Brain Mechanisms for Active Vision

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Abstract: Active vision refers to the exploration of the visual world with rapid eye movements, or saccades, guided by shifts of visual attention. Saccades perform the critical function of directing the high-resolution fovea of our eyes to any point in the visual field two to three times per second. However, the disadvantage of saccades is that each one disrupts vision, causing significant visual disturbance for which the brain must compensate. Exploring the interaction of vision and eye movements provides the opportunity to study the organization of one of the most complex, yet best-understood, brain systems. Outlining this exploration also illustrates some of the ways in which neuroscientists study neuronal systems in the brain and how they relate this brain activity to behavior. It shows the advantages and limitations of current approaches in systems neuroscience, as well as a glimpse of its potential future.

Though our perception convinces us beyond a doubt that we see the visual world as one coherent whole, we actually see a series of snapshots from which we construct a unified view of the world in our brains. Figure 1, which shows a record of the eye movements of a viewer inspecting the Georges Seurat painting *A Sunday Afternoon on the Island of La Grande Jatte*, illustrates the snapshot process. A record of the viewer’s eye movements is superimposed on the painting. The black lines show the path of the eyes as they move from one part of the painting to another. These rapid eye movements, referred to as saccades, are not only fast but frequent, occurring two to three times per second. The dots at the end of each saccade are visual fixations, the points at which the eyes come to rest. Nearly all of our useful vision occurs during fixations, because the scene is then stationary in front of the eyes. With successive fixations, the brain receives a series of snapshots of different fragments of the scene. From these fragments, we become convinced that we see the whole scene at once.
Why bother with all these saccades? Why not just hold the eye steady and inspect the painting? The reason lies in the resolving power of the retina, the receiving surface within our eyes. The retina is equipped with receptors that respond to light, dark, and color, but it does not have uniform resolution across its surface. The highest receptor density is found in the central region of the retina, called the fovea, which gives us the highest visual resolution and enables us to see small details. Retinal areas outside this central region, responding to light from the periphery, have a lower density of receptors and therefore lower resolution. Thus, the viewer enjoying the Seurat painting is essentially using the fovea to examine the rich detail, jumping from one part of the painting to another. In the three minutes of saccades shown in Figure 1, the viewer examines the details in a substantial fraction of the painting, but never sees the whole scene at once.

How do subjects pick the next object to examine? Using their peripheral retina, subjects can see objects in the field at a relatively low resolution and select ones of potential interest to examine next. This shift of attention from one item to the next accompanies each saccade. This selection is not random: if we look at the saccades superimposed on the Seurat painting (Figure 1), we can see that the trees at the top of the painting and the grass at the bottom are largely ignored; in contrast, faces, dresses, and other significant objects are frequently inspected. The Russian psychologist Alfred Yarbus was the first to note the connection between eye movements and

**Figure 1**
Examination of a Scene with Eye Movements of the Viewer Superimposed

Saccades are represented by black lines, and intervening periods when the eyes are stationary (fixations) are represented by white circles. The human viewer documented here looked at this painting, *Sunday on the Island of the La Grande Jatte* by Georges Seurat, for a period of three minutes. Note that the fixations are not to random locations, but rather to points of likely interest. A print of the painting was projected on a screen in front of the subject. Source: Figure prepared by the author.
He concluded that the location of saccades was a good indicator of the subject’s attentional shifts. Thus, the lines superimposed over the Seurat painting not only show saccades, but shifts of attention as well. Whether a particular object attracts attention is determined by a combination of the salience of the object (its color, motion, shape, brightness) and the subject’s goals at a given moment. Attention is a critical factor in active vision – some might say the most critical factor – because it determines where we look. Furthermore, the neuronal activities in the brain related to shifts in attention and the generation of saccades are closely intertwined. The term active vision, therefore, denotes the active exploration of the visual world with rapid saccadic eye movements from one point to another, each guided by a shift of attention. The goal of these saccades is to bring images to the fovea for detailed analysis. Even though these saccades displace the image of the whole visual field on the retina, the system operates so perfectly that we regard the scene as serenely stable.

Active vision comprises several functions, but of course, it is only a small part of the larger brain systems involved with sensation and motor control. In turn, the puzzle of how these systems operate to produce action is just one of many global questions about how the brain produces all behavior, including learning, memory, and emotion; and even how consciousness arises from brain activity. Considering active vision alone, however, exemplifies the classic approach of reducing the overwhelming complexities of the brain to more easily understood fragments. Galileo had to study the solar system before we could study the universe.

The rest of this essay illustrates sequential steps in the investigation of the brain functions that underlie active vision (as we have outlined it above). The first step is to move from describing the benefits of saccades to specifying the problems that these saccades produce for vision. Next, we consider the logic of a particular brain mechanism called corollary discharge, which might eliminate these problems. We then briefly review our basic knowledge of the brain pathways that process visual stimuli and produce saccadic eye movements in order to locate the source of a corollary discharge. Finally, we consider how the corollary discharge circuits we find act to minimize the disruptions generated by saccades. This is a progress report, evaluating what we know about active vision and what we do not.

Every saccade produces two major problems for vision: blurring of the image and displacement of the image. Blurring occurs when the image of the scene is swept quickly across the retina during each saccade. Image displacement is closely related to blurring: each time the saccade moves the fovea from one part of the visual scene to another, the image on the retina is displaced (imagine a movie camera moving in jerks from one spot to another). For example, suppose you are looking at the dog in the foreground of Figure 1 and make a saccade that moves the fovea to one of the umbrellas. Not only does the image on the fovea change, but everything on the retina is also displaced.

These problems should be devastating to our vision, but they are rarely reported. Some individuals are aware of the blur that occurs during saccades, but no one is aware of the displacement of images on the retina. If these displacements were visible, most people would be seasick from looking at the lurching scene in front of them. These problems are immense because they arise at the very source of all visual knowledge: the retina. The solutions to these problems are also among the brain’s most remarkable feats of information processing.
Philosophers and scientists have speculated for centuries about why we experience visual stability rather than seasickness with rapid eye movements. A solution that emerges from many of their writings is that there must be a mechanism in the brain that warns the sensory systems that a movement is about to occur. With this warning, the sensory systems would be informed of the impending disruption and could compensate for its consequences. Hermann von Helmholtz, one of the premier visual scientists of the nineteenth century, referred to this internal signal as an “effort of will,” while scientists who subsequently studied the problem in the twentieth century referred to it as an “efference copy” or “corollary discharge.” While the labels all represent the same phenomenon, I prefer corollary discharge because it suggests internal information about movement at all levels of the brain, not just at the level of movement output. The logic of the corollary discharge is outlined in Figure 2. The basic principle is that the same sensorimotor processing area that drives the movement—in this case the saccadic eye movement—also produces a copy or corollary of this drive. The signals are identical, but one leads to the movement command, while the other is directed toward other brain areas to inform them that the eye is about to move. Recipient brain areas include those devoted to processing visual information, since they would receive the visual consequences of the saccade. For saccadic eye movements, both the movement command that produces the saccade and the corollary discharge circuits almost certainly originate in the superior colliculus (a subcortical area deep within the brain). The corollary discharge is directed to the cerebral cortex, which covers the surface of the brain and is the site of the highest levels of information processing underlying much of our behavior. The concept of a corollary discharge is centuries old, but it is only in the last decade that we have begun to identify its neuronal implementation in brains similar to our own. But what of the neuronal circuits that underlie the corollary discharge in the human brain? The usual assumption is that such circuits can be identified using brain imaging, particularly functional magnetic resonance imaging (fMRI). However, because fMRI signals are derived from changes in blood flow that are averaged over several seconds, fMRI is not adequate for investigating active vision. Over that period, the eye will have moved four to six times, changing the image on the retina with each movement and precluding fMRI records from capturing the rapid developments of active vision. But if imaging does not help, how do we begin to understand the brain activity underlying our active vision?

The answer is to study the organization of neurons in animals whose vision and eye movements are similar to ours. For active vision, the animal of choice has been the Old World monkey (the Rhesus monkey). These monkeys’ visual discriminations and their range of eye movements are in most cases virtually identical to those of humans. The relevant anatomical connections in human and monkey brains are also remarkably similar. Techniques have been developed to painlessly record from brain neurons in monkeys while they perform a series of visual tasks that reveal changes in the brain during active vision. At this point, it is fair to say that most of what we know of the structure and function of the human brain for active vision is derived from studying the brains of Old World monkeys.

To determine where in the brain the corollary discharge signal originates, we first need to understand the basic organization of the pathways underlying active vision.
Experiments in dozens of laboratories around the world have built up an outline of the brain systems that underlie the analysis of visual input, the transition between visual and motor systems, and the motor output—all key phases of brain activity supporting active vision.

Figure 3 outlines the major pathways in the monkey brain for active vision. Information from the eye passes through a relay nucleus and reaches the primary visual cortex (solid arrows). This visual area includes both the primary visual cortex and at least forty visual areas (not shown) that process different aspects of vision including shape, motion, depth, and color.

From these visual areas, projections go to the highest levels of the cerebral cortex—the parietal and frontal cortex (dashed arrows)—which are more directly related to the control of movement, including saccadic eye movements. Projections from these regions of the cortex then reach many subcortical structures (wide dashed arrows) that lie in the brainstem (roughly the region of the brain between the cortex and the spinal cord). The major target is the superior colliculus, the outputs of which eventually reach the eye motor neurons that activate the eye muscles (solid arrows again).

This outline from retinal input to eye movement output is a condensed version of the complete circuit that constitutes the neuronal basis of our visual perception and visual control of our eye movements. It is a working hypothesis that the neurons in the pathway in the monkey brain have activity identical to that in the human brain. But given that monkeys and humans have similar saccadic output for a given visual input, it is a reasonable hypothesis.

Where in the pathways underlying active vision might a corollary discharge arise? We are looking for an area with projections that convey a copy of the saccadic eye movement command to the cerebral cortex. The superior colliculus is such a site: it is the origin of commands to move the eye and a source of projections back to the cortex. Figure 4 shows the circuit for two corollary discharge pathways from the superior colliculus to the cerebral cortex. One pathway projects to the frontal cortex and originates in the layers of the superior colliculus where neurons are active before each saccade. A second pathway projects to the posterior visual regions of cortex and...
Figure 3
Side View of the Monkey Brain Showing Circuits in the Brain for Visually Guided Saccades

The schematic outline of the pathway to the visual cortex is represented by solid arrows, the intervening pathways to the frontal and parietal cortex and then to the brainstem by dashed arrows, and the motor pathway to the eye muscles again by solid arrows. Source: Modified from R. H. Wurtz, “Neuronal Mechanisms of Visual Stability,” *Vision Research* 48 (2008): 2070–2089.

Figure 4
Two Corollary Discharge Circuits to the Cerebral Cortex

The corollary discharge to the frontal cortex comes from saccade-related neurons in the superior colliculus (SC) and passes through the thalamus. The corollary discharge to the visual cortex is from visual neurons in the superior colliculus (SC) and also passes through the thalamus. Source: Modified from R. H. Wurtz, K. McAlonan, J. Cavanaugh, and R. A. Berman, “Thalamic Pathways for Active Vision,” *Trends in Cognitive Science* 15 (2011): 177–184.
originates from different layers of the superior colliculus in which neurons respond to visual stimuli. As we shall see, these two pathways contribute to solving the problems generated by saccades: the first compensates for displacement on the retina; the second suppresses blur during saccades.

These projections to the cortex, whether from outside the brain (via sensory pathways) or inside the brain (via corollary discharge pathways), have a relay in the largest group of nuclei in the brain, the thalamus. Each of the senses and each internal signal have dedicated nuclei in the thalamus; thus, it is a basic feature of primate brain organization. To use the terms of air travel, every passenger flying to the cortex must change planes at the thalamus.

Having identified two corollary discharge pathways to the cortex, we can now investigate how they contribute to solving the two problems that saccades create for vision: blur and image displacement. The first problem is the blur produced by each saccade. The solution is relatively simple: suppress visual activity during the saccade and blur is suppressed as well. Early in the twentieth century, such suppression was thought to be produced by a “central anesthesia” in which activity during the saccade was simply blanked out. When it became possible in the 1960s to record directly from monkeys’ brains, it immediately became clear that neurons in the brain continue to respond to visual input quite well during saccades. There is, however, some suppression of neuronal activity during saccades, and as is the case for many biological problems, the brain provides more than one way of producing this suppression.

One mechanism that reduces blur is a purely visual phenomenon called visual masking: when a dim object is preceded or followed by a bright object, the dim object is not seen. Similar masking effects can be seen acting on neurons in the primary visual cortex. This occurs only in a well-lit environment, though it is highly effective. It is not specifically related to eye movements, however, because masking of dim objects can occur any time bright objects are also present, even in the total absence of eye movements.

Corollary discharge also contributes to the suppression of neuronal activity in neurons, as illustrated for a superior colliculus neuron in Figure 5. The neuron responds to a stimulus moving in front of the stationary eyes (left) but not when the moving eyes pass over a stationary stimulus (right). The response is only suppressed during the eye movement and the corollary discharge is only present when the eye moves. The suppression therefore is correlated with the saccadic eye movement. This suppression must be driven by a corollary discharge rather than visual masking, because masking only functions in the light and the corollary discharge–related suppression was demonstrated in the dark. Further experiments indicate that this suppression in the superior colliculus is passed through the thalamus to the visual cortex (Figure 4). The suppression can therefore act on the visual processing in the visual cortex that is thought to underlie visual perception.

The second problem for vision that may be ameliorated by the action of a corollary discharge is the displacement of images on the retina with each saccade. But the neuronal mechanisms are substantially more complicated than those used for blur suppression. To understand these mechanisms that may deal with image displacement, we must briefly consider how visual information is organized in the brain. Visual processing starts when the image of a visual scene falls on the retina and forms a retinotopic map; that is, a layout of the visual scene is mapped on the retina just as it exists in reality. As we have already
noted, the problem is that the whole map moves with each saccade and the brain must deal with the disruption in order to produce a stable visual perception. The problem could be solved in at least two ways.

One solution is for the brain to convert the retinotopic map into a spatial map higher in the chain of visual processing and continuously update it. After each saccade, the central part of the current scene would simply be pasted into the map at a location determined by the corollary discharge of the last saccade. This spatial map would be our brain’s reconstruction of the visual world and would be used for all subsequent visual processing of a scene. If our brains employed such a spatial map, the displacement problem would automatically be solved, because no displacement ever occurs on this higher-order spatial map. The fatal problem with this theory, however, is that no evidence for such a map has been identified in the last forty years of research on vision in monkeys. Researchers have found some hints of conversion to spatial coordinates, and the map itself may have been so elusive because of the way it is represented in the brain. But at this time, we lack convincing evidence that a spatiotopic map exists in the brain, despite the simple elegance of this conceptual solution for visual stability.

Another explanation of perceived visual stability is that the retinotopic map is simply updated after each saccade. This idea
is based on experimental observations of single neurons in the parietal and later the frontal cortex. In these experiments, neurons in these regions were found to have limited visual receptive fields (the areas in the visual field where visual stimuli activate neurons). As the monkey fixated on an object, a light was flashed in the receptive field. As expected, this produced a visual response (Figure 6, left). However, as the monkey prepared to make a saccade, a stimulus was flashed in the location that the receptive field would occupy after the saccade. Unexpectedly, this also activated the neuron (Figure 6, right). Why should activity be evoked from the site of the future receptive field even before the saccade was made? One possibility is that this future-field activity provides anticipatory information about an impending saccade and the location of the receptive field. Each of these pieces of information could be provided by a corollary discharge. With this anticipatory signal, the monkey would be forewarned that the impending visual disruption was due to its own saccade and not to something that happened in the outside world. We now know that this anticipatory activity in the frontal cortex is in fact dependent upon the corollary discharge, because if the corollary discharge is perturbed, the anticipatory activity of the frontal cortex neurons is greatly reduced.

The brain mechanisms by which this anticipation might work to produce visual stability are not known. One hypothesis suggests that a match occurs between the anticipatory activity in the future receptive field before the saccade and the receptive field after the saccade (this match can only occur when a saccade is made). While this hypothesis has not been tested experimentally, the necessary components of the hypothesis – a corollary discharge and future field activity – are well established. Clearly, the analysis of neuronal mechanisms is still at an early stage.

In conclusion, active vision is one of the first examples of a system in our brain that is neither sensory nor motor; rather, its whole function depends on the synchrony between visual input, movement output, and systems internal to the brain, such as those for corollary discharge. This organization allows primates to use the high-resolution fovea to examine anything anywhere in the visual field. The price paid is that eye movements generate major problems for the visual system two to three times per second. While these problems originate at the very first stages of the visual system, the solutions do not: they are spread throughout the visual system across wide regions of the brain, including the highest levels of visual processing in the parietal and frontal cortex.

Active vision creates two significant problems – blurring and displacement of retinal images – and there are two identified brain circuits that might contribute to the solutions. Both circuits rely on a corollary discharge derived from the brain areas where commands to move the eyes originate. One circuit extends from the superior colliculus to the visual cortex and may provide an input to the cortex that suppresses blur during saccades. The second circuit connects the superior colliculus and the frontal cortex and may provide the anticipatory activity that warns the frontal cortex that a disruption of visual input is about to occur as a result of the subject’s own eye movements. While we have identified two problems and two circuits, it would be premature to assume that either problem is solved exclusively by these circuits without exploring other possibilities. The work so far should be viewed as an enthusiastic start, not an assured solution.

So far, a corollary discharge has been identified in other branches of visual-motor systems, but it has not been identified in other systems in the primate brain. There
is every reason to believe, however, that a corollary discharge will be found in other systems and at multiple levels within those systems. This expectation is strengthened by the extent to which corollary discharge is used throughout the animal kingdom.

Finally, the process used to explore the neuronal basis of active vision is an excellent illustration of how neuroscientists go about identifying the neuronal mechanisms that underlie behavior. As we have seen, the study begins with the quantification of behavior that is straightforward for active vision, because methods for measuring eye movements and psychophysical measurements of visual perception are both readily available. The second step is to establish a correlation between the measured behavior and neuronal activity in the brain. Analysis of active vision, for example, depends on the extensive correlations established between behavior and neuronal activity undertaken over the last century, first on the visual system and then, when recording eye movements became possible, on the visual-motor system. From what we know now, the example of active vision demonstrates unequivocally that future progress depends first and foremost on knowing the basic organization of the relevant brain circuits. Without that knowledge, treatment of vision-related human disease is likely to proceed at a glacial pace or simply be fruitless.

After an understanding of basic brain systems has been developed, a third step then becomes possible: extracting and identifying a specific circuit – such as that for a corollary discharge – from the massive number of connections within the brain. Once the specific circuit has been identified, neuronal activity in the circuit can be interrupted in order to see how behavior is changed, which allows more specific evaluation of the circuit’s function. The final step, and frequently the most elusive one, is to develop a precise model that represents the elements of the system.

Figure 6
Anticipatory Activity of a Neuron before a Saccade that Might be Related to the Compensation for Image Displacement

Left: Response of a frontal cortex neuron to stimuli flashed in its receptive field during fixation. The smoothed histogram shows the neuron’s response. Right: Response to stimuli flashed at the receptive field’s future location. The flashes occur before the saccade; thus, the responses are in anticipation of it. Such an anticipatory signal informs the brain that the coming visual displacement is the result of the subject’s own saccade, not of some motion in the outside world. Source: Modified from M. A. Sommer and R. H. Wurtz, “Influence of the Thalamus on Spatial Visual Processing in Frontal Cortex,” *Nature* 444 (2006): 374 – 377.
and that predicts its functions: both those known and those as yet unrecognized.

We currently have only a glimpse of the neuronal basis of active vision in the brain. However, even with the limited methods we have now, an understanding of the system’s organization seems reachable. The hope is that active vision becomes an example of how a complex problem is solved by the brain in simple and clever—but not necessarily intuitive—ways.

ENDNOTES


