



HAL
open science

Saccadic Eye Movements: Basic Neural Processes

Laurent Goffart

► **To cite this version:**

Laurent Goffart. Saccadic Eye Movements: Basic Neural Processes. Reference Module in Neuroscience and Biobehavioral Psychology, , 2017, Sensorimotor systems, 10.1016/B978-0-12-809324-5.02576-1 . hal-01427818

HAL Id: hal-01427818

<https://hal.science/hal-01427818>

Submitted on 5 Mar 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

In: Reference Module in Neuroscience and Biobehavioral Psychology
<https://doi.org/10.1016/B978-0-12-809324-5.02576-1>

Saccadic Eye Movements: Basic Neural Processes

Laurent Goffart

CNRS, Marseille, France

Contact information:

Laurent Goffart, PhD

Institut de Neurosciences de la Timone,

UMR 7289 Centre National de la Recherche Scientifique Aix-Marseille Université,

Marseille,

France.

e-mail: laurent.goffart@univ-amu.fr

KEYWORDS:

Adaptation, Cerebellum, Feedback, Fovea, Gaze, Goal-directed, Interception, Motor control, Neural basis of behavior, Neurophysiology, Saccade, Space, Subcortical, Time, Vision.

ABSTRACT:

The sudden appearance of an object in the visual field triggers an orienting shift of gaze toward its location. This movement consists of an extremely rapid rotation of the two eyes, the saccade. In this article, instead of describing the paths leading from the target-evoked retinal activity to the changes in muscle tension that rotate the eyes, we take the reverse path. Starting from the muscle contractions, we proceed upstream and describe the basic neural and functional mechanisms involved in generating commands that permit the eyes and, therefore, the foveae to visually capture the location of an event.

The sudden appearance of an object in the visual field triggers an orienting shift of gaze toward its location. This movement is an extremely rapid, step-like rotation of the two eyes – the saccade – which can sometimes be accompanied by a head movement. By positioning the object's image on the retinal region of highest acuity – the fovea – the saccade increases the number of neurons activated by the presence of the object; a recruitment which likely contributes to improve its identification and localization. But saccades are not exclusively aimed at objects that suddenly appear; scanning saccades are also generated when one is exploring the environment. In this case, saccade goals are extracted from the visual scene based on their saliency or their relevance for subsequent actions (e.g., grasping an object, heading toward a location, avoiding an obstacle, or reading the next word in a sentence).

Studying the neural control of saccades is a choice research theme for investigating how the central nervous system controls goal-directed actions. Not only because the saccade is an essential movement that vertebrates use to spatially organize their sensory experience, but also because it is a relatively simple model of the transformations which are applied to sensory signals in order to generate motor commands for reaching a goal, whether it is stationary or moving. This research is facilitated by the fact that the eye movements can nowadays be recorded with very good spatial and temporal resolutions. [Figure 1](#) illustrates the trajectory and the time course of a saccade aimed at a small static target appearing in the peripheral visual field. The trajectory is shown by plotting the vertical versus the horizontal angular deviation (also commonly called position) of the eye ([Figure 1\(a\)](#)). The time course shows that, after a latency (reaction time) of approximately 150 ms, the change in eye orientation is extremely rapid (velocity profiles in [Figure 1\(c\)](#)) and ends with the maintenance of the eye in a new orientation ([Figure 1\(b\)](#)). It also shows that a secondary correction saccade sometimes follows the primary saccade.

<Figure 1 near here>

This seemingly very simple movement is actually the outcome of complex muscular and neural activities with a functional organization which is not yet completely uncovered. In this article, instead of describing a labyrinthine path from the target-evoked retinal signals to the changes in muscle tension that rotate the eyes, we take the reverse path. Starting from the muscle contractions, we proceed upstream and succinctly expose the neural mechanisms involved in generating the commands for saccadic eye movements.

Extraocular Muscles and Rotation of the Eyes

Saccades are elicited by the cooperative action of six muscles attached to the eyeball. These muscles are called extraocular muscles, to distinguish them from those located inside the eyeball and which influence the diameter of the pupil or the lens curvature. Two pairs of muscles are involved during horizontal rotations of the eyes (i.e., rotations around a vertical axis through the eyeball): the lateral rectus (LR) and medial rectus (MR) muscles. Orienting the right eye toward the right is accomplished by the contraction of the LR and the relaxation of the MR ([Figure 2\(a\)](#)) while in the left eye the MR is contracted and the LR relaxed. Conversely, shifting the gaze toward the left is produced by the MR contraction and the LR relaxation in the right eye ([Figure 2\(b\)](#)) concomitantly with LR contraction and MR relaxation in the left eye. The muscles which contract are called the agonist muscles; those which relax are called the antagonist muscles.

<Figure 2 near here>

The muscle contraction generates a torque which is delivered to the eyeball through tendinous attachments. [Figure 2\(b\)](#) shows the tension developed by the LR and MR muscles during a horizontal saccade. The agonist muscle tension waveform first shows a rapid rise in tension, which is followed by a quasi-exponential decay until a steady-state tension is achieved and maintains the eye deviated in the orbit. For the

antagonist muscle, the tension waveform shows a symmetrical pattern, with a sudden drop in tension followed by an exponential increase until the steady-state tension is also reached. These muscle tension recordings reveal that, after every saccade, the orientation of the eye is settled well before the agonist and antagonist muscle forces reach their final equilibrium. They also illustrate the three components of the innervation signal supplied to the extraocular muscles: a pulse, a slide, and a step component. The pulse and the slide quickly rotate the eyes and stop them abruptly against the viscosity of the muscles and orbital tissues, respectively. The step generates the muscle tension required to hold the eyes deviated in the orbit, overcoming the elastic forces that tend to drag the eyes toward their more central resting position.

Vertical movements of the eyes (rotations about a horizontal axis through the eyeball) are slightly more complex than horizontal ones because they are the result of a synergy between four other extraocular muscles. The joint contraction of the superior rectus (SR) and inferior oblique (IO) muscles of both eyes, combined with the relaxation of the inferior rectus (IR) and superior oblique (SO) muscles, rotates the eyes upward. The bilateral contraction of the IR and SO, combined with the relaxation of SR and IO, rotates them downward. Oblique saccades are generated by tensions simultaneously developed by the muscles rotating the eye horizontally and those rotating the eye vertically.

The Saccade Generators

The force generated by extraocular muscles is driven by the activity of motor neurons (MNs) located in cranial nerve nuclei III (oculomotor), IV (trochlear), and VI (abducens). The schema in [Figure 3](#) shows the simplest possible view of the network of motor and premotor neurons which are involved in generating a rightward horizontal saccade (leftward saccades involve a similar neuronal network but located in contralateral territories of the reticular formation). The recruitment of MNs driving the agonist muscles is ensured by a population of excitatory burst neurons (EBNs) located in the ipsilateral pontine reticular formation (path p1). These EBNs drive the activity of

abducens MNs innervating the LR of the ipsilateral eye. For contracting the MR in the contralateral eye, the EBN command is relayed to MNs in the oculomotor nucleus by internuclear neurons (INs), also located in the abducens nucleus. In parallel, EBNs activate a population of inhibitory burst neurons (IBNs) located in the ipsilateral medullary reticular formation and which inhibit the activity of EBNs and MNs in the contralateral side (path p2). The burst of ipsilateral IBNs thereby participates in the inhibition of MNs innervating the antagonist muscles (the LR of the left eye and MR of the right eye). Thus, the antagonist muscles can relax while the agonist muscles contract. Some neurons in the IBN area also burst during contralateral saccades. Hence, if these neurons also inhibit contralateral MNs, the motor command sent to the MNs innervating the agonist muscles must be viewed as the combination of two inputs: an excitatory input from ipsilateral EBNs (path p1) and an inhibitory input from the contralateral IBNs (path p3). This bilateral organization of the premotor drive during horizontal saccades explains the bilateral organization of its control by the medio-posterior cerebellum (see below).

<Figure 3 near here>

During upward and downward saccades, different neuronal populations are involved. They are located in the mesencephalic reticular formation, more specifically in the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF). One major difference between the neural control of horizontal and vertical saccades is that the former are mostly driven by a unilateral *excitatory* input (from the ipsilateral pontine reticular region), whereas the latter are driven by a bilateral *excitatory* input from the left and right riMLF.

The burst of excitatory and inhibitory burst neurons in the pontomedullary (horizontal component) and mesencephalic (vertical component) reticular formations seems to be gated by the activity of another class of inhibitory neurons located in the nucleus raphe interpositus: the omnipause neurons (OPNs). These neurons exhibit a sustained activity which is interrupted every time a saccade is generated, regardless of

the amplitude and direction of saccades. Released from this sustained inhibition, EBNs and IBNs drive the motoneurons. The resulting saccadic eye movement has a direction which is determined by the activation ratios between the horizontal and vertical saccade generators. In healthy subjects, the examination of oblique saccades shows that both the horizontal and vertical rotations are initiated simultaneously, not sequentially (see [Figure 1](#)). Moreover, some observations show that the horizontal and vertical components are coupled – when the velocity of one component is reduced, the other component is also slowed down, thereby reducing the delay between the completion of horizontal and vertical components. Despite this component-stretching mechanism, the horizontal and vertical components may end at slightly different times. The analysis of saccades generated by neurology patients is very instructive in this matter. For example, in patients suffering from Niemann–Pick type C disease, the horizontal component precedes the vertical component, resulting in the completion of the vertical component well after the end of the horizontal component. Because of their inhibitory influence on burst neurons, the OPNs are mainly considered as involved in visual fixation (i.e., maintaining still the direction of gaze). Yet, they could also be involved in coupling the horizontal and vertical saccade generators.

In [Figure 3](#), it is the same network which is involved for a horizontal saccade aimed at an object located 4° or 16° to the right. Additional mechanisms must be added to this network to generate saccades of different amplitudes and durations.

The Control of Saccade Execution

The Duration and Velocity of Saccades

In almost all studies, the duration of a saccade is defined as the time elapsed from when the eye velocity exceeds a certain threshold (e.g., 15° s^{-1}) to when it returns below it. The duration of saccades increases linearly with saccade amplitude (ranging from 1° to 50°) at a rate of approximately $2\text{--}3 \text{ ms deg}^{-1}$ in humans ($1\text{--}2 \text{ ms deg}^{-1}$ in monkeys). For saccades less than approximately 30° , the velocity profile is bell-shaped (see [Figure 1\(c\)](#)), with a rapid rise to a peak value (acceleration phase) followed by a

symmetrical decline in velocity (deceleration phase). For larger saccades, the velocity profile becomes skewed, with the duration of deceleration phase being longer than the acceleration phase. The peak velocity increases linearly with saccade amplitude until a value of approximately 15° , beyond which the increase in velocity saturates to a maximum value as large as 700° s^{-1} (900° s^{-1} in monkeys). These relationships between the amplitude and the duration or peak velocity of saccades are called the main sequences. Because their graphical shapes are relatively similar across healthy subjects, the main sequences have become a tool for studying the oculomotor deficits in brain-lesioned patients. Some authors have also suggested that common physiological systems are responsible for the generation of reactive saccades and voluntary saccades, as well as for the fast phases of optokinetic and rotatory nystagmi.

However, these duration–amplitude and peak velocity–amplitude relationships show considerable variability between subjects and even within the same subject. They are sensitive to alertness, motivation, and several other parameters such as saccade direction, initial deviation of the eyes in the orbit, and the testing conditions (i.e., whether the saccade is memory-guided or immediate). Saccade velocity also depends on the sensory nature of the target. Saccades toward somatosensory and auditory targets are much slower than saccades of comparable amplitude and direction aimed at visual targets. Saccades to a target moving away from the central visual field can also be slower than saccades of matched amplitude toward a stationary target; some are characterized by a lower peak velocity and a prolonged deceleration phase.

The Spatiotemporal Transformation

On the motor output side, the duration and velocity of saccades are defined by the temporal properties of MN discharge. For saccades initiated from the straight ahead position in the orbit, different saccade amplitudes do not involve different groups of MNs; all the MNs are recruited. Saccades of increasing amplitude are caused by increases in the duration and rate of MN discharge, the instantaneous firing rate dictating instantaneous saccade velocity (rate code). On the other side, at the sensory level, the code is topographic; targets at different locations in the visual field activate

different zones in the retina. A topographical organization is found in several regions in the brain, including the intermediate and deep layers of the superior colliculi, the neurons of which are located two to three synapses from the motor nuclei. From a macroscopic point of view, the two deep superior colliculi (dSC) form a motor map, which appears to be in agreement with the retinal map, with the anterior (rostral) regions of both colliculi representing the foveal and perifoveal visual fields, posterior (caudal) regions representing the peripheral visual field, medial regions representing the upper visual field, and the lateral regions representing the lower visual field. In the dSC, neurons emit a burst of action potentials shortly before a contralateral saccade within their movement field (i.e., the range of saccade amplitudes and directions that is preceded by a burst). In the rostral region, the burst occurs during small contralateral saccades, whereas neurons in more caudal regions burst during saccades of larger amplitude. Neurons located in the medial border burst prior to upward oblique saccades, and those located on the lateral border burst before downward oblique saccades. Interestingly, the electrical microstimulation of a particular site in the dSC evokes a saccade with amplitude and direction corresponding to those for which the stimulated neurons would maximally discharge. Thus, the amplitude and direction of impending saccades seem to be determined by the locus of active neurons in the dSC. However, this topographical code is not sufficient because a mismatch can be observed between the amplitude and direction of saccades and those expected from the topography of active neurons. Indeed, for the same target location, the amplitude and direction are very different between saccades to a permanent target and saccades to a memorized target location. The latter systematically end above the target location while the locus of collicular activity has not changed. Therefore, the topographical code seems to be completed by adjustments downstream, in the reticular formation.

Another fundamental problem is to understand how the locus of activity in the dSC is converted into the MN temporal discharge. This transformation was initially considered to be performed by a putative spatiotemporal translator (STT) located in the cerebellum. A kind of look-up table mechanism associated a specific temporal pattern of motor activity with each locus of collicular activity. But, this explanation is not

satisfactory because saccades generated toward the same target and from the same starting position can be slower in velocity (longer in duration), yet still have the same amplitude. The hypothesis was then proposed by David A. Robinson that the duration of saccades is determined by a negative feedback loop. The saccade generators would be driven by a motor error signal, a command which would result from identical comparison between two signals: a reference signal specifying the goal or desired state (e.g., desired eye orientation or desired change in eye orientation), and a feedback signal estimating the current state (current eye orientation or actual change in eye orientation). As long as there is a mismatch between these two internal signals, the motor error would drive the saccade generators until the estimated current state matches the desired one. In this framework, the motor error is a dynamic command that changes with the direction of gaze. There is no need for an explicit STT; the duration of saccades is merely determined by the time taken to zero-out the motor error command. To date, the negative feedback loop remains the simplest hypothesis proposed to account for the spatiotemporal transformation. Later, neuromimetic modeling taught us that the neuronal solutions to the computations involved in saccade generation (e.g., spatiotemporal transformation and component stretching) may not be explicitly performed by separate groups of neurons that neurophysiologists ought to identify. Instead, these computations would be implemented implicitly, emerging from the activities of populations of neurons that are massively interconnected and distributed over several neural territories.

The Feedback Control of Saccades

One of the strengths of the negative feedback control is that it endows the oculomotor system with a high level of flexibility for the execution of saccades. If for some reason – pathological (e.g., spinocerebellar degeneration), experimental (functional perturbation by brief electrical microstimulation or by systemic/local injection of pharmacological agents), or natural (drop in vigilance, blink, presence of a distractor) – the saccade is executed with a velocity which is much slower than normal or with an

altered initial trajectory, its accuracy is preserved as long as the feedback signal conveys information about the changes in horizontal and vertical velocity.

Several sources can contribute to this feedback signal. The intrasaccadic visual signals (optic flow and/or target-elicited retinal trace) could participate in estimating the direction of the ongoing saccade. In patients affected by spinocerebellar degeneration, saccades are so dramatically slowed that they can be redirected in flight toward a new target presented during their course. However, in healthy subjects, most saccades are too short in duration for visual signals to influence their trajectory. Inflow signals originating from mechanoreceptors located in the orbital tissues or from extraocular muscle proprioceptors could also be involved in the feedback. But their elimination by sectioning of the ophthalmic branch of the trigeminal nerve does not affect the accuracy and velocity of saccades, nor does it affect the ability to compensate for unexpected changes in eye position prior to saccades toward flashed targets. The estimation of the ongoing oculomotor state could also be based on copies of commands sent to the MNs (efference copy). These outflow signals could originate from collaterals of burst neurons which drive the MNs. It has indeed been shown that partial pharmacological inactivation of the region involved in the generation of horizontal saccades barely affects the accuracy of the horizontal component of oblique saccades, despite a dramatic reduction in velocity. These observations, which are consistent with the feedback control of saccade amplitude, also indicate that the number of active neurons influences the velocity of saccades. Interestingly, the cerebellar influence on saccade generation seems to involve recruiting neurons and controlling the size of the active population in the pontomedullary reticular formation.

The hypothesis of saccade accuracy being controlled by a negative feedback loop also predicts that a defect in estimating the current state should lead to dysmetric saccades. For example, an underestimation of the displacement performed by the eyes from saccade onset is expected to lead to hypermetric saccades (saccades larger in amplitude than required) because the motor error is canceled later than usually (i.e., until the time when the defective estimated displacement matches the desired displacement). Conversely, an overestimation of the actual displacement is expected to

lead to hypometric saccades (i.e., saccades that are smaller in amplitude than required). From this perspective, the generation of accurate saccades requires an adjustment of feedback signals such that they accurately represent the actual oculomotor output. This adjustment is also required because the feedback neural signals are not “commensurable” with the neural signals encoding the desired state. In fact, the neural substrate of this feedback process still remains to be discovered.

Several lines of evidence suggest that the dSC participates in the processes specifying the goal of saccades, upstream from the neural site where the motor error command is elaborated. Concerning the feedback signals, the cerebellum is likely involved in their adjustment. In particular, its medio-posterior region (composed of the vermal lobules VIc–VII and caudal fastigial nuclei), receives target-related signals from pontine nuclei, proprioceptive signals from extraocular muscles, and eye position/displacement signals from the nucleus prepositus hypoglossi/medial vestibular nucleus complex. In return, the output nuclei of the medial-posterior cerebellum, the caudal fastigial nuclei, send distributed projections to the territories housing the premotor neurons of the saccade generators. When the normal function of fastigial oculomotor region (FOR) is impaired, the horizontal component of saccades becomes severely dysmetric: it is hypometric for contralesional saccades, hypermetric for ipsilesional saccades and an ipsipulsion affects the trajectory of vertical saccades. The different deficits that affect ipsilesional and contralesional saccades indicate a bilateral control which is consistent with the discharge and the connectivity of FOR neurons.

The medio-posterior cerebellum can be excluded from the node performing the STT because its dysfunction does not break the spatial-to-temporal mapping: the saccades become dysmetric but different amplitudes still have different durations. In the reticular formation, the *nucleus reticularis tegmenti pontis* (NRTP) is a major target of both the dSC and the FOR. Because pharmacological inactivation of NRTP suppresses the generation of ipsilateral saccades, more studies are required to study its role in the spatiotemporal transformation.

The Adaptive Control of Saccades

Saccade accuracy is maintained by adaptive mechanisms which modify the visuomotor transformation to reduce the error of the following saccade. For example, a mismatch between saccade amplitude and target eccentricity characterizes saccades generated by newborns. When orienting toward a peripheral target, infants as young as 1–2 months of age frequently execute a series of consecutive saccades of approximately equal size, whether accompanied by the head or not. However, although it is hypometric, the amplitude of primary saccades increases with the eccentricity of the target.

These adaptive mechanisms have also been studied in human subjects and in monkeys with weakened muscles in one eye. When the subject monocularly views a visual target, saccades are accurate if the normal eye is used but are too small in amplitude when the weakened eye is the viewing eye. If the normal eye is patched and the subject is forced to use the weakened eye to look at visual targets, the amplitude of saccades gradually increases until they are nearly accurate. The fact that the changes are observed in both the weakened eye and the normal patched eye (which now makes hypermetric saccades) indicates that the adaptation involves a command which is issued for conjugate movements of both eyes. This flexibility of the oculomotor system to adjust saccade accuracy has also been studied in experiments in which the target is displaced to a new location during the saccade. Initially, saccades land near the first target location, but gradually they land closer to the second one (see paragraph “*Natura non facit saltus*”).

Comparable adaptive processes must also take place during the generation of saccades toward the changing location of a moving target. Interestingly, chemical lesions of cortical area MT or of its pontine target, the dorsolateral pontine nucleus (DLPN), two structures known to be involved in the processing of visual motion, impair the accuracy of saccades to moving targets without altering that of saccades to stationary targets. The DLPN being one of the major sources of motion-related visual signals to the cerebellum, the adaptation therefore involves this network that connects

saccade-related regions of the brainstem and the cerebellum through pre-cerebellar nuclei.

The Specification of the Desired State

The Varieties of Targets

Saccades are not exclusively aimed at visual targets. They can also be generated in the dark, toward sounds, and to stimuli applied to the skin surface. Localizing such stimuli is a much more complicated task. In the case of a somatosensory stimulus, estimating its location requires the knowledge of the stimulated body region (e.g., the index fingertip of the right hand) and of the posture of the limb on which the stimulus is applied. For auditory stimuli, they are localized on the basis of the interaural differences in intensity and timing of acoustic signals. The dSC seems to be the interface through which these different sensory signals have access to the saccade generators. Several saccade-related cells in the dSC are active prior to saccades to visual, somatosensory, or auditory stimuli, suggesting that the converging sensory signals are translated into oculocentric coordinates by the level of the dSC. But the differences in velocity observed between these different groups of saccades suggest that a signal specifying the desired displacement is not the unique output signal from the dSC to the saccade generators. The differences could be due to different levels of firing rate in the population of active neurons or to different population sizes. Here again, the influence of the size of the active population is consistent with the observations that regular visual saccades also have a reduced velocity and increased duration (accuracy relatively spared) during pharmacological inactivation either of small portions within the dSC or the source of one of its major cortical afferents, the frontal eye fields.

“Natura non facit saltus”

The specification of the goal of saccades should not be viewed as a discrete process but a continuous one. Several lines of evidence indeed show relationships

between the amplitude (or direction) and the reaction or response times of saccades, unraveling a dynamical process. For example, a dependence of saccade endpoints on the latency has been documented in studies testing saccades aimed at two stimuli that are simultaneously presented at close locations in the visual field. Under these conditions, the saccade endpoints are scattered along an axis joining the two target locations, characterizing an averaging process. This averaging depends on the relative properties of visual targets (size and energy) – the saccade landing position is closer to the larger or the brighter target. Moreover, it mostly occurs for short latency saccades and not when the latency exceeds 300 ms. The time window of the averaging process can be delimited by presenting the two targets in a successive manner; short-latency saccades end at the location of the first target, whereas long-latency saccades end at the second target location. The amplitude transition function defines the averaging window that accounts for endpoints that proportionately change with saccade latency. Another instance of dynamic specification has recently been reported in a study which tested saccades toward a transient moving target. For example, in response to a target that uniformly accelerates, the landing position of saccades is more eccentric with longer response time (i.e., the time interval from the target motion onset to the saccade end).

Saccades toward the Changing Location of a Moving Target

The study of saccades toward a moving target nicely illustrates the fact that the specification of the saccade goal is continuous process. In primates, the appearance of an object moving in the visual field elicits a saccadic eye movement that brings the target image onto the foveae. Such a spatiotemporal coincidence is quite remarkable when one considers that the retinal signals are transmitted to the motor neurons through multiple parallel channels connecting separate populations of neurons (characterized by different integration times) with variable conduction speeds and delays. Indeed, the flows of neural activity which are initiated by a moving target do not remain local. They propagate both “vertically” (i.e., across the successive relays interposed between the retina and the extra-ocular motoneurons) and “horizontally”

(within each relay) in the brain. Because of the delays, the foveal capture cannot be driven by the current retinal signals as they correspond to past positions of the target.

Yet, when the trajectory of an interceptive saccade is experimentally perturbed, a correction is made, sometimes after an intersaccadic pause (i.e., with presumably no premotor activities). These corrections do not bring gaze toward a future location of the target; they land next to the location where unperturbed saccades would have landed at about the same time. These observations indicate that the saccade generators are driven by a command estimating the current spatiotemporal coordinates of the target, i.e., where it is here-and-now. This estimate is likely built upon antecedent visual signals combined with adaptive signals memorized during previous experiences. When the spatiotemporal coincidence is not reached, the saccade falls short of the target and a correction (catch-up) saccade is made to bring the target image back onto the foveae. Most observations show indeed that the interceptive saccades do not orient the foveae toward any “future” location where the gaze would be maintained, waiting for the target to enter within the foveal field and initiate its pursuit.

Yet, the idea has diffused that the target motion-related signals are used to predict the “future target position” so as to assure a “spatial lead” of the gaze at the saccade end, instead of attempting a precise capture of the target. Such an idea is problematic for two fundamental reasons. Firstly, this view does not specify the horizon of predictability, i.e., how far ahead the visuo-saccadic system is able to “predict the future”. Secondly, it neglects the fact that the saccades do not orient a point without thickness (as schematized in [Figure 1](#) and [Figure 2](#)). They orient a surface of photoreceptors which is rather extended. This extent is likely linked to the fact that in their “*Umwelt*”, the animals do not track or chase after tiny objects; their visual targets have extended sizes. In other words, considering the gaze direction as a “line” is misleading because it leads us to consider as future a direction which is in fact present.

The Control of Saccade Initiation

Saccades are triggered on the basis of information gathered during the time interval that precedes saccade onset. This latency is far longer than would be expected by adding the synaptic delays and nerve conduction times between the excitation of sensors and the contraction of extraocular muscles. It reflects the time taken to extract relevant signals from sensors, to make decisions about whether, and how, to respond to the stimulus, and to start transmitting the initially-selected goal to the saccade-related motor system. In response to subsequent incoming signals, a “decision” signal is supposed to rise from an initial level until it reaches a threshold level, at which point the saccade is initiated. Thus, a rise that varies randomly between trials would account for the wide distribution of saccade latencies.

Several factors influence the latency of saccades. On the retinal side, latency depends, for instance, on the eccentricity of the target, on its energy, and on its contrast relative to the background. Saccade latency also depends on the presence and location of distractors and cues, as well as on more cognitive factors such as the probability of the target location, the presence of cues, the amount of reward associated with the target, the target valence and salience, and visuospatial attention.

Under certain experimental conditions, the distribution of saccade latencies is bimodal. The first mode, which can peak as low as 90 ms, designates the express saccades, whereas the second mode corresponds to regular saccades. The observation of express saccades is facilitated when a temporal gap separates the fixation target offset and second target appearance. Express saccades are suppressed when the deep superior colliculus is lesioned.

Conclusion

Saccadic eye movements are an important component of the animal’s behavior for exploring and spatially organizing its sensory environment. They provide a useful tool for understanding how the central nervous system relates the sensorium with the motor repertoire, how it enables the entire animal to locate objects in its environment.

The aim of the neurophysiological approach, whether it is focused on processes occurring at microscopic, mesoscopic or macroscopic scales, is to reveal the embedded neuronal organizations which subtend the behavioral performance in a world of different complexity. Further empirical research is required to determine how the basic neural processes underlying orienting movements influence the generation of other types of goal-directed movements and how they participate in the construction of spatial memory. However, this bottom-up approach should be made carefully. It is indeed possible that the brain, in its internal workings, does not support principles that are similar to those used in Newtonian mechanics.

Further Readings

- Barton, E.J., Nelson, J.S., Gandhi, N.J. and Sparks, D.L. (2003). Effects of partial lidocaine inactivation of the paramedian pontine reticular formation on saccades of macaques. *Journal of Neurophysiology* **90**, 372-386.
- Becker, W. (1989). Saccades In Carpenter R.H.S. (ed) *Eye Movements: Vision and Visual Dysfunction*. pp 95-137 London: Macmillan.
- Becker, W. and Jürgens, R. (1990). Human oblique saccades: Quantitative analysis of the relation between horizontal and vertical components. *Vision Research* **30**, 893-920.
- Cromer, J.A. and Waitzman D.M. (2007). Comparison of saccade-associated neuronal activity in the primate central mesencephalic and paramedian pontine reticular formations. *Journal of Neurophysiology* **98**, 835-850.
- Davis-López de Carrizosa, M.A., Morado-Díaz, C.J., Miller, J.M., de la Cruz, R.R. and Pastor A.M. (2011). Dual encoding of muscle tension and eye position by abducens motoneurons. *Journal of Neuroscience* **31**, 2271-2279.
- Fleuriot, J. and Goffart, L. (2012). Saccadic interception of a moving visual target after a spatiotemporal perturbation. *Journal of Neuroscience* **32**, 452-461.

- Fleuriet, J., Hugues, S. and Goffart, L. (2011). Saccadic foveation of a moving visual target in the rhesus monkey. *Journal of Neurophysiology* **105**, 883–895.
- Fuchs, A.F. Kaneko, C.R. and Scudder, C.A. (1985). Brainstem control of saccadic eye movements. *Annual Review of Neuroscience* **8**, 307-337.
- Gandhi, N.J. and Bonnadonna, D.K. (2005). Temporal interactions of air-puff-evoked blinks and saccadic eye movements: insights into motor preparation. *Journal of Neurophysiology* **93**, 1718-1729.
- Gandhi, N.J. and Katnani, H.A. (2011). Motor functions of the superior colliculus. *Annual Review of Neuroscience* **34**, 205–231.
- Goffart, L., Chen, L.L. and Sparks, D.L. (2004). Deficits in saccades and fixation during muscimol inactivation of the caudal fastigial nucleus in the rhesus monkey. *Journal of Neurophysiology* **92**, 3351-3367.
- Goffart, L., Hafed, Z.M. and Krauzlis, R.J. (2012). Visual fixation as equilibrium: evidence from superior colliculus inactivation. *Journal of Neuroscience* **32**, 10627-10636.
- Hafed, Z.M. and Krauzlis, R.J. (2012). Similarity of superior colliculus involvement in microsaccade and saccade generation. *Journal of Neurophysiology* **107**, 1904-1916.
- Harris, C.M., Jacobs, M., Shawkat, F. and Taylor, D. (1993). The development of saccadic accuracy in the first seven months. *Clinical Vision Sciences* **8**, 85-96.
- Hopp, J.J. and Fuchs, A.F. (2004). The characteristics and neuronal substrate of saccadic eye movement plasticity. *Progress in Neurobiology* **72**, 27-53.
- Jürgens, R., Becker, W. and Kornhüber H. (1981). Natural and drug-induced variations of velocity and duration of human saccadic eye movements: Evidence for a control of the neural pulse generator by local feedback. *Biological Cybernetics* **39**, 87-96.
- Kaneko, C.R. and Fuchs, A.F. (2006). Effect of pharmacological inactivation of nucleus reticularis tegmenti pontis on saccadic eye movements in the monkey. *Journal of Neurophysiology* **95**, 3698-3711.
- Keller, E.L. (1974). Participation of medial pontine reticular formation in eye movement generation in monkey. *Journal of Neurophysiology* **37**, 316-332.

- Kojima, Y., Iwamoto, Y., Robinson, F.R., Noto, C.T. and Yoshida, K. (2008). Premotor inhibitory neurons carry signals related to saccade adaptation in the monkey. *Journal of Neurophysiology* **99**, 220-230.
- Krauzlis, R.J., Lovejoy, L.P. and Zénon, A. (2013). Superior colliculus and visual spatial attention. *Annual Review of Neuroscience* **36**, 165-182.
- Miller, J.M. and Robins, D. (1992). Extraocular muscle forces in alert monkey. *Vision Research* **32**, 1099-1113.
- Moschovakis, A.K., Scudder, C.A. and Highstein, S.M. (1996). The microscopic anatomy and physiology of the mammalian saccadic system *Progress in Neurobiology* **50**, 133-254.
- Noto, C.T. and Gnadt, J.W. (2009). Saccade trajectories evoked by sequential and colliding stimulation of the monkey superior colliculus. *Brain Research* **1295**, 99-118.
- Nichols, M.J. and Sparks, D.L. (1995). Nonstationary properties of the saccadic system: new constraints on models of saccadic control. *Journal of Neurophysiology* **73**, 431- 435.
- Optican, L.M. and Robinson, D.A. (1980). Cerebellar-dependent adaptive control of primate saccadic system. *Journal of Neurophysiology* **44**, 1058-1076.
- Pellionisz, A. and Llinas R. (1982). Space-time representation in the brain: the cerebellum as a predictive space-time metric tensor. *Neuroscience* **7**, 2949-2970.
- Quinet, J. and Goffart, L. (2015). Cerebellar control of saccade dynamics: contribution of the fastigial oculomotor region. *Journal of Neurophysiology* **113**, 3323–3336.
- Quinet, J. and Goffart, L. (2015). Does the brain extrapolate the position of a transient moving target? *Journal of Neuroscience* **35**, 11780-11790.
- Robinson, D.A. (1973). Models of the saccadic eye movement control system. *Biological Cybernetics* **14**, 71-83.
- Robinson, D.A. (1975). Oculomotor control signals. In Lennerstrand, G. and Bach-Y-Rita P (eds) *Basic Mechanisms of Ocular Motility and Their Clinical Implications* pp 337-378, Oxford: Pergamon Press.

- Robinson, D.A. (1992). Implications of neural networks for how we think about brain function. *Behavioral and Brain Sciences* **15**, 644–655.
- Schlag, J., Pouget, A., Sadeghpour, S. and Schlag-Rey, M. (1998). Interactions between natural and electrically evoked saccades. III. Is the nonstationarity the result of an integrator not instantaneously reset? *Journal of Neurophysiology* **79**, 903-910.
- Scudder, C.A., Kaneko, C.R.S. and Fuchs A.F. (2002). The brainstem burst generator for saccadic eye movements: A modern synthesis. *Experimental Brain Research* **142**, 439-462.
- Sparks, D.L. and Mays, L.E. (1990). Signal transformations required for the generation of saccadic eye movements. *Annual Review of Neuroscience* **13**, 309-336.
- Sparks, D.L. (2002). The brainstem control of saccadic eye movements *Nature Reviews Neuroscience* **3**, 952-964.
- Taouali, W., Goffart, L., Alexandre, F. and Rougier, N.P. (2015). A parsimonious computational model of visual target position encoding in the superior colliculus. *Biological Cybernetics* **109**, 549-559.
- van Gisbergen, J.A.M., Robinson, D.A. and Gielen, S. (1981) A quantitative analysis of generation of saccadic eye movements by burst neurons. *Journal of Neurophysiology* **45**, 417-442.

Change History

February 2016. L Goffart updated the text and further readings to this entire article.

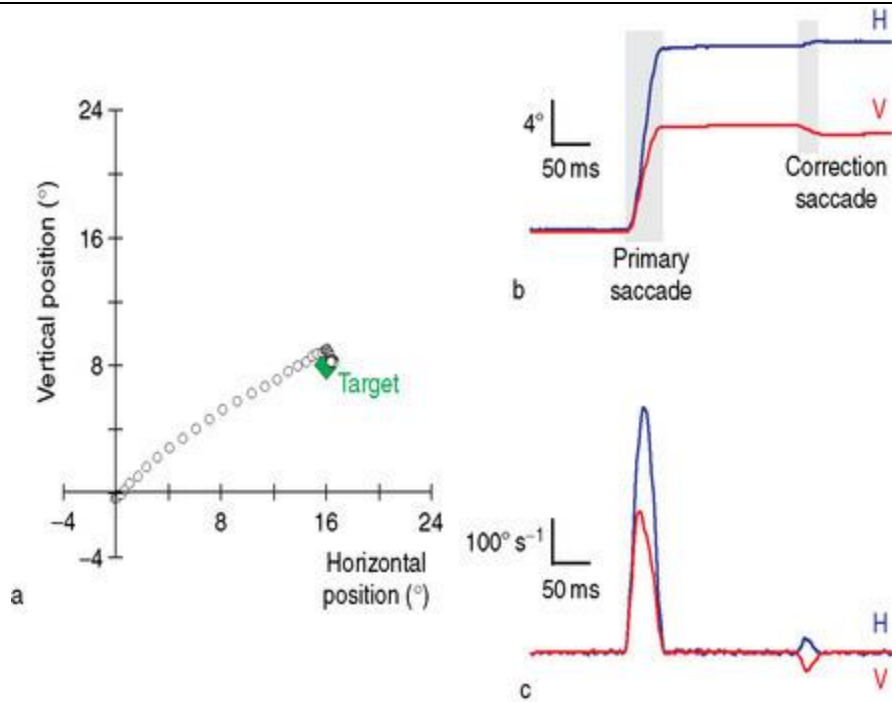


Figure 1. Oblique saccade: (a) spatial trajectory; (b) time course of eye position; (c) time course of eye velocity. The oblique saccade is initiated from a fixation stimulus located straight ahead (intersection between the axes in (a)) and aimed at peripheral static target located 16° to the right and 8° upward (green diamond). Positive values of horizontal (H) and vertical (V) positions correspond to rightward and upward eye deviations in the orbit.

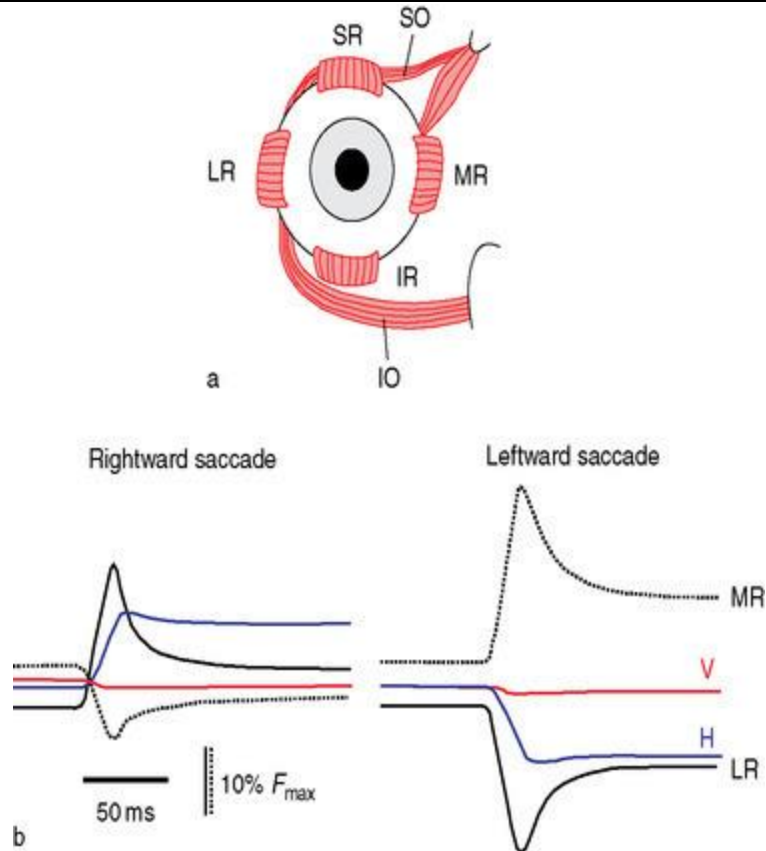


Figure 2. (a) Extraocular muscles of the right eye; (b) changes in LR and MR muscles tensions during a 16° horizontal saccade. Forces (black traces) are given as percentage of the maximum force (F_{max}). Upward deflection corresponds to muscle contraction; downward deflection corresponds to muscle relaxation. H, horizontal position; IO, inferior oblique; IR, inferior rectus; LR, lateral rectus; MR, medial rectus; SO, superior oblique; SR, superior rectus; V, vertical position. (b) Reproduced from [Miller JM and Robins D \(1992\)](#) Extraocular muscle forces in alert monkey. *Vision Research* 32: 1099–1113, with permission from Elsevier and the authors.

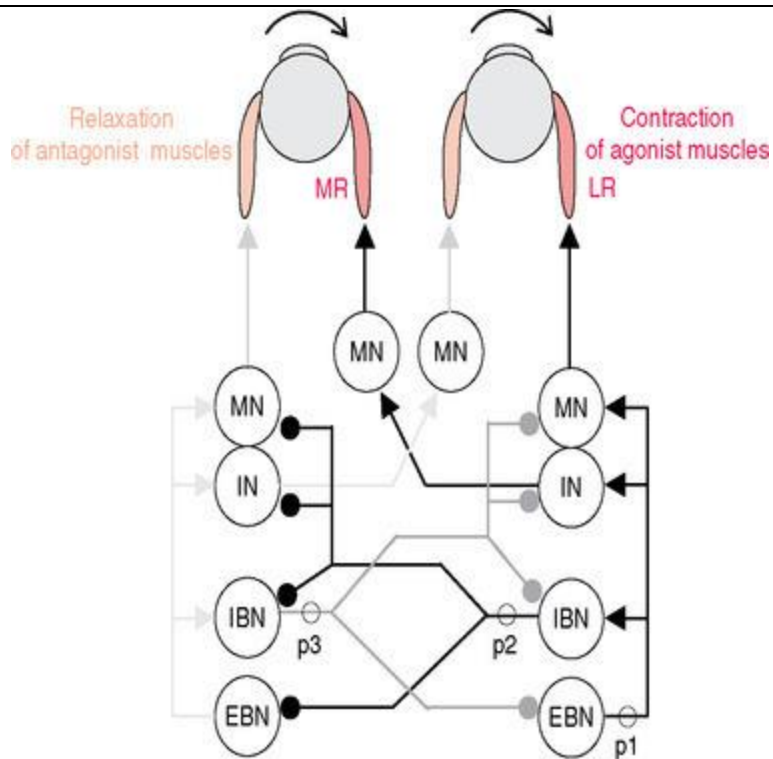


Figure 3. Neuronal organization involved during a rightward saccade. Arrows indicate excitatory connections; connecting lines ended by a circle indicate inhibitory connections. The connecting lines have different colors for clarity. EBN, excitatory burst neuron; IBN, inhibitory burst neuron; IN, interneuron; LR, lateral rectus; MN, motor neuron; MR, medial rectus; p1, path 1; p2, path 2; p3, path 3.