand, failures to inhibit a saccade are very rare. In the model, a few such instances arise from variability in countermanding strength as shown in Fig. 10.

These simulations thus provide an explanation of contradictory results in the literature. Hanes and Schall (1995) had reported evidence for faster than regular failed-to-inhibit saccades (see also Curtis, Cole, Rao, and D'Esposito (2005)), whose latencies increased with increasing SSD to normal. At very small SSD (25 ms), a small number of very slow errors were reported, however. Such rare, slow errors were also reported by Özyurt

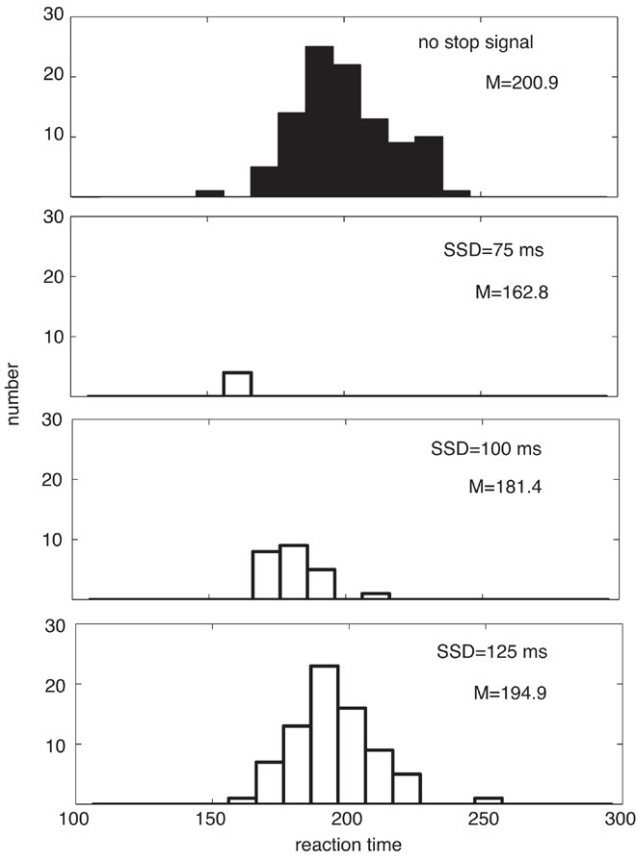

Fig. 9. Histograms of saccade latencies obtained from simulations when no stop signal is given (top) compared to latencies of failed-to-inhibit saccades at stop signal delay 75, 100, and 125 ms. The frequency of failed-to-inhibit saccades increases with increasing SSD as does the mean latency, which matches at SSD = 125 ms the mean latency of regular saccades. Noise in the initial activation level of the fixation peak was sufficient to generate these events.

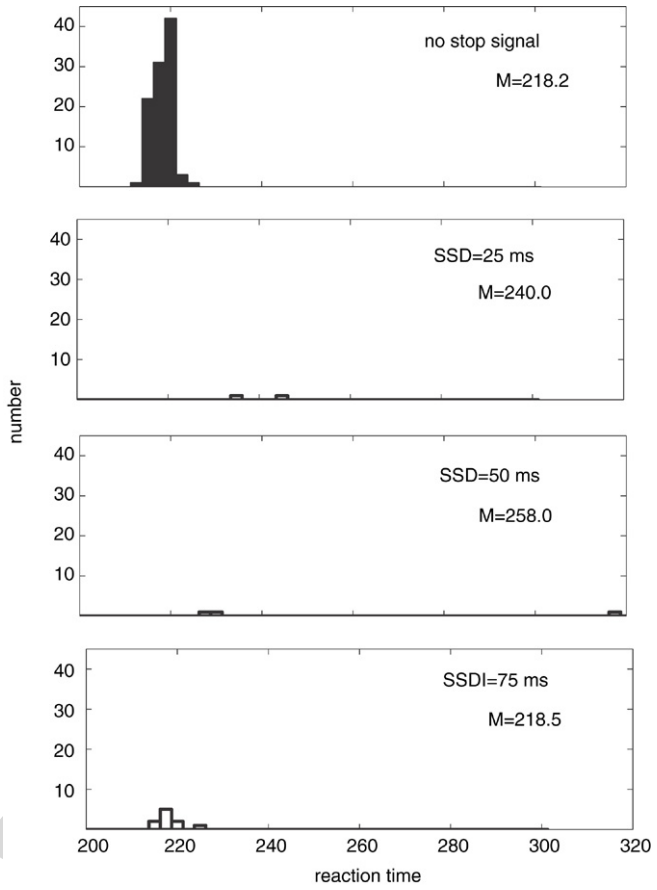

Fig. 10. Chance events induced by stochastic levels of the strength of the countermanding command give rise to rare events, in which saccades fail to be inhibited at very short SSDs of 25, 50 and 75 ms. Histograms of the associated saccade latencies are compared to those obtained in the absence of a stop signal. Latencies of these rare events are longer than regular latencies. This difference decreases with increasing SSD, disappearing at approximately 75 ms.

et al. (2003), who concluded that the race model was violated in these instances.

3.2. Selection

We now move beyond the single target case and examine how selection of one target in double target or distractor paradigms is accounted for by neuronal interaction.

In their simpler model, Kopecz and Schöner (1995) showed how a transition occurs from an fusion to a selection solution. Metrically close visual targets lead to overlapping inputs to the selection field. Local excitatory interaction may then fuse these two inputs and generate a single, self-stabilized peak at the averaged location of the two targets (top panel of Fig. 11). When visual targets are moved further apart from each other, the bimodal input to the selection field leads to a bistable state in which a peak may arise localized either over one target or over the other (bottom panel of Fig. 11). When the targets are lined up symmetrically to the initial fixation point in the direction paradigm (Fig. 1), then selection is based on stochastic fluctuations in the input or potentially from differences in initial activation from previous trials. Any asymmetry, in which one target is closer to the initial fixation point, is brighter, more probable etc., leads to different input strengths of the two targets and thus biases selection as observed in experiment. Similarly, in the eccentricity paradigm, the target closer to the initial fixation position is typically selected, which may be understood in the model based on inhomogeneous input strengths, broader and stronger for targets closer to the fovea.

The transition from monostable to bistable activation patterns with metric distance between targets is a dynamical instability, the fusion–selection instability (discussed in Bicho et al. (2000)). Averaging is not due to limited resolution of the neural map of saccadic end-points: In separate, single target trials, the two locations can be perfectly realized even for metrically close targets (Fig. 12).

Kopecz and Schöner (1995) did not model or analyze latencies. Fig. 13 shows histograms of latencies derived from our present model. Variability again is induced by stochastic inputs to the field. Note how latencies for single-target and averaging saccades are equally short (actually even slightly shorter for averaging saccades), while latencies for selection saccades are longer. This slowing down for selection is due to the inhibitory interaction between the two potential peaks in the bistable mode, which holds up the building of a movement-related peak. The slight acceleration of averaging

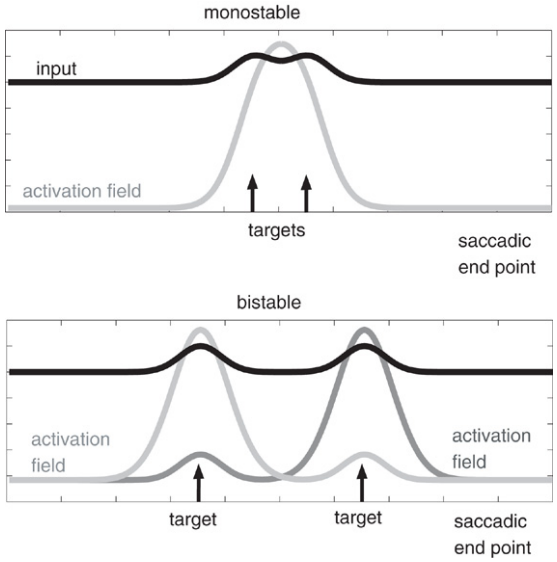

Fig. 11. The mechanism for the transition from averaging to selection with increasing metric distance of targets is illustrated by plotting the inputs to the selection field (black) and the resultant stable activation patterns (light and dark grey). For metrically close targets, excitatory interaction leads to fusion, a monostable peak centered over the averaged target location (top). For metrically distant targets, two stable patterns are possible. In each case, a peak is positioned over one target only.

saccades comes from short-distance excitatory interaction of course.

Experimentally, it is well known that averaging saccades tend to be fast, equally or more so than saccades to single targets (Chou, Sommer, & Schiller, 1999; Ottes et al., 1984; Ottes, van Gisbergen, & Eggermont, 1985). Presenting a second target leads to an increase in reaction time (Walker, Deubel, Schneider, & Findlay, 1997; Walker, Kentridge, & Findlay, 1995). This is true for a wide range of conditions, even when the second target is irrelevant and irrespective of whether the required direction of the saccade is known beforehand (overview by Findlay and Gilchrist (2003)).

Can we turn around this logic and ask, if fast saccades tend to do averaging while slow saccades tend to do selection? To answer this question we examine the time course of activation in the selection level when presented with two visual targets. Three phases can be distinguished. In a first phase, lasting only a few dozen milliseconds for realistic parameter values, sensory input drives up activation at the locations corresponding to the two visual targets. Second, as activation reaches positive levels, excitatory interaction begins to act. Finally, the inhibitory layer begins to be sufficiently activated to mediate inhibitory interaction effects.

Which phase is functionally most relevant depends on the metric distance between targets. For metrically close targets, an initially broad, input-driven activation pattern is sharpened and fused by excitatory interaction which leads to the merging of the two inputs to an averaging response (right panel of Fig. 14), largely unaffected by inhibitory interaction. For metrically distant targets, an initially bimodal, input-driven pattern is decisively reshaped by inhibition, which selects one of the two sites, leading to a single peak positioned over one of the two targets (left panel of Fig. 14).

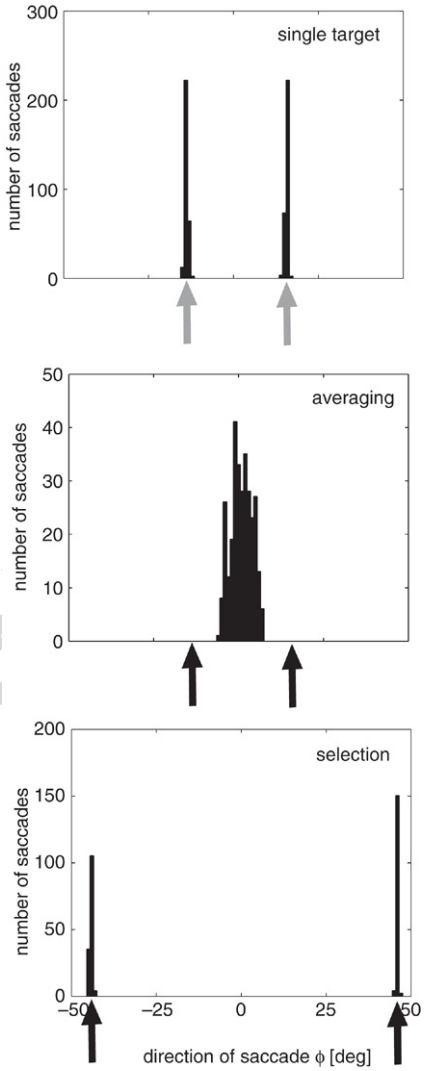

Fig. 12. Histograms of saccadic end-points of responses toward single and double target stimuli as derived from the DFT model. While for metrically distant targets saccades select one of the two target locations (bottom), for metrically close targets saccades are directed toward the center between the two targets (middle). This is not due to resolution problems or related artifacts as accuracy is sufficiently high if the targets are presented individually at either of the two locations used in the averaging condition (top). (Black arrows indicate target locations (middle and bottom); grey bars indicate target location in 50% of the trials for the single target condition; for visualization we use different scales for the ordinate).

The most interesting case arises at the cross-over between these two limit cases (Fig. 15). At that critical distance between the two targets, the earliest activation patterns (from 10 and 25 ms in the left panel of the figure) are input-driven and bimodal. During an intermediate time interval (80 ms in the figure), the now dominant excitatory interaction leads to fusion of the two peaks. Inhibition takes even longer to take effect, shifting the fused peak to one of the two target locations and thus bringing about selection (right panel, selection completed by about 190 ms, dashed line).

The answer is therefore a “yes”: in the cross-over region between averaging and selection, time should matter. If there is some way the motor response can be triggered earlier then

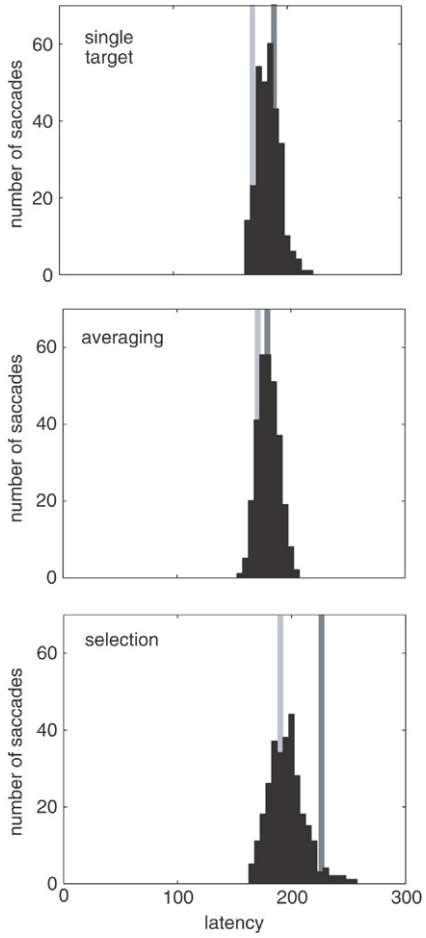

Fig. 13. Histograms of latencies for single target and double target stimuli as generated from the DFT model together with experimental mean values (grey vertical lines) reported for two participants by Ottes et al. (1985). Selection saccades are slower than averaging and than single target saccades. Averaging is similarly fast (even slightly faster) than single target saccades. Note how the means for the two participants are more discrepant in the selection than in the other conditions, which is consistent with the broader distribution generated by the model in that case.

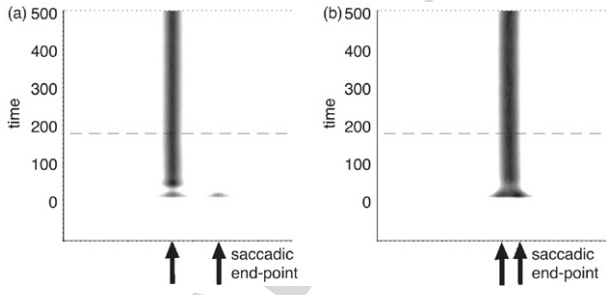

Fig. 14. Time courses of activation in the selection field for wide (left) and narrow (right) spacing of two visual targets (arrows). The activation level is coded by grey scale, darker indicates higher activation. Dashed lines mark time of movement onset according to criteria.

averaging should be favored. If motor responses are held up longer, selection should be favored.

One way the latency of saccades can be manipulated is through the speed–accuracy trade-off. If participants are encouraged to respond as quickly as possible with less regard

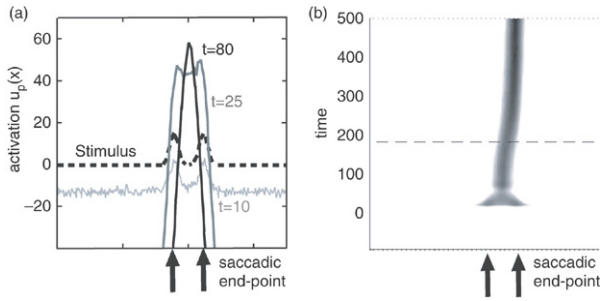

Fig. 15. Time courses of activation in the selection field for the critical spacing of two visual targets (arrows), at which cross-over from averaging to selection occurs. On the left, the activation profiles are shown as snap-shots during the first 80 ms after stimulus presentation (stimulus as a dashed line). On the right, activation is shown with a grey scale on the larger time scale, on which inhibition intervenes.

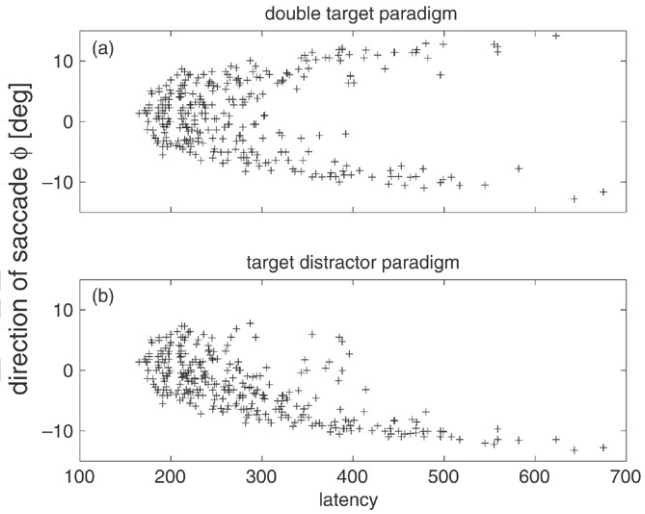

Fig. 16. Speed–accuracy trade off induced by varying fixation strength. In the double target paradigm (top) saccadic end-points (small crosses) shift from a broad distribution around the center between the two targets for low levels of fixation input, leading to short latencies, toward the selection of one of the two targets for stronger levels of fixation input leading to larger latencies. In the target–distractor paradigm (bottom), early saccades average, later saccades land at the correct target.

to precision, short latencies ensue. If accuracy is emphasized, latencies are longer and their distribution is broader (Ottes et al., 1985). The speed–accuracy trade-off can be mimicked in the model by strengthening fixation activation when accuracy is emphasized, as this shifts the balance between fixation and saccade initiation to later times, when the selection field has had more time to elaborate the metrics of the saccade. When fixation input is strengthened, the shift from averaging to selection with increasing latency can be directly observed in the model (Fig. 16). For a double target paradigm one of the two targets is selected for sufficiently large latencies, leading to a bimodal distribution. The target–distractor paradigm is modelled by turning one of the targets off after 100 ms (mimicking the effect of other neural processes that discriminate the target from the distractor based on other features like color). In this case, the longer latency saccades

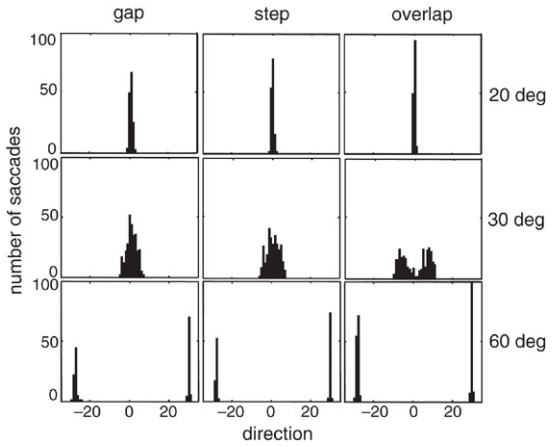

Fig. 17. Combination of the gap-step-overlap paradigm with a double target paradigm. For metrically close targets, saccades always are in the averaging mode (top row). For metrically distant targets, saccades are always in the selection mode (bottom row). For a critical metric distance the model predicts a shift from averaging in the gap condition to selection in the overlap condition.

go to the correct target, reducing the metric bias toward the distractor.

These results of the model closely match the speed–accuracy trade-offs observed in human participants by Ottes et al. (1985). Moreover, based only on the spontaneous variability of saccadic latency in behavioral studies with monkeys, Edelman and Keller (1998) have reported a very similar pattern of early averaging, late selection.

3.3. The interaction between selection and initiation

Another way how the time course of selection may be observed, is by varying latency through the fixation conditions. This can be done in the gap-step-overlap paradigm. The model predicts that for a double target paradigm near the cross-over distance between averaging and selection, the fixation condition determines which of the two will be observed (Fig. 17). Saccades speeded by the gap condition will tend to go to the averaged target location. Saccades delayed by the overlap condition will tend to select one of the target locations.

Note that this prediction is based on the assumption that the initiation and selection system evolve continuously and in parallel, and that their coupling is effective throughout that evolution. Thus, the extent to which the selection level elaborates saccadic plan depends also on the extent to which the initiation level resists the initiation of a new saccade. To our knowledge, this connection between fixation conditions and selection has not been probed empirically. Experimental confirmation of our prediction would provide support not only for this particular model, but also for the continuous and parallel processing of the two decisions involved in initiating a saccade, when and where.

4. Discussion

4.1. Relationship of the model to neurophysiology

Superior colliculus (SC) is the brain structure whose link to saccadic planning and saccade initiation has been most extensively documented. In fact, it is one of the brain areas best linked to function (e.g. Sparks and Groh (1995)). Even so, the functional interpretation of SC has more recently come under some reevaluation, in which SC is being looked at as a motor map of saccadic *goals* more so than of the movements necessary to reach these goals (review Krauzlis, Liston, and Carello (2004)). Evidence comes from catch-up saccades (Keller, Gandhi, & Weir, 2000), memory-guided saccades (Stanford & Sparks, 1994) and from the fact that SC activity is related to gaze movement, that is, combined eye–head movement, while the detailed activation patterns of head or eye muscles depend on conditions (Freedman & Sparks, 1997).

This is, of course, exactly the coding we have assumed in the model. Both the selection and the initiation field code represent the saccadic end-point. Sensory information is interpreted in terms of the saccadic end-point it specifies and localized peaks of activation are interpreted in terms of which saccadic end-point is planned. What both dynamic activation fields represent matches, therefore, what SC may represent.

A more specific mapping emerges from the observation that SC projects to so-called “omnipause” neurons known to be the final gatekeepers for saccades (Buttner-Ennever, Horn, Henn, & Cohen, 1999). SC thus contributes to making the decision to initiate a saccade. Specifically, certain neurons in rostral SC representing the central visual field are active during fixation (Munoz & Wurtz, 1993). Their activity decreases after the offset of a fixation stimulus (Dorris & Munoz, 1995; Dorris et al., 1997). Within a conceptual framework in which SC was hypothesized to support independent motor plans for fixation and for saccade generation, which compete via short-range excitation and long-range inhibition (Munoz & Fecteau, 2002), these neurons were considered “fixation neurons”. As Krauzlis et al. (2004) point out, the label per se may be misleading as these neurons do not seem to code for the independent motor plan of fixation but may represent locations on or about the fovea. This view is supported by the fact that these neurons are also active during pursuit and for small saccades (Krauzlis, Basso, & Wurtz, 1997, 2000).

This interpretation suggests that our initiation level may describe this role of SC in deciding the initiation of a saccade. The peak at the fovea is, in effect, fixation activation which competes with activation at extra-foveal sites. The outcome of that competition determines whether a saccade is initiated or not. The fixation peak may be self-stabilized in the model, so may persist even when the fixation stimulus is removed. This is consistent with the neurophysiological fact that activity within the rostral SC is dependent on visual input, but persists in the absence of stimulation (Krauzlis, 2001). Moreover, in the “gap” condition, activity in the rostral SC may decrease for some neurons during the gap (Dorris & Munoz, 1995; Krauzlis, 2003), consistent with what happens in the model as input to the self-stabilized fixation peak is removed.

If this mapping is right, then why do we need a second level, the “selection” level? There were basic reasons that pushed us to separate functionally the decision to initiate a saccade and the decision which saccadic target to select. First, it was quite difficult to balance the various forms of competition

so that the initiation of a saccade would occur even when many targets competed for selection. Fundamentally, the rules of competition are different for the two kinds of decisions. Second, the time scales of the selection and the initiation processes turned out to be quite different. To account for the temporal evolution of selection, we were led to postulate a fast neuronal dynamics. This is because the process of selecting through competition from two or more field sites that receive similar amounts of inputs, slows down the evolution of activity considerably. By contrast, the competition between the fixation peak and the movement-related activity involved in the decision to initiate a saccade is rigged: fixation is routinely overcome and movement wins. This imposes the reverse constraint: the decision making is too fast, so that to obtain the strong modulation of latency generated by gap-step-overlap conditions we needed to postulate a relatively slower dynamics at the initiation level.

There are hints in the neurophysiology of SC at such different time scales. Munoz and Wurtz (1995) characterized burst as different from build-up neurons. The activity of *buildup* neurons increases gradually in the preparation of a saccade, peaking in activation just before movement onset. *Burst* cells, by contrast, begin to fire just prior to movement onset (for a review see Findlay and Gilchrist (2003) and Schall (1995)). Could there be two subsystems in SC, effectively interacting with different strengths and different time scales, one supporting the competition for selection (the burst cells?), the other supporting the competition for initiation (the build-up cells?)?

One hint that the model is consistent with the neurophysiology of SC is recent experimental evidence linking selection to inhibitory mechanisms. Li and Basso (2005) recorded from neurons in SC while monkeys performed in an overlap paradigm with two saccadic targets which in each case fell into the receptive field of a recorded neuron. In the double-target condition, both targets were visible, and the go-signal specified one of the two targets. In the single-target condition, only one target was visible at a time. Li and Basso found that during the holding period, activation of neurons was reduced in the double target compared to the single target condition, compatible with the idea of mutual competition between populations representing the two possible targets. Once the go-signal had specified a target, activation levels were identical to single stimulus conditions. The model captures this pattern of neuronal results in detail. Fig. 18 shows time courses of activation at the selection level for two locations in the field, one coding for the saccadic end-point corresponding to one target, the other for the end-point corresponding to the other target. In single stimulus conditions activation rises first when the target is presented, and then further when the go-signal is given (black line). For the double target condition, activation at the two sites rises, but to a lower level, when the targets are presented (grey lines). Once the go-signal is given, the specified target is selected. Its activation level rises to the same level as for the single-target condition. The activation level at the non-specified location decays.

Two other structures contributing to saccadic eye movements may be related to the model. The parietal area LIP and the frontal eye fields (FEF) in the pre-motor frontal cortex are the

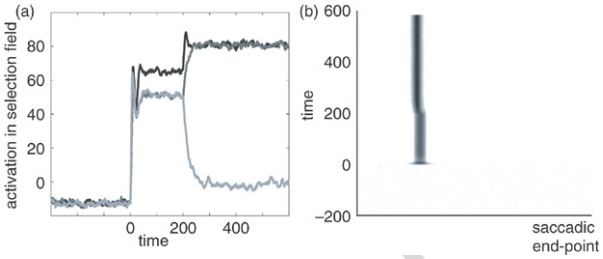

Fig. 18. An account of Li and Basso's (2005) neuronal data in SC. On the left, the time course of activation in the selection field at two field locations representing two visual targets is shown. When a single target is presented (black line), activation rises as the target is presented ($t = 0$) and again as the go-signal is given. When two targets are presented (grey lines), both locations coding for the two targets are activated first to identical, but lower levels compared to the single-target condition. The go-signal leads to selection of one target, and the corresponding activation rises to the same level as in the single-target condition. Activation at the other location decays. The right panel illustrates the spatio-temporal structure of the field in this condition through a grey-scale plot.

two cortical areas most directly linked to saccade generation, for instance, in the sense that microstimulation in either of these areas may lead to overt saccades. LIP has been implicated in generating some of the relevant coordinate transforms involved in generating saccadic eye movements, while also providing signatures of sustained activation, supporting memory for saccadic targets, not unlike the self-stabilized peaks in the field do. We have side-stepped issues of coordinate transforms in this model. It is possible that some of the functionality of LIP contributes to the dynamics postulated in our model. LIP neurons have also been shown to be affected by expected rewards associated with saccadic eye movements (review Glimcher (2003)). The FEF are thought to contribute to control and modulation of eye movements related to processes such as attention. These neural populations might be the source of the kind of modulatory influences we have postulated to understand countermanding. In comparison to SC, the FEF have been looked at as a subsidiary structure (Hanes & Wurtz, 2001), although the combined loss of FEF and superior colliculus impedes an animal to make saccades (Schiller, True, & Conway, 1980).

4.2. Comparison with other models

Our model builds on earlier work by Kopecz (1995); Kopecz and Schöner (1995) and Schöner et al. (1997). Kopecz and Schöner (1995) modelled the transition from fusion to selection, but did not address latencies nor modelled the time course of selection. Kopecz (1995) laid the basis for our treatment of the saccadic initiation, accounting for the gap-step-overlap effect for latency. His treatment did not include variability nor the metric of the planned saccade. In that model, selection and initiation were not yet integrated. Countermanding was addressed in neither model. Trappenberg, Dorris, Munoz, and Klein (2001) also built on the dynamic field framework and covered some of the same ground we do here, including an account for the gap-step-overlap effect similar to Kopecz (1995) and a study of how the presence of distractor slows down latencies depending also on the metric distance

between target and distractor. In addition, they discussed the role of target probability, the potential for express saccades and the role of exogenous factors to account for anti-saccades. Curiously, they operated the dynamic field model in the input-driven regime. As a result, they did not account for selection in the proper sense, that is, when the response is not specified by input. This requires multi-stability and cannot be understood in the input-driven regime. They also did not model the time course of selection nor countermanding.

Another class of models, that is close in spirit to ours, comes from the Grossberg lab (e.g. Grossberg, Roberts, Aguilar, and Bullock (1997)). These models share the dynamical outlook, taking time scales and integration seriously, and are based on similar neuronal principles. The cited model accounts for the gap effect, but does not address selection and its time course. What this and earlier models from that group does, however, is account for the coordinate transforms required as multiple sensory sources are integrated to control saccades. We have neglected this entire aspect, assuming sensory inputs, plans, and motor systems onto which these plans project are all aligned. Because our concepts are fundamentally compatible, accounts for these transforms as in Grossberg et al. (1997) or also Optican (2005) or Xing and Anderson (2000) could be integrated into our framework at the expense of considerably increased model complexity.

Another aspect that we neglected is the process of motor control of the actual saccadic movement. There is a large modelling literature on this, from the classical Robinson (1975) to recent work (e.g., Glasauer (2003); Lefèvre et al. (1998); Optican and Quaia (2002)). The transition from a fixation peak to an extra-foveal saccade related peak of activation is a transition to a new stable state. This makes it possible, in principle, to bring about the required switch in the motor control system in a stable and robust manner (Kopecz & Schöner, 1995). Neglecting the motor control side is thus an acceptable approximation as long as we stop comparing our model to data once a saccade is being initiated. Integrating this next level would be particularly interesting in view of recent work (Port & Wurtz, 2003) that analyzes saccadic trajectories in the presence of distractors. As shown by Walton, Sparks, and Gandhi (2005), these trajectories can be understood as arising from weighted multi-modal activity in SC, not unlike that obtained in our models, if SC is considered upstream from the local feedback loop (see also Arai and Keller (2005)), for an account that allows for SC being part of a feedback loop).

A related issue is how the dynamic field model generalizes to two dimensions. This is not a problem at the level of the fields themselves, which can be formulated in two dimensions while maintaining the same dynamic properties without problem (see Erlhagen and Schöner (2002)). At issue is only, how the two-dimensional coordinates of a saccadic end-point represented in such two-dimensional fields may be transferred to the motor system consisting of burst generators for horizontal and vertical components of a saccade. The typical assumption, that each collicular location projects onto burst generators proportionally to its horizontal or vertical component is consistent with the neuroanatomy of the corresponding neuronal connections (Moschovakis et al., 1998). The projection onto burst generators does not, therefore, present an obstacle to generalization to two dimensions either.

At a more conceptual level, our approach is consistent with the distinction between a "where" and "when" system, first proposed by van Gisbergen et al. (1981) and used by Findlay and Walker (1999) to propose a model in which there are two separate systems for response initiation and for determining the metrics of the response, paralleling the distinction we make here between selection and initiation. More recent physiological work leaves open the perspective, however, that the two systems may form a continuum rather than two discrete systems (Krauzlis et al., 2000).

A very different class of models comes from a tradition in mathematical psychology. These are sequential sampling models of reaction time that can be separated into two categories, accumulator/counter models and diffusion/random walk models (for overviews, see, e.g., Ratcliff and Smith (2004) and Smith and Ratcliff (2004)). The LATER model (Carpenter & Reddi, 2000) has been used to account for saccadic latencies. Recently some attempts have been made to link these kinds of models to neural data Ratcliff, Cherian, and Segraves (2003) and Smith and Ratcliff (2004) and to the concepts of neuronal dynamics (Usher & McClelland, 2001).

The limitations of models that postulate, somewhat abstractly, that evidence is accumulated sequentially, can be illustrated for the countermanding paradigm. Race models (Logan and Cowan, 1984) provide a theoretical account for countermanding by postulating that two independent processes for the GO- and the STOP-process accumulate evidence. Both the assumption of independence and the mapping onto different neurons is not obvious from a neurophysiological point of view (Schall, 2004a). Specifically, on GO-trials saccade-related neurons increase their activation while *at the same time*, fixation neurons decrease their activation (Dorris & Munoz, 1998; Dorris et al., 1997). The reverse is true for successful stop trials (Hanes et al., 1998; Pare & Hanes, 2003), with some evidence for mutual inhibition (Munoz and Istvan (1999); Quaia, Lefevre, and Optican (1999), review by Schall (2004a)). Finally, race models predict that saccadic latencies are faster for failed stop trials than for regular saccades. This is because on faster trials, evidence for the GO processes has an advance, leading to a higher probability of these trials leading to failure to stop. The dynamic field model has provided an alternative account for this observation (Hanes & Schall, 1995), while at the same time explaining rare but reproducible events at short stop signal delay (Hanes & Schall, 1995; Özyurt et al., 2003), in which latencies are longer than regular latencies. This is due to enhanced competition from the strengthened fixation system.

More generally, given that the mechanisms transforming stimulus-related activity into movements is not yet fully understood (Krauzlis et al., 2004), this class of models is based on the concept of a threshold mechanism by which an internal signal, which increases in time during saccadic latency, triggers action when a threshold is reached. The increase of that internal signal may either be linear in time (Carpenter & Reddi, 2000), or be governed by diffusion or counting processes as

in the sequential sampling models reviewed above. Different scenarios exist for how the threshold is defined, ranging from thresholds for the absolute level of activation to criterion differences between activity representing different options (see critical discussion in Krauzlis et al. (2004), and Schall (2004a)). Note that in spite of the simplification used in this article for reporting latencies, at a conceptual level the Dynamic Field Model brings about movement initiation by a different mechanism, that is, by a transition from one to a new stable state. This may effectively drive similar shifts from a postural to a movement state at the level of motor control (Kopecz & Schöner, 1995).

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Appendix. Parameter values

To model the double target paradigm we used $\tau_{u,\mathrm{sel}} = 10$, $\tau_{u,\mathrm{ini}} = 50$ and $\tau_v = 5$ as time constants. For sigmoidal functions we used $\beta_u = 0.6$, $\beta_v = 0.04$, $a_u = 1$ and $a_v = 150$. The difference between the sigmoidal function of u and v respectively is based on the neurophysiologically realized firing rates in the cortex (McCormick, Connors, Lighthall, & Prince, 1985). For kernels we chose $w_u = 8.4$, $\sigma_u = 0.25$ mm and $w_v = 4.5$ with the coupling between selection and initiation level $w_{\mathrm{sel,ini}} = 200$. Target input strength was defined by $S_{\mathrm{tar}} = 15$ and $\sigma_{\mathrm{sel}} = 0.125$ mm, fixation input by $S_{\mathrm{fix}} = 50$ and $\sigma_{\mathrm{fix}} = 0.5$ mm. Resting levels of respective fields were $h_{u,\mathrm{sel}} = -10$, $h_{v,\mathrm{sel}} = -100$, $h_{u,\mathrm{ini}} = -20$, $h_{v,\mathrm{ini}} = -100$. Strength of gaussian white noise was depicted by $\sigma_{n,\mathrm{sel}} = 5$ and $\sigma_{n,\mathrm{ini}} = 300$. To determine latencies we used $F_{\mathrm{thresh}} = 1$. For the countermanding paradigm we used parameter values: $\tau_{u,\mathrm{sel}} = 10$, $\tau_{u,\mathrm{ini}} = 35$, $\tau_v = 5$, $\beta_u = 0.6$, $\beta_v = 0.04$, $a_u = 1$, $a_v = 150$, $w_u = 8.4$, $\sigma_u = 0.25$ mm, $w_{\mathrm{sel,ini}} = 35$, $S_{\mathrm{tar}} = 10$, mean of initial fixation signal $s_{\mathrm{fix}} = 70$, mean of countermanding signal $s_{\mathrm{count}} = 50$, $h_{u,\mathrm{sel}} = -10$, $h_{v,\mathrm{sel}} = -100$, $h_{u,\mathrm{ini}} = -5$, $h_{v,\mathrm{ini}} = -50$, $h_{\mathrm{shift}} = -5$, $\sigma_{n,\mathrm{sel}} = 5$, $F_{\mathrm{thresh}} = 10$. In the countermanding simulations movements are initiated (cancellation fails) if $M(t)$ exceeds F_{thresh}.

The fields were simulated by numerical integration. The spatially discretized network can be interpreted as a discrete neural network (for simulations we used 1 mm = 40 neurons). For temporal integration we used the stochastic Euler procedure in which

$$\tau \dot{u}(x,t) = G(u,x,t) + \sigma \eta(x,t) \qquad (.1)$$

is approximated by

$$\tau u_i^{n+1} = \delta t G_i(u_j^n, t^n) + \sqrt{\delta t}\sigma \eta_i(t^n), \qquad (.2)$$

for small time step $\delta t = 1 < \tau$. The results were obtained using MATLAB running on workstations under Unix and on PCs under NT.

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