Oculomotor System: Models

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Introduction

The oculomotor system comprises the contents of the orbits (the globe; six extraocular eye muscles; and many fatty, fibrous, and elastic tissues) and parts of the brain. The globe undergoes a very limited amount of translation during movements, about 2 mm along the anterior–posterior axis and 0.5 mm in the frontal plane. Thus, the globe can be considered to be a spherical joint with its center fixed in the orbit. There are three main properties of the orbit that the brain must deal with. The first is the orbital mechanics created by the three-dimensional (3-D) geometry of the eye muscles and the surrounding orbital tissues. These determine the pulling action of each muscle, that is, how a contraction of a muscle is converted to a torque applied to the globe. The second is the dynamics of the orbital tissues. If the time course of the innervation sent to the muscles is not appropriate for the orbital dynamics, the eye will drift when the movement is over. The third is that 3-D rotations are not commutative. If the axis of the applied torque is not correct for the orientation of the eye, plant dynamics will cause unintended drifts or blips around other axes.

Oculomotor Plant

The oculomotor plant is very complicated, nonlinear, and still only poorly understood. Thus, many models of the plant are in use today, with linear models being used when the details can be safely lumped together to give average dynamics.

Lumped Plant Models

Early models of the plant were simplified by considering only rotations of the eye around a single (e.g., vertical) axis. Control theory models of the plant date from the suggestion by Westheimer in 1954 that rapid eye movements (called saccades) were the step response of the plant. This idea suggests a very simple saccadic system because the visual input for a saccade (a jump of the target of interest) evokes a step change in the site of innervation on the retina and, hence, in other brain areas containing maps of retinal activity. If these inputs were added together

with appropriate weights (stronger for more distant targets), they would create a steplike change in the firing rate of the neurons, which could be sent directly to the plant. Westheimer proposed that the plant was a critically damped linear system, which would foveate the target as quickly as possible without overshooting it.

In 1964, Robinson measured the mechanical properties of the plant and found that its dynamics were dominated by the viscosity of the muscles and, thus, was not critically damped but instead overdamped. The dominant time constant of the plant was $\sim$285 ms. Thus, if the innervation to the plant were changed in a steplike fashion, the eyes would drift exponentially toward the target and would only arrive after about three time constants. Because we make 3–4 saccades s^{-1}, the innervation must be able to compensate for the viscosity in the plant or our eyes would never stop drifting.

Robinson derived a fourth-order linear model of the plant's mechanics and showed that the innervation needed to drive the plant to make a saccade consisted of at least three parts: a pulse, a slide, and a step. The step determines the final orientation of the eye, the pulse generates extra force to overcome the viscous drag of the orbital tissues, and the slide compensates for some of the viscosity and the relaxation in slow orbital tissues after the rapid part of the movement is over.

The ratio of the output (eye position) to the input (innervation) in Robinson's model of the plant was represented as a transfer function (a linear input–output relationship expressed as the ratio of two complex polynomials). Robinson's plant consisted of one real zero (the value at which the numerator polynomial becomes zero), two real poles (values at which the denominator polynomial of the transfer function becomes zero), and a complex-pole pair. The complex-pole pair contributes an oscillation to the output of a movement that is very small because of the large viscosity. Thus, the plant can be usefully approximated with just two real poles and one zero. **Figure 1(a)** shows the corresponding mechanical model with three viscoelastic (or Voigt) elements, with the transfer function:

$$E/R = c \times ([sT_z + 1]/[sT_1 + 1][sT_2 + 1])$$

where s is the complex Laplace transform variable, T_1 and T_2 are the viscoelastic time constants, or plant poles, and T_z is the plant's zero (**Figure 1(b); Table 1**). The scale factor, c, is set by the net compliance of
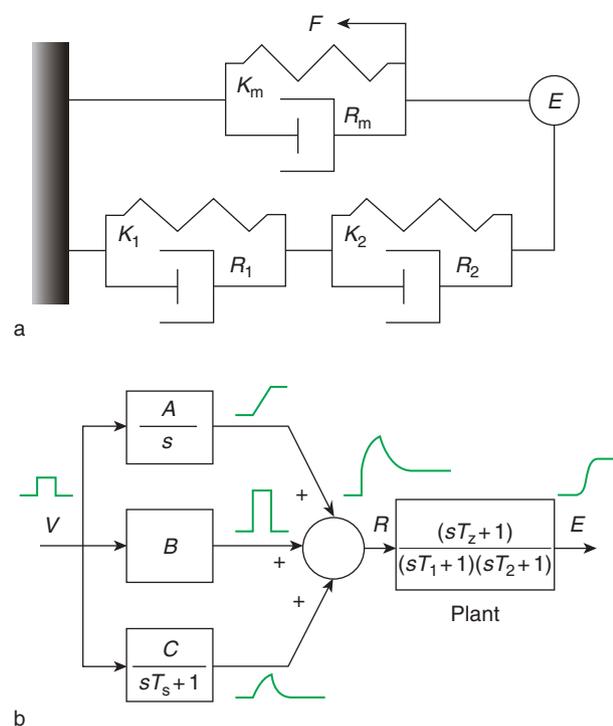
Figure 1 Second-order linear approximation to the oculomotor plant and its neural compensation: (a) mechanical model of the active and passive tissues in the plant, with the viscosity and elasticity represented by Voigt elements (parallel spring, K, and dashpot, R); (b) simplified transfer function of the model in (a) (Plant) and the circuit needed to compensate its dynamics. In (a), the force of the muscle's contractile elements is represented by F; the eyeball is represented by E, and the time constant of each Voigt element is R/K. In (b), the innervation (R) for an eye position (E) requires a pulse, slide, and step. For one-dimensional rotations, the slide and step can be obtained by integrating the pulse. Green traces show velocity command (V), step, pulse and slide of innervation, combined pulse-slide-step (R), and eye position (E). The plant has two poles (T_1 and T_2) and one zero (T_z). A, B, and C are weights and T_s is a time constant needed to build the slide of innervation (see **Table 1**). The transfer functions are shown in Laplace transform notation, so $1/s$ represents a perfect integrator, and $1/(sT+1)$ represents a leaky integrator (or low-pass filter).

Table 1 Lumped two-pole, one-zero plant model and its compensator

Model transfer function		
T_1	Dominant pole's time constant	0.136 s
T_2	Second pole's time constant	0.726 s
T_z	Plant's zero time constant	0.615 s
τ	Excitation–contraction coupling delay	0.008 s
Compensator (final common path)		
A	Step gain	1
B	Pulse gain	0.1605
C	Slide gain	0.0865
T_s	Slide time constant	0.615 s

the springs in the model $(K_1 + K_2)/(K_1 K_2)$ and the innervation–contraction coupling ratio, but it is conventionally set to 1 so that the gain of the plant is 1 for very low frequency movements. In terms of the viscosity and elasticity, the time constants are $T_i = R_i/K_i$, and the zero is given by $T_z = (R_1 + R_2)/(K_1 + K_2)$.

Numerically, the stiffnesses of the two passive elements are approximately 0.79 and 1.22 g/°, with viscosities of 0.0158 and 1.22 gs/°. (Note: Ophthalmologists measure muscle force during surgery with a spring scale calibrated in grams; 1g force is 9.8×10^{-3} N.) Thus, the time constants for the poles in the passive tissues are 0.020 and 1.000 s, and the zero is at 0.615 s. The viscoelasticity and active-force generation of the muscle is represented by another Voigt element (F, K_m, R_m). Its stiffness is about 1.58 g/° and the viscosity is about 0.316 gs/°, which gives a lumped time constant for both muscles of approximately 0.200 s. Combining the passive and active tissues, we can approximate the total plant model with two poles ($T_1 = 0.136$ s, $T_2 = 0.726$ s) and one zero ($T_z = 0.615$ s). The zero approximately cancels the second pole, making the dominant time constant about 0.150 s. Thus, in models in which the details of rapid eye movements are not important, the plant model can be reduced to just a single pole: $1/(0.150s + 1)$. When time delays are taken into account, a delay of about 8 ms is added to the plant by putting the factor $e^{-0.008s}$ in the numerator of the transfer function.

Note that these values are all approximations of a more complex, nonlinear system. As such, their values can change in different species or under different experimental conditions. For example, the dominant time constant in monkeys ranges from 0.090 to 0.200 s and in humans from approximately 0.100 to 0.200 s.

One Dimension

In one dimension, the compensatory innervation (R) needed to drive this plant can be easily determined if the pulse command (V) is known (**Figure 1(b)**). Integrating the pulse with a gain (A) gives the step, and low-pass filtering the pulse (with gain C and time constant T_s) gives the slide. With perfect plant compensation, the ratio of eye movement to innervation would simply be $1/s$ (a pure integrator). Hence, for normal saccades, the gain of the step should be 1, which fixes $A = 1$. The gain of the pulse must then be $B = T_1 T_2/T_s$, which is about 0.1605. To compensate for the zero in the plant, $T_s = T_z$, or 0.615. The gain of the slide must be $C = T_1 + T_2 - T_s - (T_1 T_2/T_s)$, which is 0.0865. **Figure 2** shows an implementation of the plant and its compensator and the output from a simulation run (cf. **Table 1**).
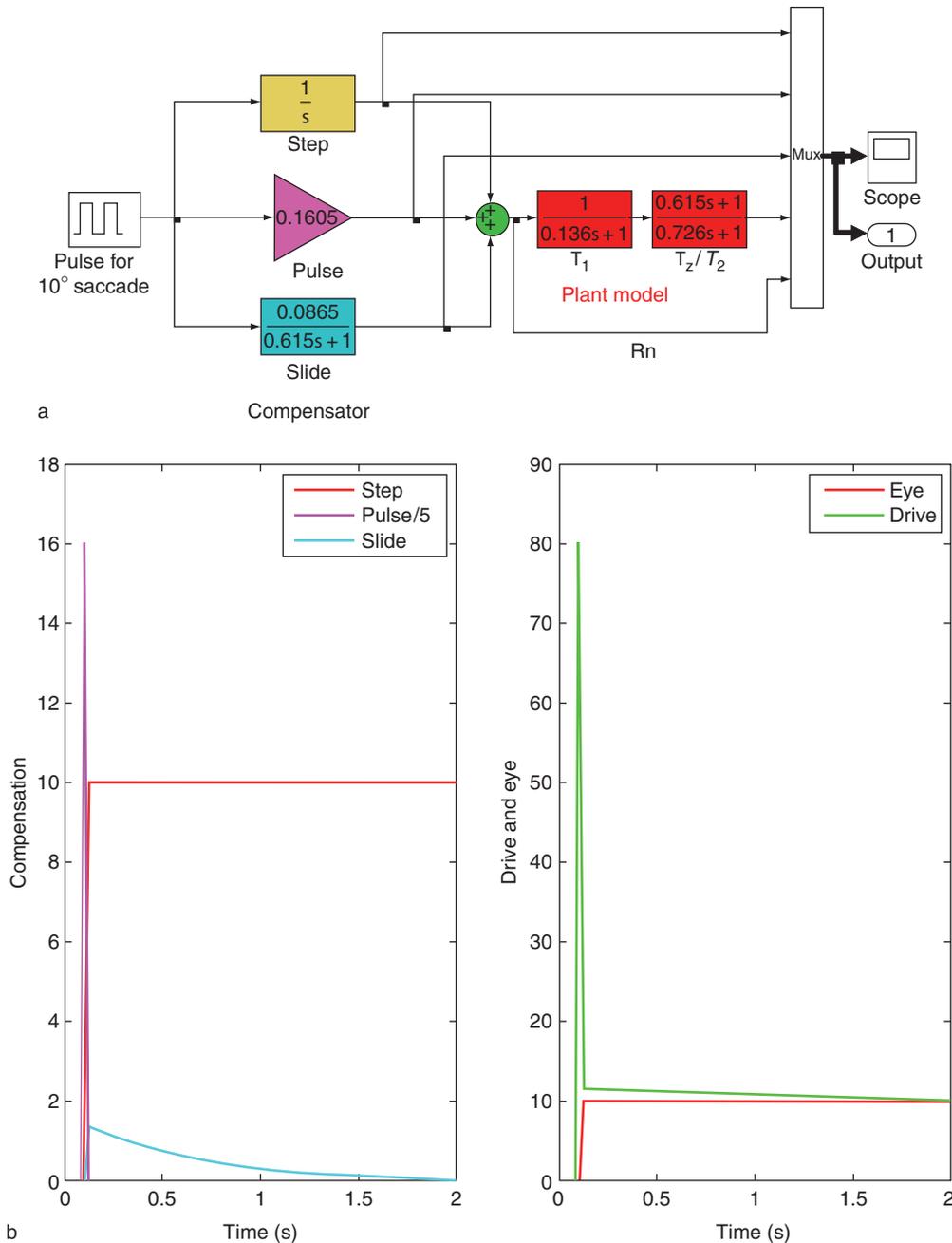
Figure 2 Simulation of the simple plant model and its neural compensator from **Figure 1**: (a) implementation of the model in MATLAB/SIMULINK visual programming environment; (b) output of a simulation run. In (a) The generator on the left puts out a pulse 500°/s high for 0.020 s (which should make an eye movement of 10°). The compensator consists of three parallel operators: an integrator (yellow), an amplifier (purple), and a low-pass filter (blue). The plant model (red) is made by combining a low-pass filter block (for T_1) with a lead–lag block (for T_z and T_2). Other blocks (white background) allow Simulink to plot the simulation output in real time (scope) and send it to the MATLAB workspace (output). In (b), the left panel shows the step, pulse (divided by 5 to scale it to fit with the others), and slide; the right panel shows the pulse–slide–step drive (green) and the output of the plant (eye). Note that the pulse part of the drive signal is about eight times bigger than the step part. Most of the energy in this pulse is eaten up by the muscles to overcome their own viscosity, and only a small part is passed to the tendon. Mux, multiplexer.

A major simplification for determining the innervation occurs in one-dimensional (1-D) models because in 1-D orientation is the integral of angular velocity, just as position is the integral of velocity. Thus, in 1-D the torque vector determined by the pulse of innervation and the torque determined by the slide and step are collinear (necessarily, because there is only one axis). Thus, if the pulse of innervation were already computed, obtaining the step of innervation would require simply the mathematical integration of

that pulse. Similarly, the slide could be obtained as the response of a leaky integrator to the pulse.

Three Dimensions

Three-dimensional rotations are more complicated than 1-D rotations because 3-D orientation is not the integral of angular velocity (i.e., rotations are not commutative). However, three dimensional muscle torques are commutative vectors, and the final eye orientation is thus determined by the balance of torques between active and passive tissues. However, during a 3-D movement, the eye deviates from a straight trajectory (e.g., with torsional blips) because of the lack of commutativity, unless the torque is modified by the current eye orientation. Torque could be modified by changing the innervation or changing the mechanics of the orbit.

Muscle Pulleys

Originally it was assumed that the muscle always went straight from its origin to its insertion, even as the eye looked into eccentric orientations. Thus, the pulling direction of the muscles would not change much as the eye moved (**Figure 3(a)**), and the brain would have to compute a new torque axis for each ocular orientation. However, in 1989 Miller used magnetic resonance imaging (MRI) of human orbits to show that the belly of the muscle did not move as the eye turned, thus creating an inflection point in the eye muscle path. Further research by Demer and colleagues found that this point corresponded to fibromuscular pulleys, which encircle the belly of each muscle (except the superior oblique, which goes through the bony trochlear pulley). Quaia and Optican showed in 1998 that, if the pulleys are in the right place (approximately halfway between the equator and the posterior pole of the eye), the amount by which the axis of action tips for the pulse will be just enough to compensate for the noncommutativity of 3-D rotations (**Figure 3(b)**), without affecting the final orientation set by the step. This makes the 3-D angular velocity vector approximately equal to the derivative of the 3-D orientation vector. That means the brain could calculate innervation in 3-D as if it were in 1-D: the pulse can simply be the derivative of the desired change in eye orientation, and the slide and step can be obtained from the pulse through (leaky) integration.

As the eye turns, the position of the pulleys must also change, so as to keep the geometrical relationship between the pulley and muscle insertion constant. If the pulley did not move, as the eye rotated the muscle insertion would run into the pulley. Fortunately, all that this requires is that the pulleys move
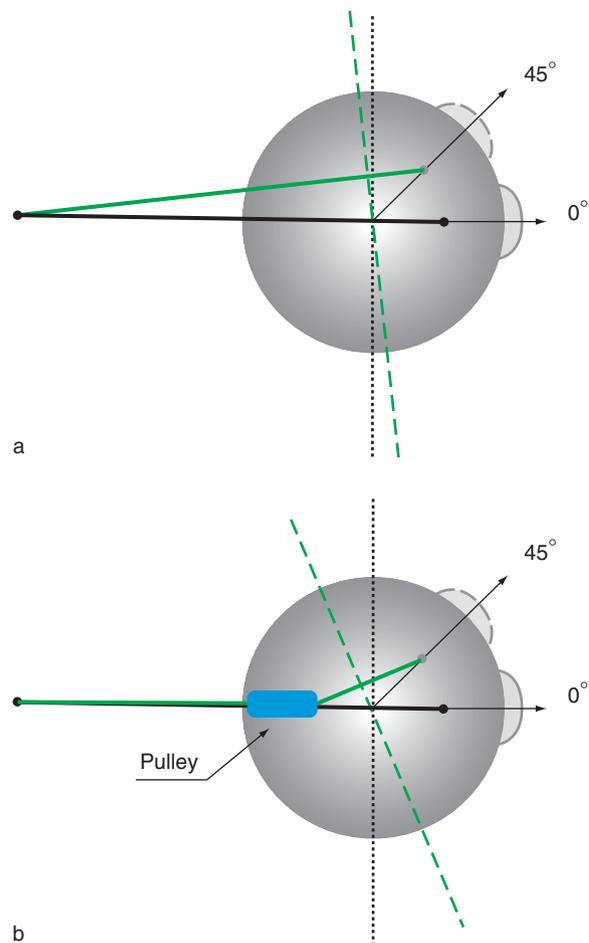

Figure 3   Effect of eye orientation on the axis of action of extraocular muscle: (a) without pulleys; (b) with pulleys. In (a), if there were no impediments the muscle would slip as the eye turned, so that the muscle always took the shortest path to the insertion. In this case, even for a 45° elevation, the axis of action (green dotted line) tips very little (~6°). In (b), a specialized structure in the orbital tissues, called a pulley (blue), holds the muscle in place as the eye turns. In this case, for a 45° elevation, the pulley redirects the axis of action of the muscle (green dotted line) by tilting it in the same direction as the globe has moved, but by only half as much (22.5°). Adapted from Quaia C and Optican LM (2003) Three-dimensional rotations of the eye. In: Kaufman PL and Alm A (eds.) *Adler's Physiology of the Eye: Clinical Application*, pp. 818–829. New York: Mosby.

back as much as the globe rotates in its direction. This may be facilitated by the fact that the orbital layers of the extraocular muscles insert on the pulley itself. Thus, as the muscle contracts, it moves both the eye and the pulley.

Eye Movement Models

Eye movements can be divided up into subtypes by their speed, latency, and whether they are voluntary

or reflexive. These include vestibular, optokinetic, smooth pursuit, saccadic, vergence, and short-latency ocular following movements. Many parts of the brain respond to several eye movement types, so the brain does not necessarily process these types independently. Saccades are the most demanding movements to model from the point of view of dynamics, and they are used as an oculomotor model prototype next.

Final Common Integrator

The task of computing oculomotor innervations comes down to generating an appropriate pulse of innervation and then integrating that to obtain a slide and a step for plant compensation. Skavenski and Robinson showed that the phase lag introduced by the brain from the head-velocity signal on the vestibular afferents to the oculomotor neurons was ~90°. This indicated that the brain must be performing an integration of desired eye velocity to get the eye position signal needed by the motor neurons. This observation gave rise to the concept of a final common path, which includes the motor neurons and the neural integrator. Any eye movement command (e.g., vestibular, pursuit, or saccade) need only generate an eye velocity command and feed it into the final common path. All the other mechanisms from there to the orbit are shared by all systems. This sharing makes the neural integrator a key element of all oculomotor movements. Lesion and single-unit recording studies have localized the neural integrator to the vestibular nuclei, midbrain interstitial nucleus of Cajal (NIC; for vertical and torsional components), the brain stem nucleus prepositus hypoglossi (NPH; for horizontal components), and corresponding parts of the cerebellum.

Saccadic System Functions

The saccadic system (**Figure 4**) takes target location in retinotopic coordinates and additional information about the movement's context (such as the orientation of the eyes and motion of the target) and produces the innervation required to get the eye (E) on target (T). In essence, the saccadic system must consist of two functional subdivisions, a sensory-to-motor transformation that changes the input signals into a desired eye orientation signal, E_d, and an inverse model of the plant that can convert an eye orientation into the corresponding innervation needed to drive the eye until $E = E_d$.

Robinson realized that an inverse plant model can be made simply by placing a forward model of the plant in a feedback loop around a high-gain amplifier (**Figure 5**). This loop defines a new signal, the instantaneous, or dynamic, motor error (m_e). It is the difference between the desired eye orientation change and
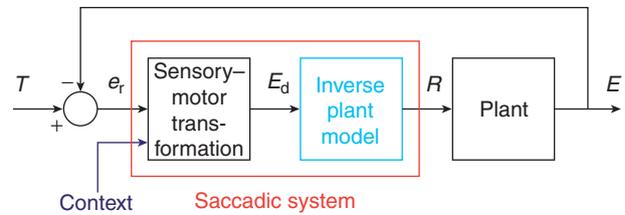

Figure 4   The saccadic system must consist of two functional subdivisions: a context-dependent sensory-to-motor transformation that changes the input signals into a desired eye orientation signal, E_d, and an inverse of the plant model that converts desired eye orientation into the innervation needed to drive the eye until $E = E_d$.
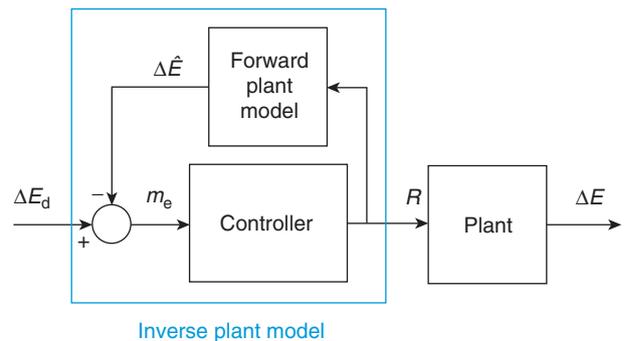

Figure 5   Local feedback loop, proposed by Robinson, that corresponds to the inverse plant model in **Figure 4**. The feedback loop contains a forward model of the plant that takes innervation as an input and puts out eye orientation. (The forward plant model is represented by a resettable leaky integrator.) The forward plant model's output, $\Delta \hat{E}$, is called the efference copy of the change in eye orientation, ΔE. The comparator subtracts efference copy from desired change in orientation to calculate dynamic (i.e., changing during the movement) motor error, m_e.

the current estimate of the eye orientation change $(\Delta E_d - \Delta \hat{E})$ obtained by feeding a copy of the innervation that goes to the plant into the forward model (the forward model can be well approximated by an integrator that resets to zero before every saccade). The summing junction that subtracts $\Delta \hat{E}$ from ΔE_d is called a comparator. If the gain of the controller is high enough, the negative feedback loop drives the output of the comparator (motor error) to zero and the eye's orientation becomes equal to the desired orientation. The sensory-to-motor transformation is much more complicated because it must take into account the current orientation of the eyes, whether the target is moving or stationary, what behavior is desired, and so on. How this transformation is carried out is not well understood, but by moving from a lumped to a distributed model various mechanisms can be proposed.

Brain Areas and the Lumped Model

The brain can calculate the needed innervation if it knows the change in eye orientation that is desired. There are two obvious areas in the brain that could provide this signal: the superior colliculus (SC) and the cerebellum (CB).

Superior colliculus It is not easy to incorporate the SC into a lumped model of the saccadic system as the source of the desired eye movement for three reasons. First, the SC contains a spatially coded map. However, the feedback signal that comes back to the comparator (**Figure 5**) is temporally coded. Thus, the SC output must be converted into a temporal code before it can feed into the comparator. The second problem is that the locus of activity on the SC encodes the desired target in retinotopic coordinates, not the movement necessary to acquire it. The third problem is that the SC cannot be the only input to the saccadic system because, after SC lesions, saccades are accurate, although they start with a longer latency and have a lower peak speed than normal. This suggests that another area must cooperate with the SC to make a movement.

Oculomotor cerebellum The CB is the area most concerned with making accurate eye movements. The oculomotor vermis (OV) and the caudal part of the fastigial nucleus (cFN) are involved in controlling saccades.

Just as SC-receptive fields form a map of visual space, the cells in the CB form a map of movement space. This map is not as well defined as the SC map, and many cells participate in almost every movement. To aid in interpretation more than to be descriptive, the midline CB can be represented as two maps (**Figure 6**). The vermis is the only cortical structure in the brain that is continuous across the midline. When unfolded, it is actually much longer than it is wide, but this conceptual map simply represents small saccades toward the center of the vermis and larger saccades toward the edges. The only output from the cerebellar cortex is the inhibitory axon of the Purkinje cell. It projects directly to the ipsilateral cFN.

The cFN in primates is a very small structure. Although it is near the midline, the left and right nuclei are not interconnected. There are inhibitory cells that project out of the cFN, but they go only to the inferior olive and are not discussed here. The outputs from the excitatory cells in the cFN cross over the midline and go to the brain stem. In particular, they project to the contralateral medium lead burst neurons (inhibitory burst neurons (IBNs) and excitatory burst neurons (EBNs)) in the brain stem.
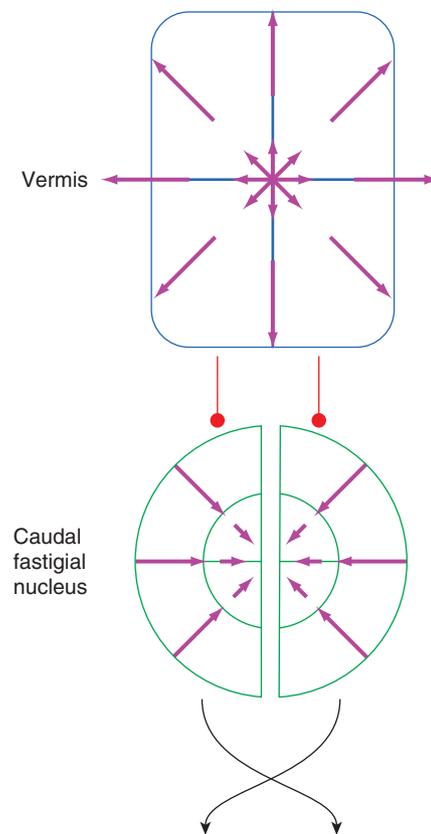

Figure 6 Conceptual maps in the cerebellum. For modeling, the cerebellum can be regarded as having two areas: the cortex and the deep cerebellar nuclei. The areas related to horizontal movements are the midline structures (posterior vermis and caudal fastigial nucleus, cFN). The vermis has a coarse map of saccade size and direction (purple arrows). The cFN is quite small, and the cells receive convergent input from many Purkinje cells. Thus, it is unlikely that they contain any kind of topographical map. Nonetheless, it is convenient to think of the cFN as repeating the map in the vermis but with the sign reversed. The output of the fastigial nucleus crosses over and goes to the brain stem on the other side. Thus, activity in the left nucleus corresponds to rightward saccades. The output of the vermis goes to the ipsilateral fastigial nucleus, but the output comes only from the Purkinje cells, which are inhibitory. Thus, a rightward saccade requires a pause in the left vermis. Correspondingly, an increase in activity in the right vermis inhibits the right fastigial nucleus, unbalancing the fastigial output to the brain stem. This results in a rightward movement.

The direction associated with activation of Purkinje cells on the left is thus a movement to the left because it inhibits the left cFN which projects to rightward premotor burst neurons (MLBNs), thus tipping the balance toward a leftward movement. In contrast, activation of the left cFN causes a rightward eye movement. It thus makes sense to extend our conceptual map of the CB to include vertical movements by placing up and down cells at the top and bottom of the vermis and down and up cells at the top and bottom of the cFN, respectively (**Figure 6**).

As more data on the role of cells in the midline CB, and related areas such as the interpositus deep cerebellar nucleus, become available, these conceptual maps will need to be updated to maintain a closer isomorphism with the brain. However, the concept of a map, at least in the cerebellar cortex, will probably still be useful.

Neuromimetic Model of the Saccadic System

A lumped model simplifies the description of a system by using simple connections of physically meaningful signals (e.g., target position and eye velocity) among complex elements (e.g., a resettable integrator). Unfortunately, the signals flowing through the brain often do not correspond to physical signals, and individual neurons are not very complex information processors. Instead, the complexity of the brain comes from the massive interconnection of many simple neurons. Thus, to build a model of the saccadic system that incorporates some of this complexity while maintaining its physiological isomorphisms with the brain requires a distributed model. If the distributed model contains elements with anatomical correspondences with the brain and if the elements carry signals observed in neurophysiological recordings, the model can be called neuromimetic.

Now that the conceptual visual (SC) and motor (CB) maps have been presented, they can be combined into a distributed neuromimetic model of the saccadic system (**Figure 7**). The cerebral cortex is the source of visual information, cognitive choices about what the eyes should look at and the context of the movement. The drive for the eye comes from two parallel pathways, the SC and the CB. These project down to the brain stem areas for controlling the eye muscles. The cerebrum and the SC cooperate to select the target. No specific signal to start the movement is needed. Once the SC determines the target, it stops sending a veto signal to the omnipause neurons (OPNs), which inhibit the saccade generator. The SC also sends a fixed-direction drive signal (in retinotopic coordinates) to the brain stem. The information about the selected target and the movement's context (e.g., target is moving) are sent to the CB. The CB sends a variable-direction drive (or pilot) signal to the brain stem. The sum of the drive from the CB and the SC form the velocity command that moves the eye. This command is also fed back to the CB. The velocity signal can be used to update the current state of the CB. If necessary, the CB can then change the direction of the pilot signal to bring the eyes to the target. When the CB decides that the movement is over, it sends a choke signal to the brain stem (presumably to contralateral IBNs) that cuts off the input to the motor neurons, stopping the saccade.
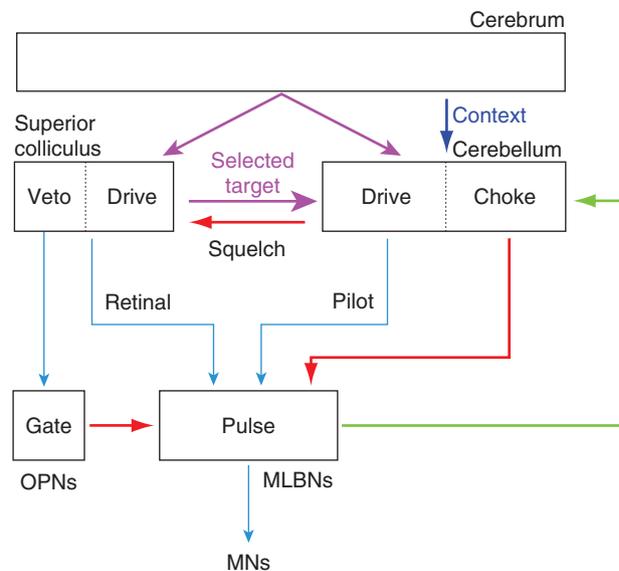

Figure 7 Distributed model of the saccadic system. The cerebral cortex and the superior colliculus (SC) determine what target to look at. The SC stops its input signal to the omnipause neurons (OPNs), shutting them off and disinhibiting the saccade generator. The SC also sends a drive command to the brain stem premotor burst neurons (MLBNs) to get the eye moving. The cerebellum sends a variable-direction drive (pilot) signal to the brain stem. The sum of the drive from the cerebellum and the SC form the velocity command that moves the eye. If the cerebellum decides that the movement is over, it sends a choke signal to the brain stem (via the contralateral inhibitory burst neurons) that cuts off the input to the motor neurons (MNs), stopping the saccade. The output of the SC is directionally fixed, and so it is called a directional, or retinal, drive. The cerebellum receives a feedback signal (efference copy of eye position and/or velocity) during the movement and, thus, can adjust its output to steer the saccade, so its output is called a pilot drive. When we compare this feedback loop with the one in **Figure 5**, it clear that the key element of Robinson's model, the local feedback loop, goes from the MLBN to the cerebellum (green arrow). Thus, the role of the forward plant model in **Figure 5** is played here by the cerebellum.

The neurons in the left SC fire for rightward targets, and the neurons in the right SC fire for leftward targets. However, cFN neurons fire for both ipsi- and contralateral saccades. How can the same neuron fire for both directions of movement and accomplish anything? One possible interpretation is suggested by the observation that cFN neurons fire with a shorter latency for contra- than for ipsilateral saccades. The contralateral burst is time-locked to the start of the movement, but the ipsilateral burst occurs with a high degree of variability, usually near the end of the movement. This timing difference of cFN activity can be exploited. In the brain stem, there is some evidence that IBNs are stronger than EBNs. This breaks the anatomical symmetry and allows the early contralateral output to be a drive signal, whereas the later ipsilateral output acts as a choke.

Thus, for a rightward saccade, the left OV must pause and the left cFN must fire, which drives the right IBNs and EBNs. The right EBNs drive the right lateral rectus and left medial rectus. The right IBNs cross over and inhibit the EBNs, IBNs, and motor neurons on the left side. Near the end of the saccade, the right vermis pauses and the right cFN fires. The output from the right cFN crosses over and drives the left IBNs and EBNs. At this time, the left IBNs and EBNs are being inhibited by the right IBNs. However, if we assume that the excitation from the right cFN is more powerful than the inhibition from the right IBNs on the left IBNs, it will begin firing. The left IBNs cross over to the right side and inhibit the right IBNs, right EBNs, and right motor neurons (probably presynaptically). This crossed inhibition chokes off the drive to the motor neurons and stops the movement.

During the movement, the CB is also sending a signal to the SC that inhibits the SC more and more toward the end of the movement (squelch). Thus, the SC is inside a feedback loop, but it is not a classic error control feedback loop. It simply causes the SC output to decay somewhat during the movement, which may make it easier to start a new saccade after the current one is completed.

A diagram of how the system works is shown in Figure 8(a). On the left side, the bullet-shape represents the retinotopic map on the SC. On the right side, the upper rectangle represents the vermis (continuous across the midline), and the two squares represent the left and right cFN oculomotor regions (FOR). The appearance of the target causes a locus of activity somewhere in the SC (blue disk). This is communicated to the CB, but the projection diverges widely and, thus. cells in most of the vermis become active, which shuts down the FOR (red arrows). The innervation for the movement is the sum of the outputs from the SC and the CB (with the sign reversed for the ipsilateral FOR, making it a choke signal). This sum drives the brain stem burst neurons, which in turn provide feedback to the vermis. During the saccade, the feedback must cause some kind of updating in the CB, which causes different parts of it to pause as the movement progresses. This can be thought of as a spreading wave across the CB cortex. However, it is important to keep in mind that the mechanism underlying this apparent spread is unknown. Other mechanisms (such as a population of neurons with different thresholds) could also cause the activity in the ipsilateral vermis to pause at the right time. Nonetheless, it is important to realize that the spread of the pause in the vermis cannot be generated simply by some kind of diffusion process within the CB or by transport delay (along the parallel fibers that cross the midline). If that were the case, the movement would

be unable to compensate for motor noise in the system. The only way the CB can steer the movement to the target is by processing instantaneous feedback information.

An example of how movement context is incorporated into the model is shown in Figure 8(b). Suppose that the target appears on the horizontal meridian, at the same eccentricity as in Figure 8(a). Then, the locus of activity in the SC would be same in both cases (blue disk). However, the CB also receives information that the target is moving. Based on its previous experience, the CB has learned that to acquire this target the initial locus of activity must be moved a bit down and to
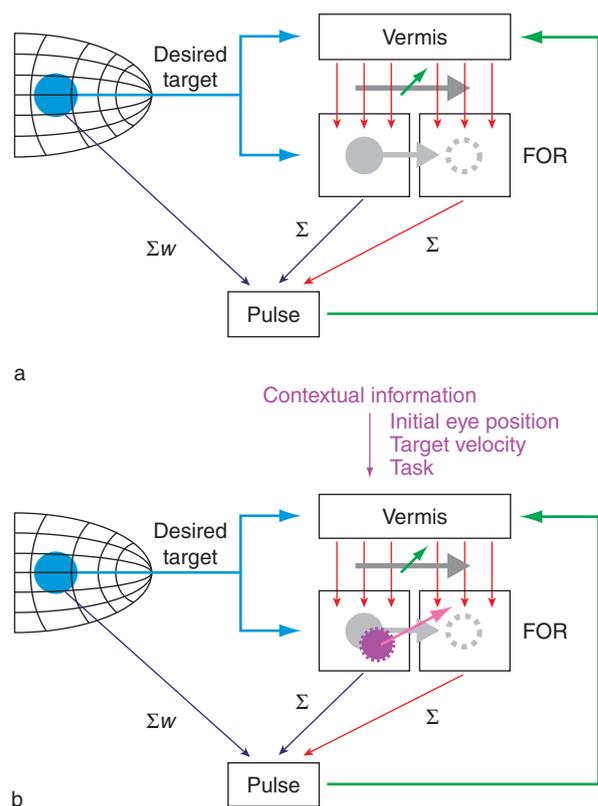

Figure 8 Interaction of drive signals from superior colliculus (SC) and cerebellum determine the saccade (two-dimensional maps of the SC and cerebellum show where activity must be for the desired eye movement): (a) under normal circumstances; (b) when contextual information (e.g., target velocity, behavioral commands, or initial eye position) indicate that a different movement is called for. In (a), the output of the SC and the corresponding output of the cerebellum are simply summed (with the sign of the choke reversed, red arrow). Feedback (green arrows) causes the activity at different places in the vermis to pause at the right time (as if a wave of inhibition were spreading across the vermis, gray arrow). In (b), the initial locus of the pause in the vermis is displaced, creating a new drive signal from the fastigial oculomotor region (FOR), as shown by the magenta disk. During the eye movement, feedback drives the spread of the Purkinje cell (PC) pause along the magenta arrow. Thus, the total output (sum of SC and cerebellar drives) gets the eye on target. $\sum_W$, weighted sum.

the right (magenta disk). The sum of the SC and CB outputs now causes the eye to move up and to the right. Feedback causes the pause in the vermis (or burst in the FOR) to move up and to the right (magenta arrow). The eye is thus successfully steered to it rendezvous with the moving target.

The Brain Does Not Have a Desired Eye Movement Signal

The division of the drive signal across different areas in the distributed model has profound consequences. The most important question is: where in the brain is the desired eye movement represented? Let us consider in more detail a saccade to a moving target. **Figure 9(a)**
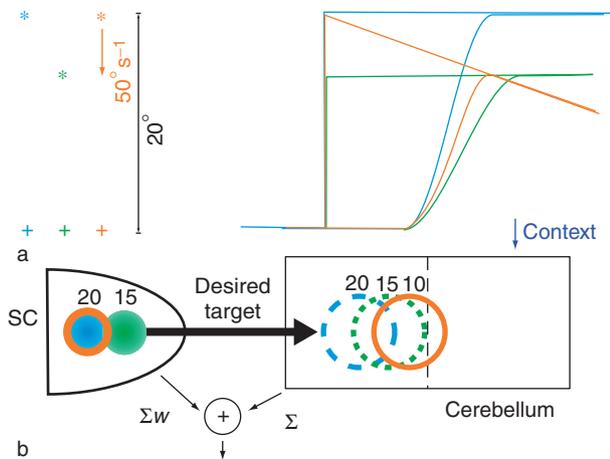

Figure 9 The brain's representation of the desired target is distributed: (a) the target configurations and the corresponding eye movements; (b) diagrams of the superior colliculus (SC; retinotopic) and cerebellum (CB; motorotopic). In (a), suppose the saccade is made from the fixation point (blue plus) to a stationary target at $20°$ (blue asterisk). The representation of the target in the (SC) is at the $20°$ locus (blue disk). The representation of the target in the CB is also at the $20°$ locus (dashed blue circle). The resultant saccade goes to the target (blue trace). If the saccade is made to a stationary target at $15°$ (green), the locus of activity in the SC is more rostral and the locus of activity in the CB is more medial (green disk and dotted circle). However, when the saccade is made to a moving target that appears at $20°$ and moves toward the fixation point at $50° s^{-1}$, the loci of activity in the SC and CB differ (b). If the saccade occurs in 100 ms, the target will have moved closer, so only a $15°$ saccade needs to be made. In this case, the locus of activity in the SC is still at the $20°$ site (orange ring) because the SC encodes the location of the appearance of the target. The CB receives the location of the original appearance, but it also receives information about the velocity of the target (Context). Based on previous experience, the CB has learned that it needs to make only a $10°$ contribution for this movement. Thus, activity starts at that site (orange ring). The total outputs of the SC and the CB are now correct to make a $15°$ movement. However, neither area alone encoded the actual size of the movement. The most fundamental signal of the lumped model, the desired eye movement, thus does not exist in the distributed model. $\sum_{W}$, weighted sum.

shows the target configurations and the corresponding eye movements. Blue symbols and traces show a saccade to a stationary target at $20°$ eccentricity. Green symbols and traces show a saccade to a stationary target at $15°$. Orange symbols and traces show a saccade to a target (*) appearing at $20°$ and moving toward the fixation point (+) at $50° s^{-1}$. If the delay between target appearance and the saccade is 100 ms, then the target will be $5°$ closer to the fixation point at the time of the saccade than when it appeared. Thus, to rendezvous with the moving target the eye needs to make only a $15°$ saccade (and then pursue the target).

Figure 9(b) shows a diagram of the SC (retinotopic) and CB (motorotopic) maps. From the results just described, we know where the activity in the SC and CB must be localized during saccades to stationary targets. For the $20°$ stationary target, the locus of activity in the SC is at the appropriate caudal site (blue disk), and in the CB the pause is at the $20°$ site (blue dashed circle). If the effective drive is the average of the retinal and pilot signals, then a $20°$ saccade results. The same argument reveals that the loci in the SC and the CB must be at the $15°$ sites for the shorter saccade to a closer target (green disk and green dotted circle).

Now, consider the orange target. The target appears at $20°$ eccentricity. Thus, the locus of activity in the SC, which knows only which target was selected in retinotopic coordinates, must be at the $20°$ site (orange circle). Where is the locus of the pause in the CB? If it were also at the $20°$ site (blue dashed circle), a $20°$ saccade would result, which would be too large. If the locus in the CB were at the $15°$ site (green dotted circle), which is the calculated size of the desired movement based on the target's motion, then the saccade would still be too big because the SC is still putting out a command for a $20°$ saccade. Instead, the pause in the CB must be at the $10°$ site (orange circle) because, if the SC puts out too much drive, the CB must put out a smaller drive and/or choke sooner. The average of those two drives gets the eye on target. Thus, neither the SC nor the CB encodes the size of the movement. Desired movement size is distributed across several brain structures and is an emergent property of the neuronal circuit.

The distributed model does not contain a signal encoding the desired eye movement. Why is this so important? Because all classical models based on control theory or optimization theory work by driving a motor error signal to zero. However, the existence of an instantaneous motor error signal depends on a comparator to calculate the difference between desired and accomplished eye movements (**Figure 10(b)**). Since Robinson's original proposal of the local feedback loop in 1975, no neurophysiological evidence
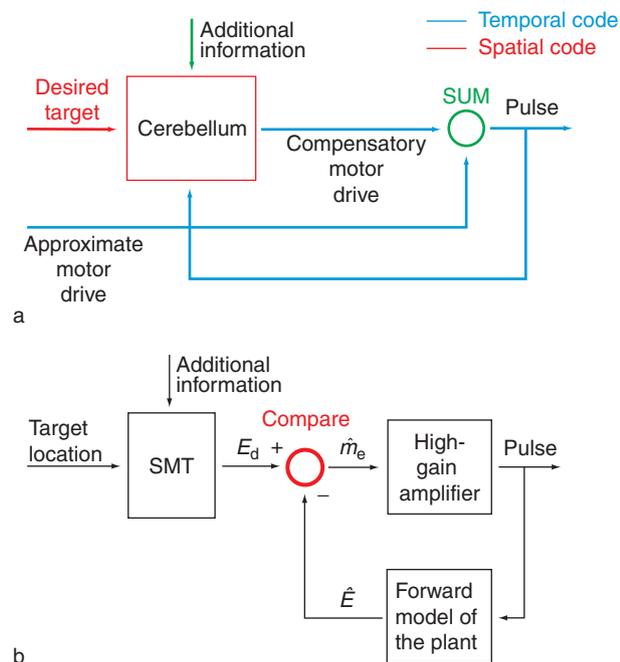
Figure 10 Comparison of models of the saccadic system: (a) distributed; (b) lumped. In (b), the lumped model, the signals all encode physical values. The input to the pulse generator is the desired eye orientation (E_d). It is compared to the efference copy of eye orientation, producing motor error (m_e). However, the distributed model of the saccadic system (a) has no signal corresponding to the desired eye movement. Thus, no comparator is present in the distributed model and there is no motor error signal. In the distributed model, the information processed in the cerebellum is spatially coded (red), but in the downstream areas information is temporally coded (blue). Note that the two types of signals never mix (i.e., there is no comparator). Instead, the temporal activity of the feedback signal causes an updating of the spatial locus of activity in the cerebellum. $\hat{E}$, estimate of eye orientation; STM, sensory-to-motor transform.

of a neuronal population encoding the desired eye movement, let alone dynamic motor error, has ever been found. The neuromimetic, distributed model does not use a desired eye movement signal, and thus it cannot compute a motor error; hence, it does not need a comparator. Instead, the model uses a desired target signal in retinotopic coordinates and knowledge of the current context of the movement, and it works because, after repeated saccades, the CB learns to make the correct contribution, on average, in that situation. Instantaneous feedback of an efference copy signal allows the CB to compensate for motor noise on an individual saccade by updating the locus of activity in the CB. Thus, the brain does

not appear to have, and does not need, either a motor error or a desired movement signal.

See also: Brainstem Control of Eye Movements; Cerebellum and Oculomotor Control; Cerebellum: Models; Cortical Control of Eye Movements; Neural Integrator Models; Oculomotor Control: Anatomical Pathways; Pursuit Eye Movements; Saccade–Pursuit Interactions; Saccades and Visual Search; Saccadic Eye Movements; Superior Colliculus; Target Selection for Pursuit and Saccades.

Further Reading

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