# Eye Movements

The neural circuitry underlying eye movements has been thoroughly investigated. As a result, scientists can turn to the eye movement system as a model for motor control, as all aspects of controlling movements are reflected in the control of eye movements. The eye movement system informs scientists about how movements are planned, how they are executed and how they are adjusted following unpredicted mechanical events or in anticipation of predicted mechanical events, occurring in the body or externally.

**Types of eye movements.** There are two classes of eye movements: those that stabilize the retinal image and those that align the fovea with a visual target (as an aside, consider the following interesting question: why have we evolved a fovea?). The vestibulo-oculomotor reflex (VOR) and optokinetic nystagmus movements belong to the first class. They compensate for head movements and movements of the visual world, respectively. While both movements serve to stabilize the visual image, they process two different inputs. The VOR is concerned with vestibular input that signals head movement (this could destabilize the visual image). The nystagmus movements are concerned with visual input that signals the motion of the visual world.

Saccades, smooth pursuit movements and vergence movements belong to the second class of eye movements.

**Eye muscles.** Each eye is controlled by six extraocular muscles. Neural commands to the muscles are carried by three cranial nerves, so the spinal cord isn't involved. Note that damage to one of the three cranial nerves affects a subset of eye movement directions and not all directions (e.g. the abducens nerve (VI) innervates only the lateral rectus muscle, which serves to turn the eye outward).

**Phasic and tonic signals.** If one were to record activity from a neuron innervating an eye muscle, one would observe that the neural activity is directly proportional to the position and velocity of the eye movement. When an eye moves, there is an initial burst of neural activity, the frequency of which determines the velocity of the eye movement. Following the burst, there is a period of sustained activity, the frequency of which determines the final position of the eye. These are referred to as the "pulse" and "step", respectively. The magnitude of the pulse (a phasic signal) determines the speed of the saccade. The duration of the pulse determines the duration of the saccade. The magnitude of the step (a tonic signal) determines the amplitude of the saccade (i.e. how far the eye moves). The direction of saccades is in the control of the "gaze centres", located in the reticular formation.

**Gaze centres.** The circuitry underlying the contol of saccades is well described. Basically, nuclei in the reticular formation (gaze centre), provide the phasic signal, while other brainstem nuclei provide the tonic signal. The gaze centre also provides an inhibitory signal (essentially the opposite of the phasic signal) to the antagonist eye muscles, allowing for a fluid saccade in the desired direction. The gatekeeper of all this is yet another gaze centre nucleus, which contains omnipause neurons. These neurons perpetually inhibit the gaze centre neurons providing the phasic signal. The sequence of neural activity that results in a saccade can occur only if the omnipause neurons are inhibited. The superior colliculus is responsible for this, providing inhibitory input to the omnipause neurons and a motor command to the neurons that provide the phasic signal.

**Superior colliculus.** This is a midbrain structure. It sends inputs to the horizontal and vertical gaze centres. The superior colliculus (SC) serves the purpose of providing motor commands to move the eyes to a desired location. The SC is topographically organized for saccadic movements. Amplitude is represented along a rostral-caudal axis. Moving caudally, SC neurons code for saccades or progressively greater amplitude. Neurons at the caudal pole of the SC code for big saccades; neurons near the rostral pole code for little saccades. Neurons at the rostral pole code for zero amplitude saccades. In other words, they're fixation neurons. The direction of saccades is represented along a lateral-medial axis.

So, the SC is the seat of motor control for the eyes. Everything downstream of the SC (like the gaze centres, the cranial nerves and the eye muscles themselves) just obeys the commands of the SC. What else is the SC for? It's involved in the production of reflexive saccades and express saccades.

**Higher saccadic centres.** Is there more to the control of eye movements than just the SC? Yes, of

course. Activity of the SC is influenced by higher saccadic centres in the cerebral cortex, which directly innervate the gaze centres and the SC. They also affect the SC indirectly, through the basal ganglia.

The frontal eye fields (FEF) control saccades through their connections to the SC and to the gaze centres. The FEFs are organized topographically, coding for saccade amplitude and direction. As a higher saccadic centre, the FEFs not only provide a motor command, they are also involved in selecting a visual target. The FEFs provide input to the gaze centres directly. They also affect the gaze centres indirectly, through connections to the SC and to the caudate (part of the basal ganglia), which in turn indirectly connects to the SC.

The basal ganglia also contain higher saccadic centres. The substantia nigra pars reticulata tonically inhibits the SC. This puts a stop to unwanted reflexive saccades. Prior to generating a purposeful, voluntary saccade, the caudate inhibits the nigra. Recall that the caudate receives input from the FEF.

The prefrontal cortex is thought to be a higher order brain area, involved in complex things like planning and memory and so on. This is certainly reflected in the control of eye movements. Activity of neurons in the dorsolateral prefrontal cortex (DLPFC) reflects the visual detection of a target and the movement of the eyes. Interestingly, their activity also reflects memory of transiently detected visual targets. These memory neurons show location-selectivity. In other words, some DLPFC neurons are responsible for remembering the location of visual targets only in one movement direction.

**Clinical implications.** Is there a reason why anyone should care about the eye movement system? This is a question that can be posed for any research topic that is driven purely by curiosity. However, the study of eye movements is frequently conducted by applied researchers and so it is of interest to a great many. Deficits in the control of eye movements provide a window into many diseases, from Alzheimer's to schizophrenia.

## Major Themes/Questions

- What is the significance of topographical organization in the eye movement system?
- What does a given deficit in eye movement control reveal about the eye movement system?
- For a given lesion in the nervous system, is it possible to predict the resulting deficits in eye movement control?
- How can one compare and contrast different types of eye movements?
- Before an eye movement is executed, what sorts of input are required?
- What mechanical events does the eye movement system anticipate and how does the control of eye movements reflect this predicitve ability?