Brain mechanisms controlling decision making and motor planning

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Abstract

Accumulator models of decision making provide a unified framework to understand decision making and motor planning. In these models, the evolution of a decision is reflected in the accumulation of sensory information into a motor plan that reaches a threshold, leading to choice behavior. While these models provide an elegant framework to understand performance and reaction times, their ability to explain complex behaviors such as decision making and motor control of sequential movements in dynamic environments is unclear. To examine and probe the limits of online modification of decision making and motor planning, an oculomotor “redirect” task was used. Here, subjects were expected to change their eye movement plan when a new saccade target appeared. Based on task performance, saccade reaction time distributions, computational models of behavior, and intracortical microstimulation of monkey frontal eye fields, we show how accumulator models can be tested and extended to study dynamic aspects of decision making and motor control.

Keywords

saccade, plan change, redirect, double step, countermanding, oculomotor

1 INTRODUCTION

An issue of central interest to cognitive neuroscience is to understand how sensory information is transformed into a motor response. Even the simple act of making a saccadic eye movement to a stimulus, which should take around 60–150 ms (Schall et al., 1995)—considering the sum of transduction delays and neural transmission times alone (Donders, 1868; Luce, 1986; Posner, 1978)—is much longer and variable, ranging from 100 to 500 ms. This implies that a significant component
of the reaction time (RT) may be required for decision making that entails determining where and when to shift the eyes. Since the time of Yarbus (1967), it has been known that eye movements are modulated by a host of factors, such as the nature of the scene being viewed, as well as the viewer’s mindset.

For example, when volunteers were asked to view a picture, saccadic eye movements were not directed to arbitrary locations but to salient, feature-rich locations, suggesting that saccades were modulated by bottom-up saliency (Cave and Wolfe, 1990; Itti and Koch, 2001; Koch and Ullman, 1985; Olshausen et al., 1993; Treisman, 1988; Wolfe, 1994; Yarbus, 1967). This notwithstanding, when the volunteers were instructed to pay attention to some aspect of the picture, say, the clothes of the people in the picture, the pattern of eye movements changed to dwell more on the clothing, suggesting that saccades were modulated in a top-down manner by the goal of the task.

It is also known that cognitive factors can modulate RTs (Posner, 1978). Saccadic RTs, for example, reduce when a cue is presented signaling the appearance of a saccade-target (Niemi and Naatanen, 1981). The appearance of the target more frequently in a particular location, as compared to another, also reduces the RT to make a saccade to the chosen location (e.g., Bichot and Schall, 2002; Dorris et al., 1999). RTs are longer when subjects are asked to be more accurate. In contrast, RTs are shorter when the subjects are asked to speed up their response, which, however, results in more errors (Chittka et al., 2009; Schouten and Bekker, 1967; Wickelgren, 1977). These observations point to a framework in which saccadic decision making and response preparation may be envisioned as a signal that represents the likelihood of the response, which accumulates to reach a threshold, at which point the saccade is triggered. In this framework, the warning signal and repetitive appearance of a target at a particular location serve to increase the likelihood of a response, thus decreasing the RT, whereas the trade-off that is observed between speed and accuracy may be explained by a shift in the threshold for responding (Reddi and Carpenter, 2000). Thus, taken together, the study of saccadic eye movements affords a simple but effective model to study how our brains make decisions leading to actions.

Using saccadic eye movements as a model system, in the first part of the review, we present evidence from recent neurophysiological studies that have provided a neural basis for accumulator models. We then present evidence from our work showing that electrical microstimulation can be used in a causal manner to test the involvement of brain areas implicated in saccade planning, as predicted by the accumulator model, and in turn validating the model. The next part of the review focuses on the ability to adapt decisions to suit the dynamic environment, a hallmark of executive control. To test the ability to rapidly modify saccadic eye movement plans, we used a paradigm called the “redirect task.” In this section, we present results from our work where we have examined the saccade planning stage, the execution stage, and finally sequences of saccades to determine whether they can be modified in the context of the redirect task. Further, we have also estimated the time taken to modify the plan/action at these various stages. Finally, we present the results from a recent microstimulation experiment performed in monkeys where the changing saccade plan was tracked in real time.
2 EVIDENCE FOR ACCUMULATOR MODELS

2.1 RTs and the LATER model

A particularly simplistic model that accounts for saccadic RTs is the LATER (Linear Approach to Threshold with Ergodic Rates) model developed by Carpenter (1981). According to this model (Fig. 1A), RT is a result of a decision signal—representing the accumulation of information that starts at a baseline and then rises at a constant rate “r” until it reaches a threshold value, at which time a saccade is triggered. “r” varies randomly from trial to trial according to a Gaussian distribution, which accounts for the variability in RTs across trials. The LATER model has only two variables, the distance and slope, whereas the baseline and threshold are considered fixed within a block of trials in the experiment. Increase in neuronal firing rate, in a LATER-like accumulation-to-threshold fashion, was initially observed in neurons in the primary motor and premotor cortical areas, prior to a wrist movement response (Riehle and Requin, 1993). LATER-like buildup in firing rate was observed prior to saccadic eye movements as well as in the superior colliculus (SC) and the frontal eye fields (FEF; Fig. 1B) in a subset of neurons (Figs. 2 and 3; Dorris et al., 1999; Hanes and Schall, 1996; Munoz and Schall, 2004).

2.2 Neural evidence for accumulation-to-threshold

Hanes and Schall (1996) provided the first clear evidence from single cells in the FEF, referred to as movement neurons, showing an increasing neuronal activity during the RT indicative of accumulation-to-threshold, which was subsequently verified by Brown et al. (2008). The saccade was triggered when the discharge rate of the movement neuron reached a threshold, which was unique for the neuron, and did not vary with RT (Fig. 3). Furthermore, most of the variability in the RT was accounted for by the variability in the rate of increase in the movement-related neuronal activity to threshold. This finding is consistent with observations from presaccadic neuronal activity in SC (Dorris et al., 1997) and lateralized readiness potentials measured from primary motor areas prior to forelimb movements (Gratton et al., 1988).

To determine whether the accumulating activity was necessary for the impending saccade, Hanes and Schall (1996) randomly interleaved a few catch trials into the simple saccade RT task, in which a signal was given to “stop” the saccade being planned. If the accumulating neuronal activity was related to saccade preparation, they reasoned that the activity of these cells would not reach the threshold on trials in which the monkey withheld the saccade successfully. Consistent with this prediction, they observed that the activity of movement neurons did not rise to threshold in trials in which the monkey successfully withheld the saccade, suggesting that the accumulating activity of movement neurons is indeed necessary for saccade generation.
(A) LATER model. On presentation of a stimulus, the decision signal (in green) rises linearly from the baseline at the rate $r$. On reaching the threshold, the saccade is initiated. On different trials, $r$ varies randomly about a mean according to a Gaussian distribution, which gives rise to a right skewed reaction time distribution. (B) Schematic representation of the visuo-saccadic circuitry in the monkey brain. In the midsagittal view, the visual signal from the eye is shown going through the optic nerve (in yellow) to the lateral geniculate nucleus (LGN) and then to the primary cortical visual area (V1). In the dorsal view, signal from V1 is shown feeding into ventral pathway visual areas (orange arrows) and dorsal pathway visual areas (green arrows). Signals from both the pathways reach LIP and FEF, which project to the SC both directly and via the basal ganglia. FEF, LIP, and SC control saccade generation (where and when to shift gaze) via a saccade generator circuitry (red and green patches) in the midbrain and pons, seen in the midsagittal view. Excitatory burst neurons (red patches) and inhibitory burst neurons (green patches) for horizontal (H) and vertical/torsional (V) components of eye movements are served by independent nuclei. Final motor commands are carried to extraocular muscles via three cranial nerves (III, IV, VI), represented by red-green lines. Patches with broken boundaries depict nuclei that are not at surface level. AS, arcuate sulcus; PS, principle sulcus; CS, central sulcus; IPS, intraparietal sulcus; LaS, lateral sulcus; STS, superior temporal; LuS, lunate sulcus.

*Figure adapted with permission from Reddi et al. (2003).*
2.3 Accumulation represents accrual of information

Although the variable rate of increase in neuronal activity accounts for the variability in RTs, whether this accumulating activity represents accrual of sensory information is not clear from simple RT tasks per se. To test this hypothesis, Roitman and Shadlen (2002) trained monkeys to view a random-dot stimulus (Fig. 4; Britten et al., 1992) in which a fraction of the dots moved coherently—either toward or away from the response field (RF) of the neuron that was being recorded from. The fraction
of coherently moving dots could be varied to vary the motion signal strength over noise. Two peripheral targets were presented: one placed in the neuron’s RF and the other in the diametrically opposite location. The monkey had to discriminate the net direction of motion and indicate the decision by making a saccade to the target presented in that direction. By recording from single neurons in the lateral intraparietal area (LIP), a progressive increase in firing rate was observed following the appearance of the stimulus, and the rate of increase in firing rate was modulated by the strength of the motion signal. Furthermore, when the firing rate reached a threshold level of activity, the saccade was initiated. In other words, when the motion strength was stronger, these neurons showed a faster increase in neuronal activity, so the threshold was attained sooner resulting in shorter RTs, whereas, when the motion strength was weaker, the increase in the neuronal activity was slower, so the threshold was attained later resulting in longer RTs. These observations are consistent with the notion that LIP neurons accumulate sensory evidence up to a threshold following which the saccade results. Similar results were obtained from other sensorimotor brain regions, like the SC (Horwitz and Newsome, 2001) and the FEF (Kim and Shadlen, 1999) that

**FIGURE 3**

Evidence of accumulation-to-threshold. Following the appearance of the stimulus (at 0 ms), the activity of FEF movement-related neuron rises (e.g., shown in blue) to a fixed threshold firing rate (dashed horizontal line) at which point the saccade is initiated. Trial-to-trial variability in the time of initiation of the saccade originates from the variable time taken for the activity to reach threshold. To illustrate this, trials were subdivided into fast (green part of the reaction time distribution histogram), medium (blue), and slow (yellow) reaction time groups and the average buildup activity of the movement neuron for each of these groups is shown in the corresponding color.

*Figure modified with permission from Schall and Thompson (1999).*
Accumulation represents accrual of sensory evidence. (A) Motion direction discrimination task. In this task, following a fixation period, two targets appear (black-filled circles), one in the RF (gray patch) and the other opposite to it. Subsequently, the random-dot stimulus appears at the center. The monkey has to indicate the direction of the moving dots by making a saccade to one of the filled circles. RT, reaction time. (B) The response of LIP neurons when the monkey is discriminating the direction of motion. The average firing rate of 54 neurons is shown for six different motion strength conditions (in different colors). Left: the responses are aligned to the onset of random-dot stimulus. Right: the responses are aligned to the saccade
are also involved in oculomotor control. Further, in order to causally test whether this accumulating activity, seen in the LIP and the FEF, etc., determines choice or merely reflects it, Gold and Shadlen (2000, 2003) administered microampere currents strong enough to evoke a saccade reliably in the FEF, when the monkeys were discerning motion direction. The arrangement of stimuli was changed such that the saccade evoked by microstimulation was perpendicular to the choice saccades (Fig. 5A).

It is known from earlier studies that the direction of the electrically evoked saccade is influenced by the eye movement being planned (Sparks and Mays, 1983). In other words, if the monkey were to initiate an upward saccade to signal upward motion (Saccade “2” in Fig. 5B), then the horizontal saccade evoked by the electrical stimulation (Saccade “1” in Fig. 5B) would deviate away from the horizontal direction, the RF at the site of stimulation, to land slightly upward (Saccade “3” in Fig. 5B). If the accumulating activity is indicative of the evolving saccade plan, which is reflected by the deviation of the saccade away from the RF, then the extent of deviation should be modulated by factors that affect the state of the saccade plan like the stimulus-viewing duration and the stimulus motion strength. Consistent with this hypothesis, it was observed that the saccade deviation in fact increased with increase in stimulus-viewing duration as well as motion strength, suggesting that the accumulating activity represents accrual of information, on the basis of which the saccade choice is made.

Ramakrishnan et al. (2012) verified the validity of the accumulator model in the context of a simple RT task in which monkeys were trained to make a saccadic eye movement to the stimulus that appeared on a computer monitor. The saccade target was in the direction orthogonal to the stimulation-evoked saccade, like in the previously described experiment (Fig. 6A); however, stimulation pulses were administered at various time points on different trials (at 30, 60, 90, 120, 150, or 180 ms following target onset) to sample the RT period (200 ms on average). If the accumulating activity is causally linked to response preparation, then the stimulation-evoked saccade is expected to interact with the formative saccade at various stages of response preparation. Thus, the direction of the resultant averaged saccade is expected to change systematically. In other words, if the stimulation pulse was administered early during saccade preparation, the resultant saccade is expected to land close to the RF at the site of stimulation, whereas if the stimulation was administered at a later stage of saccade preparation, the resultant saccade is expected to...
The evolving saccadic decision is seen in the oculomotor system. (A) Microstimulation-interrupted direction discrimination task. In this task, the monkey has to decide the net direction of motion like in the direction discrimination task (refer Fig. 4). However, while the monkey is discriminating, a microstimulation pulse is administered to the FEF to evoke a saccade in the orthogonal direction (rightward in this case). Following the evoked saccade, the monkey initiates a voluntary saccade to the saccade target. (B) Eye movement trajectories. When the monkey is stimulated while fixating at the center (0, 0), an evoked saccade results (shown by trajectory 1). However, if the monkey is viewing upward motion and therefore planning the upward saccade (shown by trajectory 2), then stimulation results in a saccade that deviates in the upward direction (shown by trajectory 3). (C) Extent of deviation. The average amount of deviation, representative of accumulating activity, depended on motion strength and viewing time. This shows that oculomotor system is causally involved in decision making and, more importantly, the accumulating activity may represent the evolving decision.

Figure taken with permission from Gold and Shadlen (2000).

to land further away from the RF, closer to the target. Consistent with the predictions from the accumulator model, it was observed that stimulation early in the RT resulted in saccades that landed close to the RF. The saccade deviation increased with the time of stimulation, monotonically, till the maximum was attained close to the time of the voluntary saccade onset (Fig. 6E). Additionally, if the rate of
FIGURE 6
Evoked saccade deviation in no-step trials. (A) The plot shows the evoked saccade endpoint locations when the monkey is fixating (on the black-filled square) but planning a saccade to the target (green-filled square) and the stimulation is administered early (<100 ms after target onset; black dots) and late (>100 ms after target onset; purple dots).
increase in saccade deviation was indicative of the rate of response preparation, then sessions with faster increase in saccade deviation are expected to be associated with shorter average saccade RT and vice versa. Since the stimulation pulse was administered in random 50% of the trials in every session, the saccade deviation profile and the average saccade RT could be obtained from the stimulated and nonstimulated trials, respectively. Further, since the mean RT varied across sessions, the session-wise mean RT and the rate of increase in saccade deviation could be compared across sessions. This comparison showed that the RT was inversely correlated with the rate of increase in saccade deviation (Fig. 6F), establishing a causal relationship between saccadic response preparation and the accumulating activity in the oculomotor network, as assessed by microstimulation in the FEF.

3 EXTENDING ACCUMULATOR MODELS TO ACCOUNT FOR SACCADE PLAN MODIFICATION

3.1 Countermanding paradigm: Canceling a saccadic response

In these experiments, while subjects are preparing a saccade to a peripheral stimulus, a second, centrally appearing stimulus instructs them to cancel the saccade plan on random catch trials (Fig. 7A). In general, if the second stimulus appears after a shorter interval called the stop signal delay, subjects cancel the saccade plan more
(A) a No-stop trial

b Stop trial

Stop signal delay

Reaction time

(B) Probability of error vs. Stop signal delay (ms)

(C) a Noncanceled

b Canceled

Stop signal delay (ms)

Reaction time

Activation

GO

STOP

Noncanceled

Canceled
A race model framework has often been used to understand the basis for performance in the countermanding task. The model involves a LATER-like GO process that accumulates to threshold following the appearance of the peripheral target. The GO process initiates a saccade upon reaching a threshold level of activation. However, on trials in which the saccade is to be canceled, a STOP process accumulates to threshold. Trials in which the STOP process reaches the threshold before the GO process are trials in which the saccade is successfully withheld (Fig. 7C), whereas trials in which the GO process reaches threshold are error trials. Such a race model can successfully account for performance of subjects, as assessed by the probability of error trials, which are the catch trials in which the saccade was initiated, as a function of the stop signal delay. Note, however, that the race model assumes that the STOP process can stop the GO process anytime until the GO process reaches threshold. In other words, the model assumes that the impending saccade can be canceled anytime during the planning stage. This may not be possible if part of the response preparation period includes a ballistic stage, which is a response processing stage that is not amenable to modification (De Jong et al., 1990; Logan, 1981; McGarry and Franks, 1997; McGarry et al., 2000). To test this, Kornylo et al. (2003) modified the race model to include a terminal ballistic stage. In other words, in this race model, after a certain time point, the GO process was deemed unstoppable. The optimal duration of such a ballistic stage that was needed to fit the performance profiles was assessed by simulating the modified race model. The duration of the ballistic stage estimated in this way was found to be very short (9–25 ms in monkeys and 28–60 ms in humans). This time period is, interestingly, equivalent to the

FIGURE 7
(A) Countermanding task. Trials begin with the presentation of a central fixation box, which disappears after a variable fixation time. Simultaneously, a target appears at an eccentric location. On a fraction of trials, after a delay, referred to as stop signal delay (SSD), the fixation box (shown in red) reappears (in (b)). In these trials, the saccade to the target is required to be withheld (stop signal trials). During trials when the stop signal is not presented (no-stop trial; in (a)), a saccade is required to be initiated to the target (represented by an arrow to the target). In stop trials, subjects sometimes withheld the saccadic response successfully (canceled trials) and sometimes did not (noncanceled trials). (B) Inhibition functions. Plots showing the probability of making a saccade to the first target a function of SSD. (C) Race model of countermanding behavior. Following the appearance of the target (green box), a GO process (green line) rises to threshold (gray horizontal bar), triggering off a saccade to the target. In stop trials, a stop signal is presented (red box) which gives rise to a STOP process (red line) that races to threshold. If the STOP process reaches threshold before the GO process, then the saccade is canceled successfully (upper panel), whereas if the GO process reaches threshold first, then the saccade is not canceled (lower panel).
3.2 **The double-step task: Modifying a saccade plan**

The double-step task is another paradigm that is used to study how saccadic response preparation can be modified. In this task, a peripheral saccade target is stepped to a new position, while the saccade to the initial location is being prepared but not yet executed. The correct response involves modifying the current saccade plan to make a saccade to the new target. Such behavior allows the assessment of the subject’s control over the saccade under preparation by measuring the probability of trials in which the response is modified successfully, much like in the countermanding task. Initially, it was observed that saccades to each of the targets were executed in tandem even if the target had stepped to the new location before the first saccade began, which spurred the debate on whether saccade programming is ballistic (Westheimer, 1954). However, a number of studies have challenged this view by showing that the saccade to the first stimulus can be modified (e.g., Komoda et al., 1973; Lisberger et al., 1975; Wheeless et al., 1966). The redirect task, which is a modified version of the double-step task, has also been successfully used to probe the ability to modify saccade plans (Ramakrishnan et al., 2010; Ray et al., 2004). In this task, the initial target stays on, instead of shifting to a new location, even after the new peripheral stimulus appears (Fig. 8A and B). Subjects have to modify the saccade plan to the initial target to make one to the new target, like in the double-step task. However, in some trials, subjects are unable to modify the saccade plan to the initial target leading to an erroneous response. The probability of error trials, which is an index of the ability to modify the initial response, increases with the delay in the appearance of the new stimulus, which is called the target step delay (Fig. 8C). This suggests that subjects find it harder to modify the initial saccade plan when the new target appears later, a trend that is consistent with the countermanding task. In general, these observations suggest that a saccade plan can be modified or canceled during the response preparation stage; however, it gets progressively harder to do so later in time, presumably because of advancing commitment to the initial response.

3.3 **Race model approach**

*GO–GO model*: Theoretically, the simplest model that can account for performance in a redirect task involves the use of two independent LATER-like accumulators—GO1 and GO2, which represent saccade preparation to the initial and final target, respectively (Becker and Jurgens, 1979). However, GO–GO models fail to explain the compensation function in the redirect task (Ramakrishnan et al., 2012). This result is because such a model does not allow for the cancelation of the saccade
No-step trial

(A) No-step trial

(B) Step trial

Target step delay

(A1) No-step response

(B1) Successful response

(B2) Erroneous response

(C) Probability of error

Target step delay (ms)
preparation to the first target, and therefore, the proportion of error trials is much more than expected.

**GO–STOP–GO model:** One way to modify the race model is by including a STOP process, developed to account for performance in the countermanding task, to inhibit the GO1 process, to allow the GO2 process to initiate the saccade to the final target (Ramakrishnan et al., 2010). In other words, the competition between the GO1 process and the STOP process will decide whether the first response is canceled or not. Following successful cancelation of the GO1 process, the GO2 process sets off the saccade to the new target (see Fig. 9A). Error trials are those in which the saccade to the first target is initiated. In these trials, according to the race model, the GO1 process reached the threshold before the STOP process (Fig. 9A, green part of the distribution). Success trials are the ones in which the STOP process beat the GO1 process to the threshold (Fig. 9A; red part of the distribution). The inherent variability in the rate of accumulation of the GO1 process, from trial to trial, gives rise to a fraction of trials that are successfully modified at every target step delay. Implementation of such a race model allows one to arrive at the rate of accumulation of the STOP process, in order to predict the fraction of error trials as a function of the target step delay.

The race model, however, assumes that the STOP process does not have any variability. This assumption is unwarranted since the STOP process too is a biological process and may be subject to variability. In other words, the rate of accumulation of the STOP process may vary across trials as well. Therefore, in the modified race model, the rates of the STOP process were also modeled as a Gaussian distribution, like that of the GO process (Fig. 9B). Knowledge of both distributions enable the estimation of the fraction of error trials for a given target step delay. In practice, however, given the limited number of trials per subject, it may not be possible to sample the entire distribution of the STOP process, especially at the extremities, whereas it may be easier to sample the central part of the distribution (two standard deviations).

**FIGURE 8**
Illustration of the temporal sequence of stimuli and behavior in the redirect task. The task comprises (A) no-step trials, when a single target (green square) appeared on the screen, and (B) step trials, when a second target (red square) appeared after a target step delay (TSD). In no-step trials, subjects were required to foveate the target by making a saccade, shown in yellow, to the target (A1). In step trials, subjects were required to modify the saccade plan and initiate a saccade to the final target. Sometimes, they successfully compensated for the target step (yellow) (B1). On other occasions, they failed to compensate, which resulted in an erroneous saccade to the initial target (yellow) followed by a corrective saccade to the final target (magenta) (B2). (C) Compensation functions. Plots showing the probability of making a saccade to the first target as a function of TSD. Data for nine representative subjects, superimposed by the best-fit Weibull function, show that the probability of making an erroneous first saccade increases with increasing TSD.

*Figure taken with permission from Ramakrishnan et al. (2010).*
3 Extending accumulator models to account for saccade plan

(A) No-step reaction time distribution

(B) Latency of the slowest erroneous response

(C) a Multi saccade gaze shift b Midflight modification c Hypometric error

Activation

Time from initial target onset (ms)

100 ms
deviations on either side of the mean, Fig. 9B). The estimate of the probability of error trials is, therefore, underestimated. The underestimation is, however, only due to the right tail of the STOP distribution, because the trials with slower STOP process rates are the ones that result in errors, which is about 2.5%. However, it may be possible that the distribution of the STOP process is non-Gaussian. Nevertheless, based on Chebyshev’s theorem, the upper limit of the underestimation of the percentage of error trials is not expected to exceed 12.5% (Ramakrishnan et al., 2010). In summary, even if the rates of GO and STOP processes are both variable across trials, the proportion of error trials can be found with the upper limit of the underestimation being limited to 12.5%.

The race model, as mentioned earlier, assumes that the STOP process can inhibit the GO process anytime during planning stage. However, if parts of the saccade planning stage are not amenable to inhibitory control, that is, if the preparatory stage involves a ballistic stage, the proportion of error trials should increase and, as a result, the underestimation should be more than 12.5%. This criterion, which is robust to the inherent variability of the GO and STOP processes and to the unavoidable sampling variation.
errors, was used to detect the presence of a ballistic stage during saccade planning and execution. Using this method, Ramakrishnan et al. (2010) tested for the presence of a ballistic stage in the saccade planning phase and found the underestimation of the error trial probability to be limited to 10 %, less than 12.5 %, which meant that saccade planning phase may not involve a ballistic stage. Or, in race model terms, the STOP process can inhibit the GO process till it reached threshold. This result is congruent with earlier work based on the countermanding task that reported the ballistic stage to be limited, perhaps, to the final efferent pathway (Kornylo et al., 2003).

Having tested the planning stage, Ramakrishnan et al. (2010) probed the saccade execution stage for the presence of a ballistic stage, that is, whether the saccade can be modified midflight or not. Large amplitude saccades provide a longer saccade execution duration, which is beneficial in testing whether the saccade can be interrupted during this late stage. Therefore, subjects were made to perform a version of the redirect task in which the target eccentricity from the center was increased to 30°, which increased the saccade duration to about 70 ms. The long saccade duration sometimes allowed subjects to interrupt the saccade midflight and make a compensatory saccade to the new target (see panel (b) in Fig. 9C). Using the race model-based framework, the probability of error trials, trials in which they could not change the saccade plan midflight, could be estimated and the underestimation in this case was found to be ~13 %, suggesting that saccade execution stage, like the saccade planning stage, did not, perhaps, involve a ballistic stage. In other words, saccades could be modified anytime during movement execution as well.

In about 13% of the trials, on average across subjects, subjects made a sequence of two saccades, a multisaccade gaze shift (panel (a) in Fig. 9C), to foveate the eccentrically located target. These multisaccade gaze shifts provided Ramakrishnan et al. (2010) the opportunity to test for the ability of the oculomotor system to modify a saccade plan in redirect trials during saccade sequences. In other words, the saccade sequence allowed the authors to test whether the saccade plan could be modified following the primary saccade (p) in a multisaccade sequence. The error trials in this case comprised trials in which the primary saccade was followed by a secondary saccade (s) to foveate the initial target (panel (c) in Fig. 9C) despite the appearance of the final target before the primary saccade was initiated. Even though the parameters of the rate of accumulation of the secondary saccade are not known, because, presumably, the parameters of the STOP process remain the same, the probability of error trials can, nevertheless, be estimated. When this analysis was carried out, it was found that a surprisingly large fraction of trials turned out to be error trials, almost 78 % on average across subjects, which was about six times more than expected. In other words, secondary saccades of a large fraction of trials involving multisaccades could not be compensated despite there being enough time to modify the plan. This strongly indicated the presence of a ballistic stage in the programming of multisaccade sequences, suggesting that the response preparation stage of the secondary saccade involved stages of processing that were not amenable to modification.

By providing a computational basis for the behavior in the redirect task, the race model allowed the estimation of the time taken to modify the saccade plan, called the target step reaction time (TSRT; Ramakrishnan et al., 2010), analogous to the stop
signal reaction time (SSRT) of the countermanding task (Logan and Cowan, 1994) which was 107 ms on average, across subjects. However, the TSRT to modify the saccade during execution was significantly higher at 152 ms across subjects, on average. Nevertheless, a significant subject-wise correlation \(r = 0.77; p = 0.0008\) between the two TSRT estimates suggested that inhibitory control might engage similar mechanisms even though it took more time to modify the saccade during execution. In contrast, subjects failed to modify the secondary saccade even when they had about 215 ms, on average. In other words, even though the saccade control processes could effectively modify the saccade plan in about 107 ms, more than double that time was insufficient to modify the secondary saccade, rendering it opaque to control processes. Thus, the intersaccadic interval before the secondary saccade onset in multisaccade gaze shifts may be a potential point of no return in saccadic response preparation, which may be the first clear demonstration of it in sensorimotor response preparation. Although it is puzzling that the secondary saccade is refractive to the new stimulus, stopping the secondary saccade may be a lot harder because they may be programmed as a package along with the primary saccade of multisaccade gaze shifts. Alternately, the primary hypometric saccade of multisaccade gaze shifts may also be a consequence of the motor command not fully specifying the goal. In such a scenario, it is possible that the error correction system might, by priority, engage the oculomotor circuitry to correct the hypometric saccade, which might prevent new visual input from modifying the saccadic response. Knowing whether this observation is applicable to saccade sequences, in general, or is limited to multisaccade gaze shifts may shed some light on the basis for the point of no return.

### 3.4 Time course of the change of plan as assessed with microstimulation

It is known from earlier work that electrical microstimulation administered to the FEF to evoke a saccade orthogonal in direction to the one being planned results in an averaged saccade whose direction indicates the state of the saccade being planned (Gold and Shadlen, 2000, 2003; see also Kustov and Robinson, 1996; Sparks and Mays, 1983; Fig. 6E). Ramakrishnan et al. (2012) extended this technique to assess the state of the saccade plan while it was being modified. For this, the authors trained two monkeys to perform the redirect task. During a step trial, while the monkey was changing the saccade plan, they administered stimulation currents to the FEF at six different time points, spaced by 30 ms, following the final target onset. The experimental design was such that stimulation alone would produce an electrically evoked saccade in the direction orthogonal to both the initial and final targets. If microstimulation could reveal the state of the saccade plan, they reasoned that stimulation administered soon after the final target onset would evoke a saccade that should interact with the saccade plan to the initial target, to produce a saccade that lands in between the initial target and RF (Fig. 10A, middle row of panels), since saccade preparation to the initial target is yet to be modified. However, when the monkey is stimulated long after final target onset, the stimulation-evoked saccade
Extending accumulator models to account for saccade plan

(A)  

(B)  

(C)  

(D)
should interact with the saccade plan to the final target to produce a saccade that lands in between the final target and the RF (Fig. 10A, bottom row of panels), as the saccade preparation to the initial target should be modified by then. As predicted, the evoked saccade direction, as indicated by saccade deviation from the RF, gradually shifted from the initial target direction to the final target direction (Fig. 10B and C). The time when the deviation profile crossed the RF—the crossover time—an estimate of the time when the plan changed, was about 100 ms, on average. Furthermore, session-wise comparisons of crossover time with TSRT, which was estimated from the nonstimulated trials of the session, was reasonably well correlated ($r=0.45; p<0.003$; 43 sites, two monkeys; Fig. 10D). Taken together, these data suggest that microstimulation is a powerful tool to visualize the time course of covert cognitive processes changes plans in real time.

4 CONCLUSION

The body of work reviewed in this chapter suggests that saccadic decision making and motor planning can be envisioned as an accumulation of activity within a selective population of neurons distributed within the oculomotor system. These plans may be modified anytime during the planning and execution stage. Successful plan modification takes about 107 ms on average and the time course of this process can be tracked by electrical microstimulation as well as single unit recordings. The

FIGURE 10

Evoked saccade deviation in step trials. (A) In the first row of panels, when a stimulation pulse (blue oscillations) is delivered, a saccade (blue arrow) is evoked. The middle and bottom rows represent a short TSD (16 ms) trial that is microstimulated by either a short latency (10 ms) or a long latency (140 ms) pulse. The subsequent panel shows the evoked saccade, the saccade under preparation, and the averaged saccade as blue, black, and red arrows, respectively. The black arrows are drawn short of the target to represent saccades under preparation. The rightmost panels show the observed saccade. The dots forming the saccade represent the eye position samples. At early stimulation times, the resultant averaged saccade deviates toward the initial target, but at later stimulation times, it deviates toward the final target. (B) The evoked saccade deviation profile in a typical session for TSD = 80 ms is shown. (C) The averaged saccade deviation profile from three TSDs (16, 80, 144 ms) is shown after aligning each of them to the onset of the final target. In (B) and (C), the median of the deviation (red circles) is fit by a weighted-smoothing spline (solid black line). The dashed blue lines represent 95% confidence limits. Crossover time (CT) represents the time when the deviation profile crosses the RF (denoted by the red arrow), as estimated from the fit. In (D), the TSRT obtained from the race model-based analysis is plotted against the crossover time obtained from the observed evoked saccade deviation profile as a scatter plot ($N=43$ sites). Each gray-filled circle represents data from a session. Dashed-black line represents the line of unity slope.

*Figure adapted with permission from Ramakrishnan et al. (2012).*
congruence between patterns of activity obtained from electrophysiological measurements in single neurons, physiological perturbations of the oculomotor network by microstimulation, and computational modeling provide firm grounding to the belief that race models are a logical framework to understand how motor plans and decisions are prepared or modified by the brain. On the other hand, the failure of the race model to explain secondary saccades of multisaccade gaze shifts suggest a genuine failure of inhibitory control and provides the necessary evidence of a ballistic stage that intervenes during more complex movements involving multiple saccades. Although, why and how such ballistic stages are implemented by the brain remain a matter of speculation, we hope that the success of accumulator and race models in the domain of oculomotor control can be extended to the study of motor control involving other effectors such as hand movements, as well as in the study of natural movements that require temporal coordination between multiple independent effectors.

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References


CHAPTER 17 Brain mechanisms controlling decision making