3

When vision for action fails

The startling visual dissociations we have described in Dee Fletcher point to the existence of two relatively independent visual systems within the brain—one for conscious perception, which is severely damaged in Dee, and another for the unconscious control of action, which is largely preserved. But skeptics could argue that all we have documented in Dee is a case of someone with poor vision. Maybe you do not need as much visual information to guide your actions as you do to perceive and recognize people and objects. Dee's vision might be good enough for picking something up, but not good enough for telling what it is. In other words, maybe there is only one visual system, not two, and in Dee's case it is simply functioning below some threshold level. On the face of it, this might seem like a valid argument. But if it were true, then there shouldn't be any cases of brain-injured individuals who show the *opposite* pattern of deficits and spared visual abilities to that seen in Dee. It should always be conscious perception that suffers first. Yet as we shall see in this chapter, such patients do exist. Moreover, the part of the visual brain that is damaged in these individuals is quite different from that damaged in Dee.

Bálint's syndrome

Even at the beginning of the twentieth century, neurologists were describing cases of patients whose 'visual' problems could be characterized as *visuomotor* in nature. In other words, they were describing cases where a patient had a specific problem in translating vision into action. Later work has gone on to show that

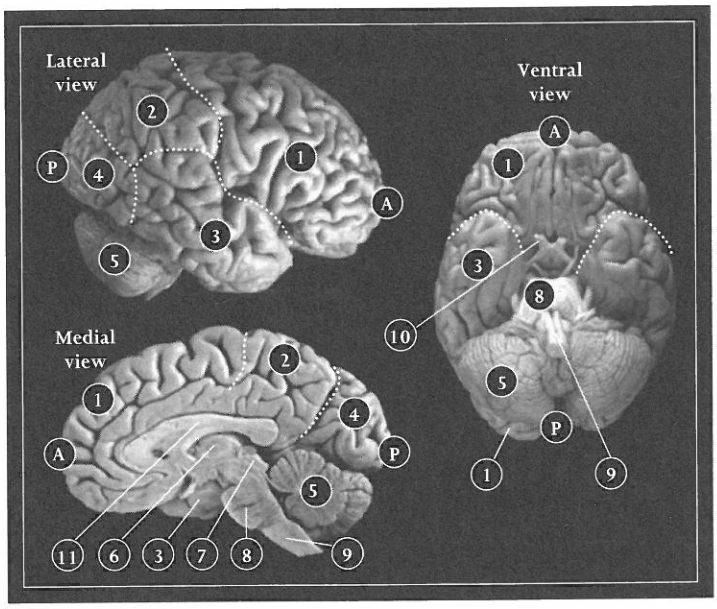
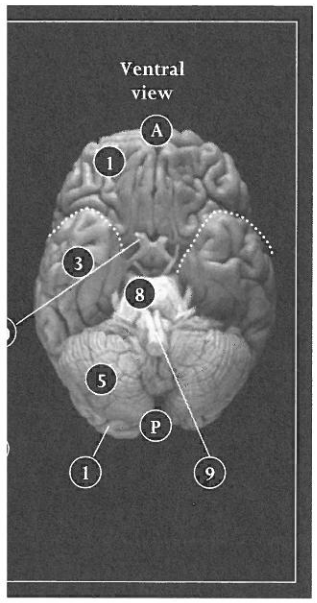


Figure 3.1
The human brain showing the lateral, medial, and ventral surfaces.
Key: A anterior; P posterior;
1 Frontal lobe; 2 Parietal lobe;
3 Temporal lobe; 4 Occipital lobe;
5 Cerebellum; 6 Thalamus;
7 Superior colliculus; 8 Pons;
9 Medulla; 10 Optic nerve;
11 Corpus callosum.

at least some of these patients show remarkably intact visual perception—despite having profound difficulties performing even simple visually guided movements. In short, the clinical picture they present is the mirror image of Dee Fletcher's.

The Hungarian neurologist Rudolph Bálint was the first to document a patient with this kind of problem, in 1909. The patient was a middle-aged man who suffered a massive stroke to both sides of the brain in a region called the parietal lobe (see Figure 3.1). Although the man complained of problems with his eyesight, he certainly was not agnosic in the way that Lissauer's and Freud's patients were. He could recognize objects and people, and could even read. He did tend to ignore objects on his left side and had some difficulty moving his eyes from one object to another. But his big problem was not a failure to recognize objects, but rather an inability to reach out and pick them up. Instead of reaching directly toward an object, he would grope in its general direction much like a blind man, often missing it by a few inches. Unlike a blind man, however, he could see the object perfectly well; he just couldn't guide his hand toward it. Bálint coined the term 'optic ataxia' (*optische Ataxie*) to refer to this problem in visually guided reaching.

Bálint's first thought was that this difficulty in reaching toward objects might be due to a general failure to locate where the



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objects were in his field of vision. But it turned out that the patient showed the problem only when he used his right hand. When he used his left hand to reach for the same object, his reaches were pretty accurate. This means that there could not have been a general problem in *seeing* where something was. In other words, this was not a visuospatial deficit. After further testing, Bálint discovered that the man's reaching difficulty was not a purely motor problem either—some kind of general difficulty in moving his right arm correctly. He deduced this from asking the patient to point to different parts of his own body using his right hand with his eyes closed: there was no problem.

So the optic ataxia that Bálint's patient suffered from was a truly 'visuomotor' disorder, in the sense that the patient could not use visual information about the location of the target to control a reaching movement with his (right) arm. Thus, although his deficit affected behavior directed at visual targets, it could not be explained away either as a general problem in either visuospatial processing or motor control. Unfortunately, this simple point has been largely overlooked by subsequent generations of neurologists, particularly in the English-speaking world. This may have been partly because Bálint's report remained untranslated for many years. Instead, most British and North American neurologists have followed the influential English physician and scientist, Gordon Holmes, and attributed these kinds of reaching difficulties to a general disorder in visuospatial perception—a deficit which would necessarily affect all spatially directed behavior, whatever form that behavior might take.

What has gone wrong in optic ataxia?

It was not in fact until the 1980s that the true nature of optic ataxia became apparent, in large part through the work of the French neurologists Marie-Thérèse Perenin and Alain Vighetto. They made detailed video recordings of patients with optic ataxia in a number of different visuomotor tests. Like Bálint, they observed that the patients made errors in reaching toward target objects placed in different spatial locations. Nevertheless, the patients were able to give accurate verbal descriptions of the relative location of the very objects to which they could not direct their hand. Like Bálint, Perenin and Vighetto demonstrated that

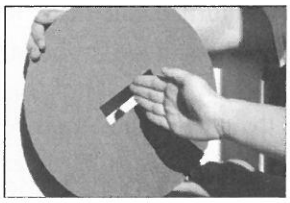

Figure 3.2

Marie-Thérèse Perenin and Alain Vighetto discovered that patients with 'optic ataxia' not only have problems reaching to point to something accurately, but also tend to direct their hand at the wrong angle when trying to pass it through a slot. The same patients, however, often have no problem describing the orientation of the slot in words.

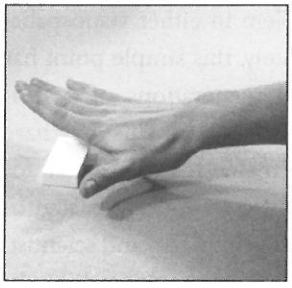

Figure 3.3

A typical example of a poor grip in a patient with optic ataxia. Her posture resembles someone groping in the dark for an object they know is there, although in this case the patient can see the object perfectly well.

the patients also had no difficulty in directing hand movements towards different parts of their body.

In another test, Perenin and Vighetto examined the ability of these patients to reach out and pass their hand through an open slot cut in a disk, which could be positioned at different orientations at random (see Figure 3.2). Not only did the patients tend to make spatial errors, in which their hand missed the slot altogether, but they also made orientation errors, in which the hand would approach the slot at the wrong angle. Yet several of these same patients could easily tell one orientation of the slot from another when asked to do so. So their failure to deal with the orientation of the slot when reaching was not due a perceptual difficulty in telling apart the different orientations. Again their problem was visuomotor in nature—in this case a problem in guiding their hand as they tried to pass it at the correct angle through the slot. (Of course when their hand made contact with the disk they could correct themselves using touch, and then pass their hand through the slot.)

As described in the previous chapter, of course, it was by borrowing Perenin and Vighetto's slot task that we were first able to provide a convincing demonstration of Dee Fletcher's preserved visuomotor abilities in the context of her profound visual form agnosia. In other words, the work with optic ataxic patients nicely complements Dee's pattern of performance on the slot task. So, does this neat contrast between the two kinds of patients also extend to the other tests we found Dee to be good at, such as grasping objects of different sizes?

Again the relevant evidence was gathered in France, in this case by Marc Jeannerod, who, as we noted in the previous chapter, pioneered the application of quantitative methods to the analysis of visually guided grasping in healthy volunteers. Importantly, Jeannerod went on to show that the well-regulated patterns of movement that typify the normal person's reaching and grasping behavior were severely disrupted in patients with optic ataxia. Instead of first opening the hand during the early part of the reach, and then gradually closing it as it moved toward the target object, the optic ataxic patient would keep the hand widely opened throughout the movement, much as a person would do if reaching blindfolded toward the object (see Figure 3.3). Just a few years ago, Jeannerod and his colleagues tested a patient,

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Anne Thiérry, who has a large area of damage to the parietal lobe on both sides of the brain, very much like Bálint's original case. They used similar matching and grasping tasks to those we had used earlier with Dee. Anne was found to show poor scaling of her grip while reaching for objects of different sizes, while remaining well able to demonstrate the sizes of the objects by use of her forefinger and thumb. This result again complements perfectly our findings with Dee.

The work we have summarized so far shows that optic ataxic patients not only have problems directing their actions to visual targets in space, but also have trouble with other visuomotor tasks in which object size and orientation are the critical factors. At the same time, when asked to *distinguish between* objects on the basis of their size, orientation or relative location, many of these patients do quite well. As we saw, this pattern of behavior is the converse of what we found with Dee. And it does not end there. We tested an optic ataxic patient called Ruth Vickers with the 'Blake' shapes described in the last chapter. We were interested to see if she would show the opposite pattern of results to that shown by Dee.

Ruth was a middle-aged housewife from rural Ontario who had recently suffered two strokes, one on each side of the brain, the second stroke occurring within a week of the first. Brain imaging showed that the damage was almost symmetrically located in the parietal lobe, again rather like Bálint's patient (see Figure 3.4).

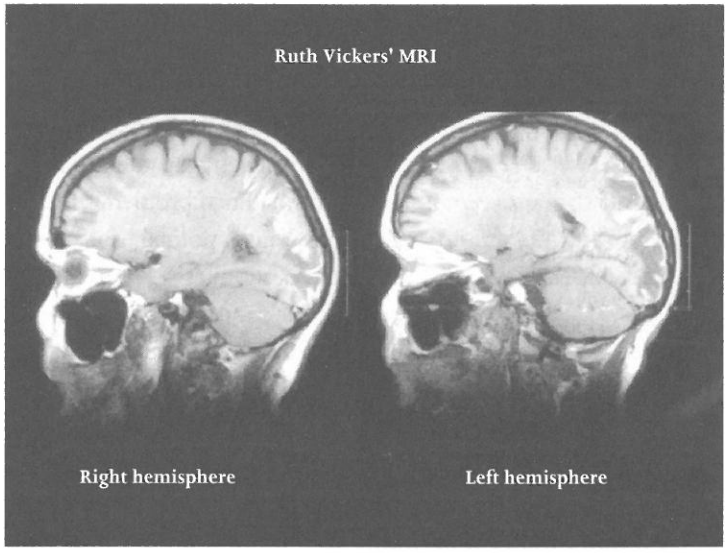

Figure 3.4
MRI scan of Ruth Vickers' brain showing a 'slice' from front to back through each half of the brain. The white areas at the back show the degenerated tissue on both sides of the brain resulting from Ruth's two strokes.

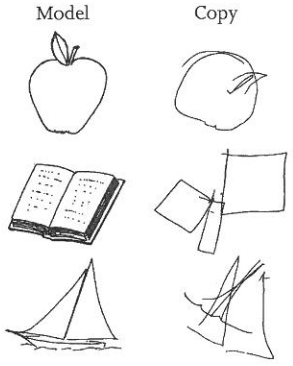

Figure 3.5

Unlike Dee, Ruth Vickers had no difficulty recognizing and naming the drawings shown on the left. Even when she was asked to copy them, she was able to capture many of the features of the drawings. Nonetheless it is obvious that she had difficulties coordinating her movements as she did her drawings.

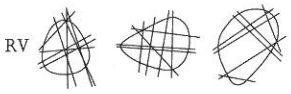

Figure 3.6

Examples of Ruth's attempts to pick up Blake shapes. Unlike Dee, she often grasped the shapes at inappropriate points, so that the shape would slip out of her fingers. From Goodale, M.A., Meenan, J.P., Bülthoff, H.H., Nicolle, D.A., Murphy, K.J., & Racicot, C.I. (1994). Separate neural pathways for the visual analysis of object shape in perception and prehension. *Current Biology*, 4(7), 604–610 (Figure 5).

Her clinical picture initially looked very much like that described by Bálint. Although Ruth's symptoms had cleared to some degree by the time we saw her, it was obvious that she still had severe optic ataxia. She could not reach with any degree of accuracy to objects that she could see but was not looking at directly. She could, however, reach reasonably accurately to objects directly in her line of sight.

Nevertheless, the reaches Ruth made to pick up objects that she was looking at, although spatially accurate, were far from normal. Like Anne Thiérry, she would open her hand wide as she reached out, no matter how big or small the objects were, showing none of the grip scaling typically seen in normal people. Yet despite this, when asked to show us how big she thought the object was using her finger and thumb, she performed quite creditably, again just like Anne. And she could describe most of the objects and pictures we showed her without any difficulty. In fact, although her strokes had left her unable to control a pencil or pen very well, she could draw quite recognizable copies of pictures she was shown (see Figure 3.5). In other words, Ruth's visual experience of the world seemed pretty intact, and she could readily convey to us what she saw—in complete contrast to Dee Fletcher.

Because Ruth could distinguish between many different shapes and patterns, we did not expect her to have much difficulty with the smooth pebble-like shapes we had tested Dee with earlier. We were right—when she was presented with a pair of these Blake shapes she could generally tell us whether or not the two shapes were the same. Although she sometimes made mistakes, particularly when two identical shapes were presented in different orientations, her performance was much better than Dee's. When it came to picking up the shapes, however, the opposite was the case. Ruth had real problems. Instead of gripping the Blake shapes at stable 'grasp points', she positioned her finger and thumb almost at random (see Figure 3.6). This inevitably meant that after her fingers contacted the pebble she had to correct her grip by means of touch—if she did not, the pebble would often slip from her grasp. In other words, although some part of her brain could clearly analyze the shape of these objects, her hand was unable to use that information.

All of these studies help us to define what it is that has gone wrong in optic ataxia. The patients tested by Perenin, Vighetto and

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Jeannerod, as well as by us, were all typical cases of this condition. Their brain damage was in the same region of the parietal lobe as that damaged in Bálint's patient, and they showed a similar inaccuracy when reaching out to targets in different parts of visual space. The fact that Perenin and Vighetto's patients could describe the location of the objects to which they could not reach supports Bálint's belief that optic ataxia was a visuomotor deficit and not, as Gordon Holmes maintained, part of larger overarching deficit in spatial perception.

But there is another important reason to doubt Holmes's account. As we have seen, many patients with optic ataxia demonstrate deficits that cannot be usefully construed as 'spatial', in the sense of seeing *where* an object is. For example, they do not rotate their wrist or open their grasp appropriately when picking up objects. Ruth Vickers, the patient we studied in Canada, could not direct her grasp to the appropriate points on the edges of the object that she was trying to pick up. In short, the range of visual attributes that are no longer accessible to the mechanisms controlling skilled motor output is much broader than originally thought, even by Bálint. So it is not useful to think about optic ataxia in terms of a deficit in spatial coding, some kind of a problem in seeing the spatial location of objects in the world. It now makes much more sense to think of it as a *visuomotor* disorder instead. To borrow some terminology from robotic engineering, optic ataxia can be seen as a disruption of the control systems connecting the sensors (dealing with the input) and the actuators (providing the output). These control systems would take all the relevant optical information from the sensors and re-code it for the programming and control of the goal-directed movements of the robot. In the human brain, analogous systems have to transform visual information about the size, shape, orientation, motion, and spatial location of the goal object, into a code for programming and controlling the person's skilled motor acts.

Summary

We have seen in this chapter that, in many different respects, optic ataxic patients present a quite opposite pattern of visual disabilities (and spared visual abilities) to what we saw in Dee Fletcher. This has important theoretical implications for our interpretation

of what is going on in Dee (and indeed in the optic ataxic patients). Going back to the concern with which we began this chapter, it cannot simply be the case that brain damage degrades the quality of Dee's visual experience in an undifferentiated way, so that some tasks can still be done while other 'more difficult' ones cannot. If this were true, then Ruth and Anne should show the same pattern of deficits and spared abilities as Dee. But of course they show the opposite. There is no way that a unitary general-purpose visual system can explain this.

Conversely, the fact that Dee Fletcher shows intact visuomotor control in the face of a profound perceptual loss also undercuts a common account of what causes optic ataxia. Some scientists have argued that optic ataxia is simply a 'disconnection' between visual perception and action, in which the perceptual information just cannot get through to the motor system. According to this intuitively reasonable view, there is only one kind of visual processing, which not only provides our conscious perception, but also the visual guidance for all our actions. But if this were the case, then how could Dee, whose visual perception of object form has been lost, carry out actions based on object form? If she does not have the perception, she should not be able to perform the visually guided actions. In short, Dee's spared abilities disprove this disconnection account of optic ataxia.

The existence of opposite patterns of lost and spared abilities in two kinds of patients is known in the trade as a 'double dissociation'. What a double dissociation shows is that when brain damage impairs one task (a recognition test, for example) but not another (such as a test of visuomotor skill), that difference cannot simply be put down to the second task being easier than the first. The other half of the double dissociation (in this case a visuomotor impairment coexisting with intact visual perception) rules that out.

What a double dissociation can also suggest—but cannot prove—is that different, quasi-independent brain systems (or brain 'modules' as they are sometimes called) are handling each of the two abilities that are dissociated. Establishing such modularity requires independent evidence from other kinds of research such as brain anatomy. In the next chapter we will discuss some of the kinds of evidence that support the idea of modularity in the organization of the visual system.

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4

The origins of vision: from modules to models

For most of us, sight is our pre-eminent sense. We do not just *respond* to visual stimuli: we *see* them as integral components of a visual world that has depth, substance and most important of all, a continuing existence separate from ourselves. It is through seeing that we gain most of our knowledge about external reality, and the possession of that knowledge, in turn, powerfully affects the way we see other things. In fact visual knowledge determines much of the basic content of our consciousness. Visual knowledge allows us to plan future actions, to picture the consequences of those actions, and to relive (sometimes with pleasure, sometimes with regret) what we have seen and done in the past. Vision affects the way we feel, as well as the way we think. Visual experiences can evoke powerful emotions, both positive and negative—as can the visual memories of what we have experienced before. Given the importance of vision in our mental life, it is not surprising that our language is full of visual metaphors. We can 'see the point', if we are not 'blind to the facts'; and occasionally show 'foresight' (though perhaps more often 'hindsight') by 'seeing the consequences' of our actions in our 'mind's eye'.

It is tempting to think that the delivery of such vivid experiences and the knowledge they impart is the entire raison d'être for vision. But the visual brain did not begin—in evolutionary terms—as a system designed to deliver conscious visual experience. That aspect of vision, while clearly extremely important, is a relative newcomer on the evolutionary landscape. So how did vision come on the scene originally?

To answer this question, we have to turn to evolutionary biology and ask: 'What is vision good for?' The answer from a biological

point of view is quite straightforward. Vision evolved only because it somehow improved an animal's fitness—in other words, improved its ability to survive and reproduce. Natural selection, the differential survival of individuals in a population, ultimately depends on what animals *do* with the vision they have, not on what they *experience*. It must have been the case therefore that vision began, in the mists of evolutionary time, as a way of guiding an organism's behavior. It was the practical effectiveness of our ancestors' behavior that shaped the ways our eyes and brains evolved. There was never any selection pressure for internal 'picture shows'—only for what vision could do in the service of external action. This is not to say that visual thinking, visual knowledge, and even visual experience did not arise through natural selection. But the only way this could have happened is through the benefits these mental processes have for behavior. Before returning to the intricacies of human vision, let us consider for a moment what kind of a role vision plays in the life of simpler organisms, which presumably do not have any mental life at all.

The origins of vision

A single-cell organism like the *Euglena*, which uses light as a source of energy, changes its pattern of swimming according to the different levels of illumination it encounters in its watery world. Such behavior keeps *Euglena* in regions of the environment where an important resource, sunlight, is available. But although this behavior is controlled by light, no one would seriously argue that the *Euglena* 'sees' the light or that it has some sort of internal model of the outside world. The simplest and most obvious way to understand this behavior is that it works as a simple reflex, translating light levels into changes in the rate and direction of swimming. Of course, a mechanism of this sort, although activated by light, is far less complicated than the visual systems of multicellular organisms. But even in complex organisms like vertebrates, many aspects of vision can be understood entirely as systems for controlling movement, without reference to perceptual experience or to any general-purpose representation of the outside world.

Vertebrates have a broad range of different visually guided behaviors. What is surprising is that these different patterns of activity are governed by quite independent visual control systems. The neurobiologist David Ingle, for example, showed during the

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1970s that when frogs catch prey they use a quite separate visuomotor control module from the one that guides them around visual obstacles blocking their path. These modules run on parallel tracks from the eye right through the brain to the motor output systems that execute the behavior. Ingle demonstrated the existence of these modules by taking advantage of the fact that nerves in the frog's brain, unlike those in the mammalian brain, can regenerate new connections when damaged. In his experiments, he was able to 'rewire' the visuomotor module for prey catching by first removing a structure called the optic tectum on one side. The optic nerves that brought information from the eye to the optic tectum on the damaged side of the brain were severed by this surgery. A few weeks later, however, the cut nerves re-grew, but finding their normal destination missing, crossed back over and connected with the remaining optic tectum on the other side of the brain. As a result, when these 'rewired' frogs were later tested with artificial prey objects, they turned and snapped their tongue to catch the prey—but *in the opposite direction* (see Figure 4.1). This

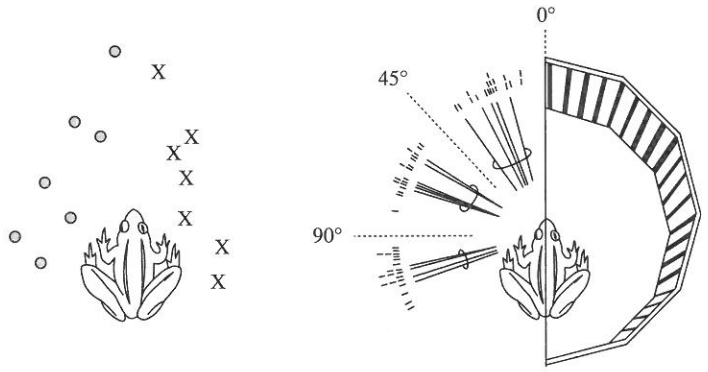


Figure 4.1

The dissociation between prey-catching behavior and visually-guided barrier avoidance in a 'rewired' frog. The drawing on the left shows that when a fake worm was presented to the eye opposite the missing optic tectum (at points shown by circles), the frog snapped at a mirror-image point on the other side (crosses). This is because the eye has become hooked up to the optic tectum on the wrong side of the brain. The optic tectum interprets the signals from this eye as if they were coming from the other eye (its usual source of visual input). The drawing on the right shows the directions in which the 'rewired' frog jumped in response to a gentle touch from behind in the presence of a barrier. The barrier was sometimes extended beyond the midline to positions 45° or 90° into the visual field of the rewired eye. A successful escape required the frog to turn and jump just enough to clear the edge of the barrier. The rewired frogs always cleared the barrier successfully, just like normal frogs. This is because only the eye's projections to the optic tectum were in fact rewired: the other projections, including those supporting barrier avoidance behavior, remained correctly hooked up. From Ingle, D.J. (1973). Two visual systems in the frog. *Science*, 181, 1053–1055 (Figures 1 & 2).

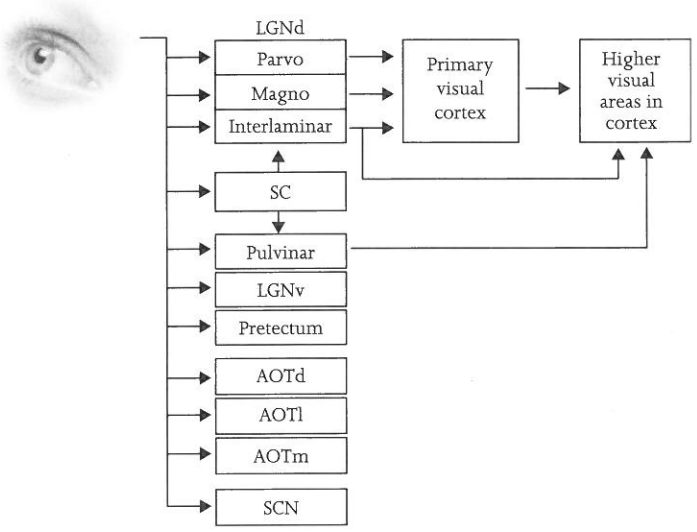
'mirror-imaged' behavior reflected the fact that the prey-catching system in these frogs was now wired up the wrong way around.

But this did not mean that their entire visual world was reversed. When Ingle tested the same frogs' ability to jump around a barrier blocking their route, their movements remained quite normal, even when the edge of the barrier was located in the same part of space where they made prey-catching errors (see Figure 4.1). It was as though the frogs saw the world correctly when skirting around a barrier, but saw the world mirror-imaged when snapping at prey. In fact, Ingle discovered that the optic nerves were still hooked up normally to a separate obstacle avoidance module in a part of the brain quite separate from the optic tectum. This part of the brain, which sits just in front of optic tectum, is called the pretectum. Ingle was subsequently able to selectively rewire the pretectum in another group of frogs. These animals jumped right into an obstacle placed in front of them instead of avoiding it, yet still continued to show normal prey catching.

What did these rewired frogs 'see'? There is no sensible answer to this. The question only makes sense if you believe that the brain has a single visual representation of the outside world that governs all of an animal's behavior. Ingle's experiments reveal that this cannot possibly be true. Once you accept that there are separate visuomotor modules in the brain of the frog, the puzzle disappears. We now know that there are at least five separate visuomotor modules in the brains of frogs and toads, each looking after a different kind of visually guided behavior and each having distinct input and output pathways. Obviously the outputs of these different modules have to be coordinated, but in no sense are they all guided by a single visual representation of the world residing somewhere in the frog's brain.

The same kind of visuomotor 'modularity' exists in mammals. Evidence for this can be seen even in the anatomy of the visual system. As Box 4.1 makes clear, the retina sends its optic nerve fibers to a number of different sites in the brain. Each of these brain structures in turn gives rise to a distinctive set of outgoing connections. The existence of these separate input–output lines in the mammalian brain suggests that they may each be responsible for controlling a different kind of behavior—in much the same way as they are in the frog. The mammalian brain is more

Box 4.1 Routes from the eye to the brain



Neurons in the retina send information to a number of distinct target areas in the brain. The two largest pathways from the eye to the brain in humans and other mammals are the ones projecting to the superior colliculus (SC) and the dorsal part of the lateral geniculate nucleus in the thalamus (LGNd). The pathway to the SC is a much more ancient system (in the evolutionary sense) and is the most prominent pathway in other vertebrates such as amphibians, reptiles, and birds. The SC (or optic tectum, as it is called in non-mammalian animals) is a layered structure forming the roof (Latin: *tectum*) of the midbrain. It is interconnected with a large number of other brain structures, including motor nuclei in the brainstem and spinal cord. It also sends inputs to a number of different sites in the cerebral cortex. The SC appears to play an essential role in the control of the rapid eye and head movements that animals make toward important or interesting objects in their visual world.

The pathway to the LGNd is the most prominent visual pathway in humans and other higher mammals. Neurons in the primate LGNd project in turn to the cerebral cortex, with almost all of the fibers ending up in the primary visual area, or striate cortex (often nowadays termed area V1) in the occipital lobe. This set of projections and its cortical elaborations probably constitute the best-studied neural system in the whole of neuroscience. Scientists' fascination with the so-called 'geniculo– striate' pathway is related to the fact that our subjective experience of the world depends on the integrity of this projection system (see the section on 'Blindsight' in Chapter 5).

Although the projections to the SC and LGNd are the most prominent visual pathways in the human brain, there are a number of other retinal pathways that are not nearly so well

studied as the first two. One of the earliest pathways to leave the optic nerve consists of a small bundle of fibers that project to the so-called suprachiasmatic nucleus (SCN). The visual inputs to the SCN are important for synchronizing our biorhythms with the day–night cycle.

There are also projections to the ventral portion of the lateral geniculate nucleus (LGNv), the pulvinar nucleus and various pretectal nuclei, and a set of three nuclei in the brainstem known collectively as the nuclei of the accessory optic tract (AOT). The different functions of these various projections are not yet well understood—although they appear to play a critical role in the mediation of a number of 'automatic' reactions to visual stimuli. The AOT have been implicated in the visual control of posture and certain aspects of locomotion, and have been shown to be sensitive to the optic flow on the retina that is created as we move through the world. The AOT also plays an important role in controlling the alternating fast and slow eye movements that we make when looking at a large visual stimulus, such as a train, passing before our eyes. Retinal projections to one area in the pretectum are thought to be part of the circuitry controlling the pupillary light reflex—the constriction of the pupil as we move into a brightly lit environment such as that found on the beach or the ski slopes. There is also some evidence from studies in amphibians and lower mammals that certain pretectal nuclei play a role in visually guided obstacle avoidance during locomotion. However almost nothing is known about the functions of the other pretectal nuclei, the ventral part of the lateral geniculate nucleus, or the pulvinar.

complex than that of the frog, but the same principles of modularity still seem to apply. In rats and gerbils, for example, orientation movements of the head and eyes toward morsels of food are served by brain circuits that are quite separate from those dealing with obstacles that need to be avoided while the animal is running around. In fact, these brain circuits in the mammal are directly homologous to the circuits we have already mentioned in frogs and toads, reflecting a common ancestry. For example, the circuit controlling orientation movements of the head and eyes in rats and gerbils involves the optic tectum (or superior colliculus as it is called in mammals), the same structure in the frog that controls turning and snapping the tongue at flies.

The fact that each part of the animal's behavioral repertoire has its own separate visual control system refutes the common assumption that all behavior is controlled by a single general-purpose representation of the visual world. Instead, it seems, vision evolved, not as a single system that allowed organisms to 'see' the world, but as an expanding collection of relatively independent visuomotor modules.

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Vision for perception

Of course, in complex animals such as humans and other primates, such as monkeys, vision has evolved beyond a set of discrete visuomotor modules. Much of our own behavior is certainly not rigidly bound by our sensory input. Even frogs can learn to some degree from their previous visual encounters with the world—but humans and other higher primates can use their previous visual experience and knowledge of the visual world in much more flexible ways so as to guide what they do in the future. We can internally rehearse different courses of action, for example, often using visual imagery in doing so, before deciding what to do.

In other words, vision can serve action not just in the here and now, but also 'off-line'—at other times and in other places. To do this, the visual brain creates a rich and detailed representation of the visual scene that the animal is looking at. We do not know what animals experience, but in humans at least, these perceptual representations are normally conscious. We experience them, and thereby we can communicate them to others. The visual mechanisms that generate these representations are quite different from the simple visuomotor modules of amphibians described earlier, and appear to have arisen more recently in evolutionary time. Rather than being linked directly to specific motor outputs, these new mechanisms create a perceptual representation that can be used for many different purposes. Moreover, as we mentioned in Chapter 1, our perception of the world is not slavishly driven by the pattern of light on the eye but is also shaped by our memories, emotions, and expectations. Visuomotor mechanisms may be driven largely bottom-up but perception has an important top-down component as well. The memories that affect our perception in this top-down way are themselves built up from previous perceptions. As a result of all this two-way traffic, perception and memory literally blend into one another. After all, we have visual experiences in our dreams, and these must be generated entirely by top-down processes derived from memory.

These general-purpose representations confer a big advantage in that they allow us to choose a goal, plan ahead, and decide upon a course of action. But on the other hand they do not have any *direct* contact with the motor system. The on-line visual

control of our actions still remains the responsibility of dedicated visuomotor modules that are similar in principle to those found in frogs and toads.

It is important to bear in mind that when people talk about what they 'see', they are talking only about the products of their perceptual system. Yet until recently researchers on vision have seen no need to go further than perceptual reports when gathering their data. In fact, a very important tradition in visual research, called psychophysics, depends entirely on what people report about what they can and cannot see. It has always been assumed that this is all there is to vision. Admittedly, psychophysics, which was founded by the nineteenth-century German physicist turned philosopher, Gustav Fechner, has told us a great deal about the capacities and limits of the perceptual system. But it has told us nothing about how vision controls the skilled movements that we make. The reason that psychophysics has failed in this regard is because the visuomotor machinery governing our actions is simply not accessible to conscious report. We may have a conscious visual experience of a coffee cup in front of us, but this experience will tell us little about the particular visual information that enables us to pick up the cup.

Vision for action

Alongside the evolution of perceptual systems in the brains of higher mammals such as humans, the visuomotor systems in turn have become progressively more complex. The main reason for this is that the movements we make have themselves become more complex. In our primate ancestors, one of the great landmarks in evolution was the emergence of the prehensile hand—a device that is capable of grasping objects and manipulating them with great dexterity. But just as the development of any sophisticated piece of machinery, such as an industrial robot, needs an equally sophisticated computer to control it, the evolution of the primate hand would have been useless without the coevolution of an equally intricate control system. The control of eye movements too has become more sophisticated and has become closely linked with the control of our hand movements. All of these changes, in other words, were accompanied by the evolution of new brain circuitry. Many of these new control systems in the brain have strong links

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to and from the basic modules in those older parts of the brain that were already present in simpler vertebrates like frogs and toads.

A good example of the way that these connections operate can be seen in the control of rapid (saccadic) eye movements in primates, such as monkeys and humans. We have seen already that head and eye movements in rodents are controlled by the same basic structures (the optic tectum, or superior colliculus) that control prey-catching in frogs. These same structures retain a central role in the machinery that programs head and eye movements in primates. But now these ancient visuomotor circuits have become subject to regulation and refinement by newer brain structures, where more intricate computations can be brought into play.

At first sight this may seem a puzzle—why didn't nature devise totally new systems from the ground up? In his book *Evolving Brains*, the American neurobiologist John Allman tells the story of how, on a visit to a power generation plant during the 1970s, he was struck by the side by side coexistence of several control systems for the generators dating from different periods in the life of the plant. There were pneumatic controls and a system of controls based on vacuum tube technology, along with several generations of computer-based control systems. All of these systems were being used to control the processes of electrical generation at the plant. When he asked the reason for this strange mix, he was told that the demand for power had always been too great for the plant ever to be shut down. As Allman points out:

> The brain has evolved in the same manner as the control systems in this power plant. The brain, like the power plant, can never be shut down and fundamentally reconfigured, even between generations. All the old control systems must remain in place, and new ones with additional capacities are added and integrated in such a way as to enhance survival.

It seems, however, that while these expanded visuomotor systems in higher mammals govern much more complex behaviors, they remain essentially automatic and are no more accessible to consciousness than those in the frog (or the *Euglena* for that matter). They might carry out more sophisticated and subtle computations on the visual information they receive, but they can do this perfectly well without a visual representation of the world. In fact, these visuomotor networks no more need conscious representations of the world than does an industrial robot. The primary

role of perceptual representations is not in the *execution* of actions, but rather in helping the person or animal to arrive at a decision to act in a particular way.

As we shall now see in the last section of this chapter, there has been a massive expansion in primates of the areas devoted to visual processing in the most prominent part of the mammalian brain—the cerebral cortex. We can understand this development by seeing it as reflecting the two closely related developments that we have outlined above. One development is the emergence of *perceptual systems* for identifying objects in the visual world and attaching meaning and significance to them. And the other is the emergence of more complex *visuomotor control systems* that permit the execution of skilled actions directed at those objects.

The sites of sight: Two visual streams in the primate cortex

In 1982 a seminal article appeared in the literature that has been cited more frequently than any other paper in the field of visual neuroscience, before or since. It was called 'Two cortical visual systems' and was written by two eminent American neuroscientists, Leslie Ungerleider and Mort Mishkin. They summarized converging experimental evidence mostly derived from monkeys, whose visual brains and visual abilities are closely similar to ours. Signals from the eyes first arrive at the cerebral cortex (the outer shell of gray matter that forms the evolutionary pinnacle of the brain) in a small area at the back called the primary visual area (V1). Ungerleider and Mishkin argued convincingly that the signals were then routed forwards along two quite separate pathways within the cortex (see Figure 4.2). One of these routes, which they called the dorsal visual pathway, ended up in part of the brain at the top of the cerebral hemispheres, the posterior parietal region. The other (the so-called ventral visual pathway) ended up at the bottom and sides of the hemispheres, in the inferior temporal region. These two pathways are now often called the dorsal and ventral *streams* of visual processing.

Many more visual areas have been discovered in the last twenty years and as a result there is a far more complicated pattern of interconnections than anyone thought possible back in 1982 (see Plate 2, bottom). Nevertheless, the basic wiring plan

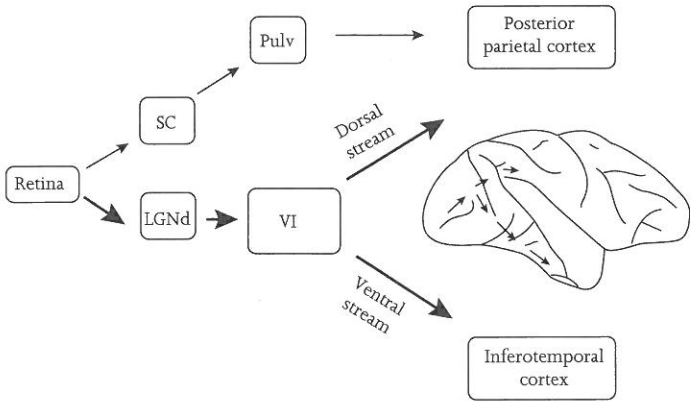

Figure 4.2

A schematic diagram of Ungerleider and Mishkin's original (1982) model of the two streams of visual processing in primate cerebral cortex. The brain illustrated is that of an Old World monkey. The ventral stream receives most of its visual input from the primary visual cortex (V1), which in turn receives its input from the lateral geniculate nucleus (LGNd) of the thalamus. The dorsal stream also receives input from V1, but in addition gets a substantial input from the superior colliculus (SC) via the pulvinar (Pulv), another nucleus in the thalamus. From Milner, A.D. & Goodale, M.A. (1995). Visual Brain in Action, Oxford University Press (Figure 3.1).

first identified by Ungerleider and Mishkin still stands: a dorsal stream going to the posterior parietal cortex and a ventral stream going to the inferior temporal cortex. What is remarkable is that the division of labor between these two information highways in the monkey's brain appears to map rather nicely onto the distinction we have been making between 'vision for action' and 'vision for perception' in humans.

The evidence for this mapping comes from two complementary kinds of research. First, there is evidence from lesion experiments, in which the dorsal and ventral streams in monkeys have been separately damaged to see what effects this damage might have on different kinds of visual behavior. Second, there is evidence from single-cell recording, in which the kinds of visual information that are encoded in individual nerve cells (neurons) can be monitored.

Doing without one stream

Studying how brain damage affects behavior in animals has had a long history. Even in mid-Victorian times, experimentally minded neurologists had begun to make selective lesions of brain tissue in animals, in the hope of gaining some understanding of the many brain-damaged people entering their clinics. The Scottish

neurologist David Ferrier was a pioneer in this field. During the 1860s, he removed most of what we now call the dorsal stream in a monkey, and discovered that it would mis-reach and fumble for food items set out in front of it. In a similar vein, work by Mitch Glickstein in England has shown that small lesions in the dorsal stream can make a monkey unable to pry food morsels out of narrow slots set at different orientations. The monkey is far from blind, but it cannot use vision to insert its finger and thumb at the right angle to get the food. It eventually does it by touch, but its initial efforts, under visual guidance, fail. Yet these same monkeys had no difficulty in telling apart different visual patterns, including lines of different orientation. These observations and a host of others have demonstrated that dorsal stream damage in the monkey results in very similar abilities and disabilities to those we saw in Ruth Vickers and Anne Thiérry. In other words, monkeys with dorsal-stream lesions show major problems in vision for action but evidently not in vision for perception.

In direct contrast, Heinrich Klüver and Paul Bucy, working at the University of Chicago in the 1930s, found that monkeys with lesions of the temporal lobes, including what we now know as the ventral stream, did not have any visuomotor problems at all, but did have difficulties in recognizing familiar objects, and in learning to distinguish between new ones. Klüver and Bucy referred to these problems as symptoms of 'visual agnosia', and indeed they do look very like the problems that Dee Fletcher has. Moreover, like Dee, these monkeys with ventral-stream lesions had no problem using their vision to pick up small objects. The influential neuroscientist, Karl Pribram, once noted that monkeys with ventral-stream lesions that had been trained for months to no avail to distinguish between simple visual patterns, would sit in their cages snatching flies out of the air with great dexterity. Mitch Glickstein recently confirmed that such monkeys do indeed retain excellent visuomotor skills. He found that monkeys with ventral-stream damage had no problem at all using their finger and thumb to retrieve food items embedded in narrow slots—quite unlike his monkeys with dorsal-stream lesions.

Eavesdropping on neurons in the brain

By the 1950s physiologists had devised methods for recording the electrical activity of individual neurons in the living

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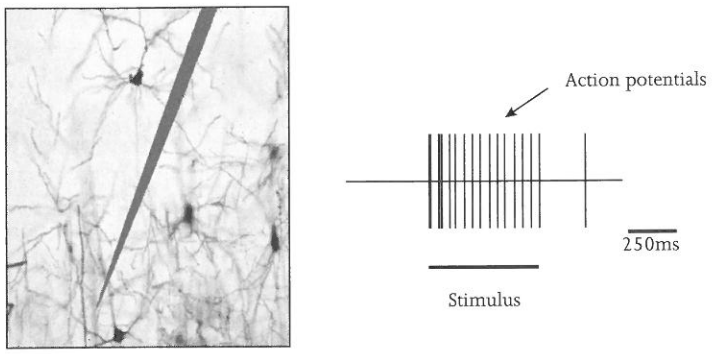


Figure 4.3

The photograph shows the tip of a microelectrode superimposed on a stained section of brain tissue, to show the relative size of the electrode in relation to typical neurons. (In reality the neurons would be much more densely packed than shown here, since only a minority of cells show up using this particular kind of histological stain.) Alongside the photograph is a diagram representing a train of action potentials recorded by such a microelectrode when the adjacent neuron is activated by a suitable visual stimulus.

brain (see Figure 4.3). The American Nobel Laureates David Hubel and Torsten Wiesel used these techniques to study the visual system, and found in the late 1950s that neurons in primary visual cortex (area V1) would 'fire' (i.e. give a small electrical response) every time a visual edge or line was shown to the eye, so long as it was shown at the right orientation and in the right location within the field of view. They discovered, in other words, that these neurons are 'encoding' the orientation and position of particular edges that make up a visual scene out there in the world. Different neurons prefer (or are 'tuned' to) different orientations of edges (see Figure 4.4). Other neurons are tuned for the colors of objects, and still others code the direction in which an object is moving. The distribution of these neurons within primary visual cortex is not haphazard. Neurons tuned to a particular orientation, for example, are clustered together in columns which run through the depth of the cortex. When Hubel and Wiesel explored visual areas beyond primary visual cortex, they found neurons that coded for more complicated visual features.

The 1960s and early 1970s heralded great advances in single-cell recording as investigators pushed well beyond the early visual areas, out into the dorsal and ventral streams. It soon became apparent that neurons in the two streams coded the visual world very differently.

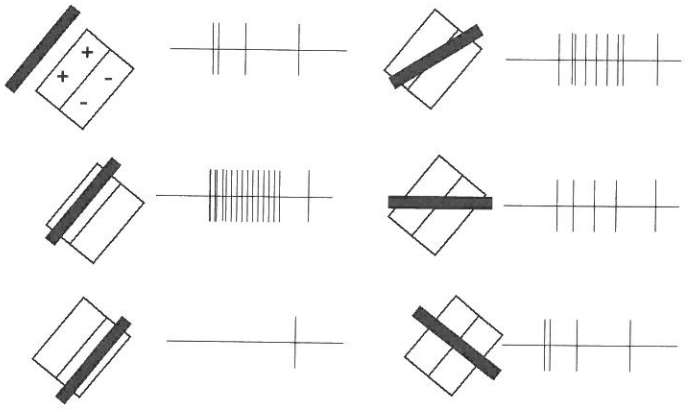
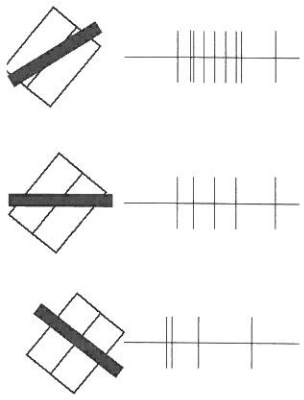


Figure 4.4

A diagram illustrating a neuron in area V1 that responds selectively to the orientation at which an edge or bar is shown to a monkey. The rectangle marks the location in space where the bar has to be presented for the neuron to respond (the neuron's receptive field). The plus signs indicate a region of the receptive field where presenting a small stimulus will result in an increase in the firing of the neuron. The negative signs indicate a region where presenting a small stimulus will result in a decrease in firing. This means that the orientation of the bar is critical in determining the firing rate of the neuron. Other neurons will have their receptive fields organized differently and thus will 'prefer' a different orientation of the bar.

The first person to probe the inferior temporal cortex, deep in the ventral stream, was Charles Gross at Princeton University. He found that neurons here were not satisfied with simple lines and edges, but needed to 'see' much more complex visual patterns before they would fire. In fact some neurons were so specific that they remained 'silent' until a hand or a face was shown to the monkey (see Figure 4.5). Keiji Tanaka, a neuroscientist working in Tokyo, has found clusters of neurons in inferior temporal cortex analogous to those previously found in area V1—only this time the neurons do not share a simple preference like 45°-oriented edges; they share a preference for a particular complex pattern of features.

Although the neurons in the ventral stream are quite fussy about the kind of object they respond to, a good number of them are not at all fussy about the particular viewpoint from which the object is seen or even where it is within the field of view. The neurons are also largely oblivious to the lighting conditions or the distance of the object from the eye. Neurons with these characteristics are exactly what is needed to identify a particular object across a wide range of viewing conditions—the kind of neurons that one would expect to see in a pathway specialized for perception.



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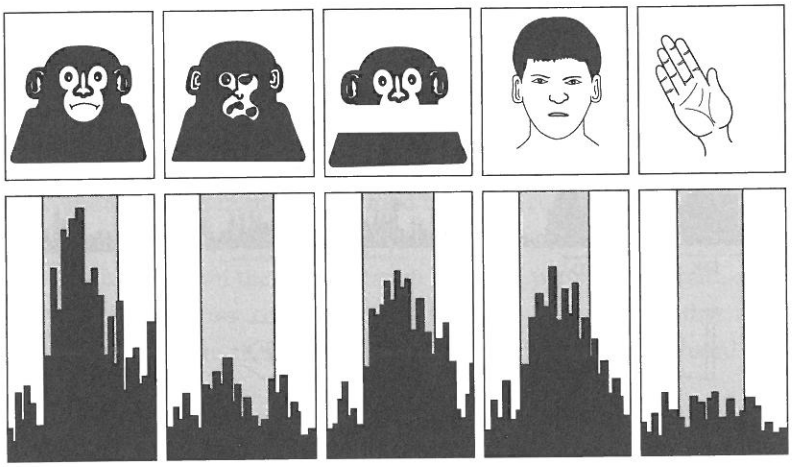


Figure 4.5

An example of a 'face cell' recorded within the ventral stream of a monkey's brain. This particular cell responded well to pictures of human or monkey faces seen in full front view, but less well when the picture was jumbled or when other objects such as a hand were shown to the monkey. The responses of the neuron are shown in the graphs below each image. The gray area on each graph shows the time over which the picture of the face or other object was shown to the monkey.

The next important development was in the mid-1970s, when scientists began to record from visual neurons in the dorsal stream. Working independently, Vernon Mountcastle at Johns Hopkins University and Juhani Hyvärinen in Helsinki, Finland, were the first to explore the properties of these neurons in detail. The surprising thing about neurons deep in the dorsal stream is the fact that although they are visually responsive, most of them fire strongly only when the monkey actually responds in some way to the visual target. For example, some neurons fire only when the monkey reaches out toward a target; others require that the monkey flick their eyes (i.e. make saccades) toward a stationary target; and still others fire to a moving target but only if the monkey follows it with its eyes. A particularly fascinating group of neurons, which were studied in detail during the 1990s by Hideo Sakata and his colleagues in Tokyo, respond when the monkey grasps or manipulates a target of a particular shape and orientation (see Figure 4.6).

These different subsets of neurons are clustered in somewhat separate regions of the posterior parietal cortex, with the 'grasp neurons', for example, located mostly toward the front end of the

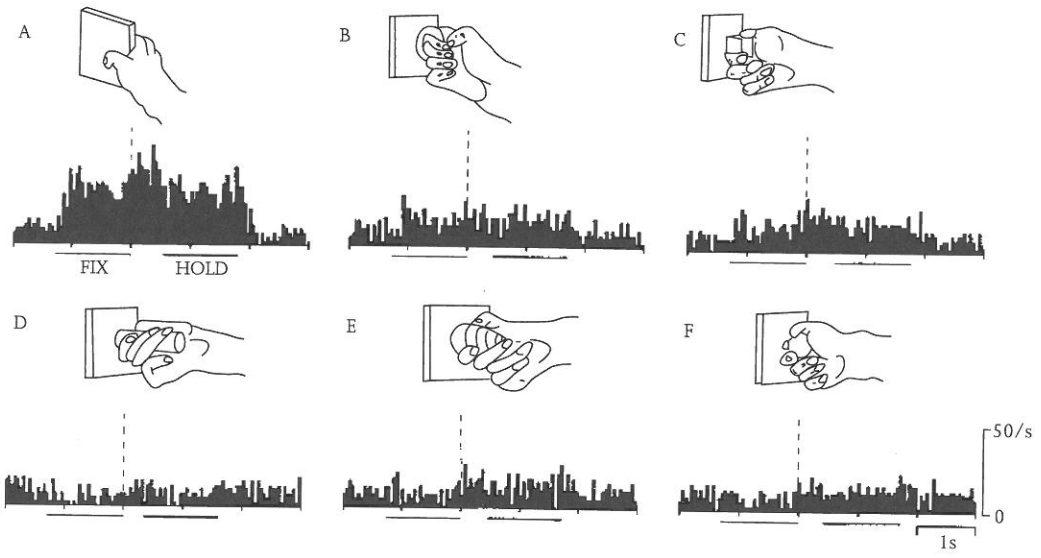


Figure 4.6

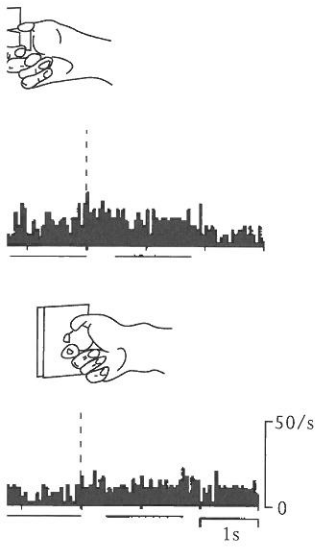
The activity of a neuron in area AIP when a monkey looks at and then grasps six different kinds of solid shapes. As the graphs below each shape show, the neuron responds best when the monkey grasps a vertically-oriented square plate. The neuron begins to fire when the monkey is first shown the object (marked 'fix' for fixation) and continues to fire after the monkey has grasped it (marked 'hold'). From Murata, A., Gallese, V., Luppino, G., Kaseda, M., & Sakata, H. (2000). Selectivity for the shape, size, and orientation of objects for grasping in neurons of monkey parietal AIP. *Journal of Neurophysiology*, 83, 2580–2601 (Figure 4).

region (area AIP). But despite their differences, what most of these neurons in the dorsal stream have in common is that they do not fire unless the monkey not only sees an object but in some way acts upon it as well. These are just the sorts of neurons you would expect to see in a 'vision for action' pathway.

Where do the two pathways lead?

The evidence reviewed above suggests that the ventral stream of visual processing in the monkey is the main conduit for transforming visual signals into perception whereas the dorsal stream plays the critical role in transforming visual signals into action. This division of labor is reflected in the outputs of the two visual pathways.

Consider the dorsal stream first. As we mentioned earlier, the behavioral repertoire of primates is much broader than that of the frog or even the gerbil. Fine hand and finger movements in particular imposed new demands on the visual system, and the



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(2000). Selectivity for the shape,
eurons of monkey parietal AIP. *Journal*

differences, what most of
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ts that the ventral stream of
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the visual system, and the

evolutionary development of the dorsal stream can be seen as a response to these demands. It is no accident that the visuomotor areas in the posterior parietal cortex sit right next to the cortical areas that get tactile information from the hand and arm. These visuomotor areas are also intimately linked with parts of the motor cortex in the frontal lobe that send commands to the lower parts of the brain and the spinal cord. In fact, there are also direct pathways from the dorsal stream to lower parts of the brain, such as the superior colliculus, and to other way-stations that send instructions to the eye muscles and to parts of the spinal cord that control the limbs.

The ventral stream has none of these direct connections with motor systems. Instead, as befitting its role in perception and recognition, it interfaces with structures in the temporal and frontal lobes that have been implicated in memory, emotion and social behavior. It is especially interesting, in the light of what we said earlier about the role of memory in perception, that these connections are very much two-way. Yet ultimately the perceptual system has to influence behavior. If it didn't, we wouldn't have one! The difference from the dorsal stream is that the ventral stream connections with the motor systems producing the behavior are by their very nature highly indirect. In fact, the connections can never be fully specified since the range of behavior that perception can influence is essentially infinite.

Summary

Vision serves behavior, but it does so in a variety of direct and indirect ways. What we can learn from studying animals other than ourselves is that there is not just one way of seeing, because we see for so many different purposes. Just as there is no sense in asking what David Ingle's frogs 'see', it is important to realize that in many contexts it will make no sense to ask ourselves the same question. We are aware of what *one* of our visual systems tells us about the world, because we are privy to its products—but there remains a whole realm of visual processing that we can never experience or reflect on. We are certainly aware of the actions that these visuomotor systems control, but we have no direct experience of the visual information they use.