CHAPTER 8
OCULOMOTOR CONTROL AND THE CONTROL OF ATTENTION

KEY THEMES

- Four regions that contribute importantly to oculomotor control are the superior colliculus, regions of occipital and parietal cortex (particularly V1 and LIP), regions of frontal cortex (the frontal eye field (FEF) and the supplementary eye field (SEF)), and the brainstem oculomotor nuclei that send commands to the extraocular muscles.
- Although superficial layers of the superior colliculus receive direct projections from the retina, the deep layers contain multimodal sensory neurons and movement neurons to which posterior cortical neurons project, and which, in turn, project to the brainstem oculomotor nuclei.
- FEF neurons, which project directly to the brainstem oculomotor nuclei, encode vector saccades, whereas SEF neurons encode goal-oriented saccades.
- Saccade planning and the endogenous control of attention both engage a dorsal frontoparietal network that includes FEF and IPS/LIP.
- The reflexive, exogenous capture of attention is associated with a right hemisphere-biased ventral cortical system, including the temporal-parietal junction and the inferior PFC, regions also associated with hemispatial neglect.
- Microstimulation of monkey FEF produces retinotopically specific behavior that is equivalent to the effects of cuing the endogenous control of attention, and also produces attention-like effects in V4 neurons whose receptive fields overlap with the movement field of the stimulation site.

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- Multivariate pattern analysis (MVPA) can dramatically increase the sensitivity of neuroimaging data analyses, and provide novel insights about information processing in the brain.
- MVPA has been used to demonstrate that the same systems that control eye movements are also used to control attention.

CONTENTS

ATTENTION AND ACTION

We have reached the final chapter of *Section II*, themed *Sensation, Perception, Attention, and Action*. Whereas we have seen in previous chapters how strongly perception and attention are linked (e.g., the attentional amplification of one sensory input channel over others, the dependence on attention of experience-dependent plasticity, unilateral neglect), this chapter will emphasize even more strongly than did the previous two how closely intertwined are the constructs of attention and action. At another level, this is the last chapter for which it will be necessary to begin with an overview of the anatomy of the system of interest. We haven't, of course, covered "everything," but upon completion of this chapter, the reader will have had an introduction to all of the major systems and principles of functional neuroanatomy, and will have the foundation on which to build a more detailed understanding of the intricacies of any particular component. Similarly, if the author has done his job, we'll see in every chapter following this one – i.e., in every chapter detailing some aspect of "high-level cognition" – that the principles underlying memory storage and retrieval, cognitive control, decision making, language, emotion, and so on, can be understood as variations on the principles introduced in this section on the more "elemental" functions of the brain.

WHYS AND HOWS OF EYE MOVEMENTS

Let's take a moment to recall one of the reasons why the ability to move the eyes is so important: it enables the visual system to disproportionately devote processing resources and, therefore, acuity, to just a small area of the visual field. If you need detailed information about an object located 15° to the left of your current point of fixation, you can obtain it within ~300 ms by moving your center of gaze to this location. The trade-off for the "cost" of those 300 ms is not having to drag around a visual cortex that's the size of the room that you're currently sitting in, or, alternatively, not having to survive with a visual system with uniformly horribly low resolution. Because there are some instances when a few tens of milliseconds can matter, however, we'll see that one feature of the oculomotor system is multiple pathways that control the eyes in different contexts.

Three categories of eye movements

Eye movements fall into three categories: smooth pursuit, vergence shifts, and saccades. We won't spend much time considering the first two, but will define them briefly here. If you hold out your thumb and fixate the nail, then slowly move your head from side to side, all the while maintaining fixation on your thumbnail, you are performing smooth-pursuit eye movements. (If your eyes hadn't been moving to compensate for the back-and-forth motion of your head, your thumbnail wouldn't have remained in your center of gaze.) The equivalent eye movements are generated if you now hold your head still and move your thumb back and forth. Thus, smooth-pursuit eye movements enable you to maintain fixation on an object while it, you, or both, are moving. During smooth pursuit, the two eyes move "together" (i.e., the movements of one eye are identical to those of the other). These are referred to as **conjugate eye movements**.

Vergence shifts occur when an object of interest is moving toward or away from you, and in order to fixate it with both eyes, the two must move in opposite directions: toward each other if the object is looming; away from each other if it is receding (*Tip 8.1*).

The third category, saccadic eye movements, is the focus of this chapter. The word *saccade* is French for a jerky movement, and was first associated with eye

Tip 8.1

Signals from stretch receptors on the extraocular muscles convey information about the positions of the eyes relative to each other (i.e., how parallel vs. converging). This information contributes to the perception of depth (i.e., seeing in 3-D), and of motion in depth (i.e., motion toward and away from you, as opposed to motion along a 2-D surface, such as when looking at a movie screen or a computer monitor).

movements by the French ophthalmologist Emile Javal (1839–1907), who held a mirror at an angle while reading and observed that his eyes made a series of brief fixations, punctuated by abrupt conjugate movements, rather than a smooth, continuous drift across the page as most (who had ever given it any thought) had previously assumed. Saccades are of primary interest here because of their central importance to many domains of cognition (reading being just one of them), and because they have been used extensively in neuroscience and psychology research as an indirect measure of cognitive operations. Because monkeys cannot be verbally instructed how to respond on experimental tasks, nor can they tell you what they are thinking, saccades are a way for the animal to indicate its choice in an experiment. (E.g., *Which is the most recent item that you saw?* or *Which of these stimuli is different from the others?*) And although it would be disingenuous to suggest that training a monkey to perform a cognitive task is ever "easy," it is the case that saccadic eye movements are natural behaviors for monkeys (and humans), and their use as a dependent measure can at least give the researcher a "head start" in the training process. For humans, the saccade itself can be of fundamental interest, if it is a fundamental part of the behavior of interest (e.g., reading, or visual search). Additionally, scientists have devised clever ways to use eye movements to assay the dynamics of cognitive operations as they are being carried out, as opposed to, or in addition to, retrospective reports that come at the end of each trial. (E.g., whether the eyes start to drift toward word *X* or word *Y*, both displayed on a screen, can provide insight into the mental operations engaged while a person is listening to a semantically ambiguous sentence.)

THE ORGANIZATION OF THE OCULOMOTOR SYSTEM

An overview of the circuitry

There are many factors that can prompt an eye movement. I can decide, while taking a pause as I type this sentence, to look up at a painting hanging on the wall of my living room. My eyes can move reflexively toward the source of a sound, or to a touch (the cat walks into the room, then jumps up onto the couch next to me). I can scan the shelves of a grocery store, looking for that particular brand of soy sauce. Much of the discussion here, however, will be framed in terms of visually guided saccades. That is, what are the steps that transpire between the detection on the retina of a visual stimulus, and the saccade that brings that stimulus into foveal vision. Along the way, we'll note instances where the principles may be similar or different for these other factors that can also trigger saccades.

Although the principal pathway for visual processing in most mammalian species is the retino-geniculo-striate pathway, it "wasn't always that way." In fish and amphibians, the primary site of visual processing is a midbrain structure known as the optic tectum (*Tip 8.2*). In the mammal, the homologue is the superior colliculus ("higher hill" in Latin; the inferior colliculus of the auditory system, *Figure 4.4*, is the "lower hill"), a structure that sits immediately caudal to the thalamus on the "roof" ("tectum") of the brainstem. There is a direct, monosynaptic pathway from the retina to the superior colliculus, which will receive more consideration further along in this chapter. The superior colliculus projects to the **brainstem oculomotor nuclei**, which house the local circuitry

Tip 8.2

The implication here is that modern fish/amphibians and mammals share a common ancestor for which, as continues to be the case for fish/amphibians, the optic tectum was the sole brain structure that interceded between retina and brainstem oculomotor nuclei. This does not imply, however, that the optic tectum of modern fish and amphibians remains unchanged, "frozen in time," from that of the common ancestor. Indeed, the optic tectum of many modern aquatic species is much more complicated than the mammalian superior colliculus – subsequent evolutionary changes in the visual systems of some species have been most pronounced in the optic tectum, and in those of others, in cerebral cortex.

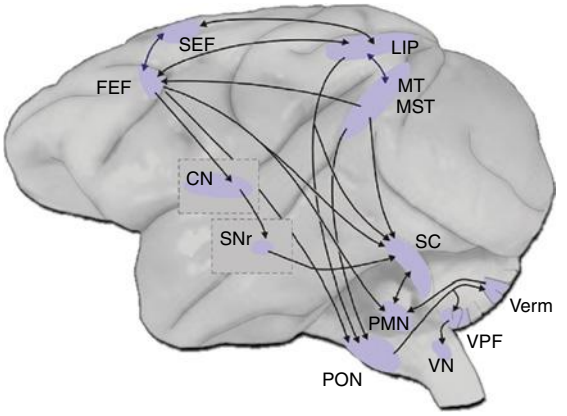

FIGURE 8.1 Important nodes and connections for oculomotor control. Note that among this diagram's simplifications are the absence of the V1 projection to SC, and the integration of LIP and FEF signals in the "oculomotor" cortico-basal ganglia-thalamic loop. Abbreviations: CN = caudate nucleus; FEF = frontal eye field; LIP = lateral intraparietal area; MT = middle temporal area; MST = medial superior temporal area; PMN = brain stem (pre)motor nuclei (PPRF, riMLF, cMRF); PON = precerebellar pontine nuclei; SC = superior colliculus (intermediate and deep layers); SEF = supplementary eye field; SNr = substantia nigra pars reticulata; Verm = oculomotor vermis (cerebellum, lobules VI and VII); VN = vestibular nuclei; VPF = ventral paraflocculus (cerebellum). Source: Krauzlis, 2005. Reproduced with permission of SAGE Publications.

and oculomotor neurons, analogous to the motor circuitry of the spinal cord (*Chapter 7*), that process descending motor commands and send axons that innervate the extraocular muscles. Within cortex, regions contributing to oculomotor control can be divided into posterior and anterior systems, the posterior including, most prominently, V1, LIP, and V4, and the anterior comprised of the frontal eye field (FEF) and the supplementary eye field (SEF, also referred to as the medial eye field (MEF) or dorsomedial frontal cortex (DMFC)). The primary distinction between the posterior and anterior systems is that the former regions project to the superior colliculus – superior colliculus lesions abolish the ability of electrical stimulation of posterior cortical areas to elicit saccades – whereas the anterior regions, although sending projections to the superior colliculus, also send projections directly to the brainstem oculomotor nuclei (*Figure 8.1*).

Tip 8.3

Keep in mind that most, if not all, of the cortical areas to be discussed here can't be considered "specialized" for oculomotor control; the label "oculomotor system" is simply an economical way to say "regions in the brain that can contribute to oculomotor control."

Let's now consider each of these major elements of the "oculomotor system" in turn (*Tip 8.3*).

The superior colliculus

In primates, the superior colliculi (one on each side of the midline) are each about the size of a half-globe of a pea, and roughly divided into three layers: superficial, intermediate, and deep. It is the deep layers that send efferents to the brainstem oculomotor nuclei. The superficial layers receive direct input from the retina, via a specialized class of retinal ganglion cells called *w cells*. The deep layers are of greater interest, because they receive the cortical efferents illustrated in *Figure 8.1*, as well as sensory information from the visual, auditory, and tactile modalities. There are two interleaved populations of cells: sensory neurons that have multimodal receptive fields and represent locations in retinotopic coordinates; and saccade neurons whose bursting activity brings the center of gaze into the receptive field of adjacent sensory neurons. Thus, the superior colliculus employs a vector code, in that activity in a certain area always produces a saccade of the same direction and magnitude (*Figure 8.2*; *Tip 8.4*).

At the rostral-most end of the superior colliculus, in the deep layers, are fixation neurons. In effect, in relation to the motor map illustrated in *Figure 8.2.B*, these command saccades of 0°. These neurons maintain a tonically elevated firing rate while one is fixating a stationary location. (Thus, at least from the reductionist perspective of activity in the superior colliculus, the term "passive fixation" that one frequently sees in the literature is a misnomer.)

At any moment in time, most of the motor units in the deep layers of the superior colliculus are under tonic inhibition from the substantia nigra pars reticulata, an arrangement analogous to the tonic inhibition of thalamus by the GPi that we detailed in *Chapter 7*.

FIGURE 8.2 The functional organization of the superior colliculus.

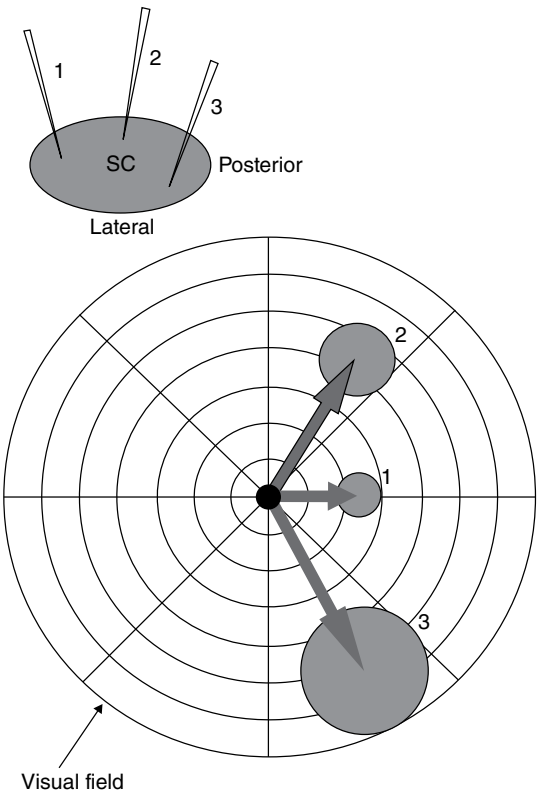

FIGURE 8.2.A Cartoon of the effects of recording from and stimulating at three electrode locations in the superior colliculus in the left hemisphere. The gray circles are (visual) sensory receptive fields, and the arrows indicate the magnitude and direction of the saccade generated when the neuron is stimulated. Source: Schiller, 1998. Reproduced with permission of Lawrence Erlbaum Associates.

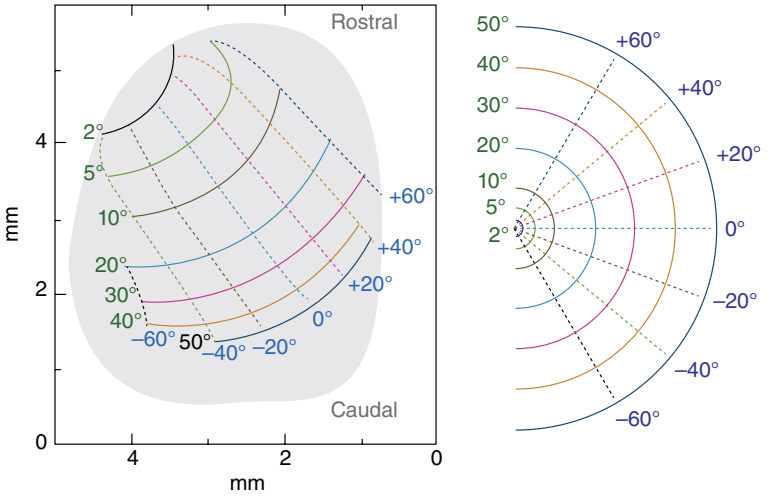

FIGURE 8.2.B Left panel: motor map in deep layers of the left superior colliculus; right panel, corresponding regions of the right visual field. Rostral areas represent small-amplitude saccades, caudal areas large amplitude saccades. Rostral and lateral to the 2° line (i.e., in the upper-left corner of the image) are fixation neurons. See Gandhi and Katnani, 2011. Source: Gandhi and Katnani, 2011. Reproduced with permission of Annual Reviews.

Tip 8.4

There is a potential for terminological confusion here, in that one might see an analogy to the cochlear nucleus, where "place code" refers to the fact that activity from one place along the cochlear nucleus always corresponds to a particular frequency; in the superior colliculus, activity at one place always produces saccades of the same metrics. In motor systems, however, the word "place" is used to refer to a location in the outside world. See *Figure 8.3*.

Tip 8.5

The property of "modulation" is traditionally ascribed to neurotransmitters ascending to cortex from subcortical nuclei (e.g., dopamine (see *Chapter 7*), norepinephrine, serotonin, and acetylcholine). However, Sherman and Guillery (2006) have proposed that many cortical glutamatergic neurons also play a modulatory, as opposed to a "driving" or an "information carrying," role (more detail at *Web Link 8.1*).

The posterior system

Not too much ink needs to be spilt here, because the oculomotor functions of LIP were covered in some detail in *Chapter 6*. Although neurons from layer 5 of V1 send projections to the superior colliculus, their contribution seems to be modulatory, rather than driving. This assessment comes from the fact that, whereas microstimulation of V1 can produce saccades, the currents required to do so are markedly higher than those required in other cortical areas, such as the FEF, or in the superior colliculus itself. However, cooling V1, a method that reversibly inactivates a region of cortex, has the effect of abolishing visual sensory responses in the deep layers (*Tip 8.5*). Also consistent with a modulatory function of visual cortex on the superior colliculus is the association of oscillatory state in occipital cortex with the generation of **express saccades**. (For a more detailed treatment of the distinction between "driving" vs. modulatory signals, see *Web Link 8.1*.)

Express saccades have a latency that can be as much as one half that of standard saccade latency – in one recent study in humans, 90–135 ms after target onset for express saccades, 145–240 for standard saccades – and are often seen in response to the abrupt appearance of an unexpected stimulus. They are also readily produced experimentally with a "gap" paradigm, in which a gap of a few hundred ms is inserted between the offset of the fixation point that began the trial and the onset of the target to which the subject must make a saccade. Although it would be tempting to postulate that express saccades are generated by a "short circuit" from retina to superior colliculus to brainstem oculomotor nuclei, the anatomy would seem to argue against this, because, as far as we know, retinocollicular projections terminate exclusively in superficial layers of the superior colliculus, and these do not project directly to the deep layers. An alternative account is that "advanced motor preparation of saccadic programs" (Paré and Munoz, 1996, p. 3666) enables the cortical system to generate saccades more quickly than is typical. Consistent with this idea is the fact that express saccades are more likely to be generated on trials with elevated oscillatory synchrony in the low alpha-band range, measured at occipital EEG electrodes.

Consistent with their role in processing visual motion, the contributions of areas MT and MST to oculomotor control are particularly important for guiding smooth pursuit movements.

The frontal eye field

As illustrated in *Figure 8.1*, the FEF in the monkey is located in the rostral bank of the arcuate sulcus, immediately anterior to premotor cortex. Among the first to study the FEF was David Ferrier, whose involvement in nineteenth-century research on the neural bases of vision made an appearance in *Chapter 1*. Ferrier (1875) found that electrical stimulation of this region produced "turning of the eyes and head to the opposite side" (p. 424), and that a unilateral lesion impaired the abilities of the animal to orient to stimuli presented contralateral to the lesion. Based on these and other observations, he stated most presciently in 1890 that "my hypothesis is that the

power of attention is intimately related to the volitional movements of the head and eyes" (Ferrier, 1890, p. 151). Consistent with this idea has been the subsequent description, in monkeys and humans, of unilateral neglect after damage to the FEF.

As indicated earlier, a distinguishing characteristic of the FEF is its low threshold for saccade generation with microstimulation. Although its organization isn't strictly retinotopic, it's generally the case that small, contralaterally directed saccades are produced by microstimulation in the ventrolateral limb of the anterior bank of the arcuate sulcus, larger amplitude saccades by microstimulation in the dorsomedial limb. This region encodes vector saccades, in that repeated stimulation of the same site produces a saccade of the same metrics, regardless of the starting point of the eye (*Figure 8.3*). A sustained pulse of stimulation produces a series of discrete saccades, all of the same metric. This is an interesting phenomenon that illustrates the recoding that happens in the brainstem oculomotor nuclei, because sustained microstimulation of a single location in one of these nuclei produces a very different effect – a saccade whose magnitude is linearly dependent on the duration of the stimulation train. (The same is true, of course, for the extraocular muscles: they will only contract so long as ACh is being released into the neuromuscular junction.)

The supplementary eye field

Located dorsal and somewhat caudal to the FEF (*Figure 8.1*), the SEF lies just rostral to the SMA (*Chapter 7*). Its functions are associated with more "cognitive" aspects of oculomotor control. For example, when two targets are presented, and the monkey will receive a reward for saccading to either one (i.e., its choice), SEF, FEF, and LIP all show activity related to the impending choice, but that of the SEF starts earliest and is most robust. SEF activity is also prominent during the **antisaccade task**, when the rule is to make a saccade 180° opposite to the location of the target. The coding principle is also different between SEF and FEF, as is illustrated in *Figure 8.3*. Whereas microstimulation of the FEF neuron produces a saccade with roughly the same metrics regardless of the starting point of the eyes, microstimulation of a SEF neuron always produces a saccade that lands in the same "termination zone." This is reminiscent of what

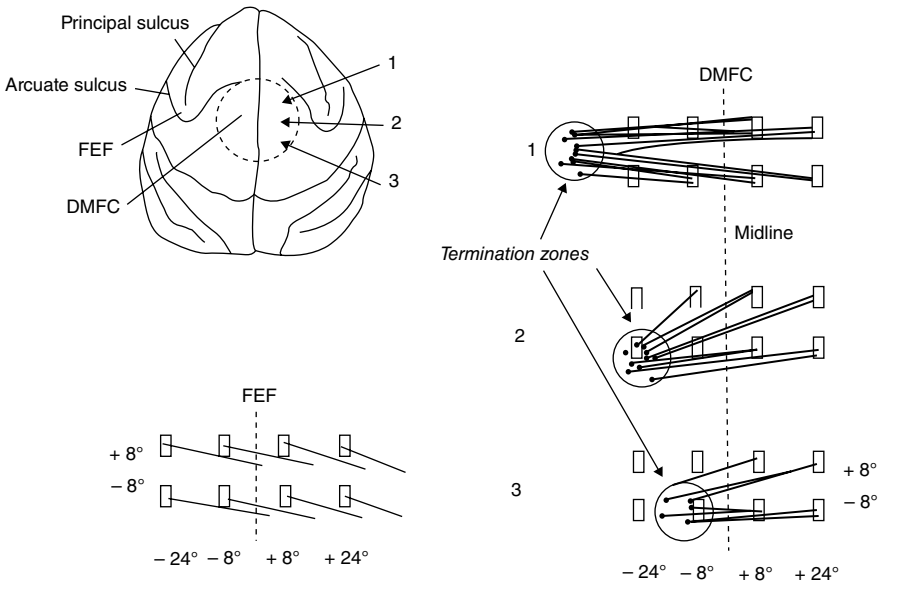

FIGURE 8.3 The effects of microstimulation of one neuron in FEF and of three neurons in SEF (referred to as DMFC by these authors), the location of each indicated with an arrow, from each of eight starting fixation points. Source: Schiller, 1998. Reproduced with permission of Lawrence Erlbaum Associates.

we saw in the spinal cord in *Chapter* 7, where stimulation of one location will always bring the forepaw to the same location in space, regardless of its starting location. This also highlights the terminological ambiguity referenced in *Tip 8.4*: a functional principle that has been ascribed to the SEF is a "place code," in that activity in one set of neurons always brings the center of gaze to the same place. (Though, recall from *Chapter* 7 that the similar phenomenon in rostal LIP was referred to as "goal-directed.")

THE CONTROL OF EYE MOVEMENTS, AND OF ATTENTION, IN HUMANS

Human oculomotor control

A classic task for assessing oculomotor control is the oculomotor delayed-response task, in which the subject begins by fixating a central location, the to-be-acquired location is cued by a flashed stimulus, but the subject must withhold the saccade to that location until the offset of the fixation point. This temporal separation allows for the dissociation of "sensory" activity related to presentation of the cue from motor activity prompted by fixation offset. (The interpretation of "delay-period activity" sometimes observed between cue offset and fixation offset is less straightforward, as we shall see further along in this chapter, and again in *Chapter 13*.) *Figure 8.4* illustrates this task, together with examples of activity measured from the monkey and the human. Note that in the human, there is robust delay-period activity in the intraparietal sulcus (IPS; a superior portion of which is generally thought to be homologous to LIP in the monkey), as well as at two distinct frontal foci (*Figure 8.4.C*). The human FEF has classically been associated with the more dorsal of the two, just rostral and ventrolateral to the point of intersection of the precentral sulcus and the superior frontal sulcus. However, the more ventral focus of elevated activity in frontal cortex is also frequently observed. Note that although the frontal regions illustrated in *Figure 8.4.C* show elevated activity during each epoch of the trial (*cue*, *delay*, *response*), these regions were identified statistically as having elevated activity during the delay period, regardless of whether or not they were differentially responsive during the *cue* and/or *response* epochs. Doing so requires an event-related procedure, some principles of which are described in *Methodology Box 8.1*, and in *Web Clip 3.2* and *Web Clip 8.1*.

The advent of event-related designs for fMRI seems to have spontaneously, independently occurred more-or-less at the same time in many laboratories in the late 1990s, including (from east to west) at the Functional Imaging Laboratory (FIL) of the University College London (UK), the Massachusetts General Hospital (Boston), the University of Pennsylvania (Philadelphia), the National Institutes of Health (Bethesda, MD), and Washington University in St. Louis (*Tip. 8.7*). It is at the latter of these where we'll next turn, to consider influential work on the control of attention.

Tip 8.6

The distinction between delayed response – in which the subject knows what the correct response is, but needs to withhold it until the "go" signal (e.g., the offset of the fixation point) – and delayed recognition – in which the response can't be known until the presentation of the second stimulus – makes the former a test of motor preparation and the latter a test of short-term memory. (This distinction will be a focus of *Chapter 13*.)

Tip 8.7

The author remembers these as heady times for the cognitive neuroimaging community, finishing up his graduate studies, as he was, near one of these centers, and moving on to a postdoctoral fellowship at another. Buy him a drink sometime and he'll tell you some stories.

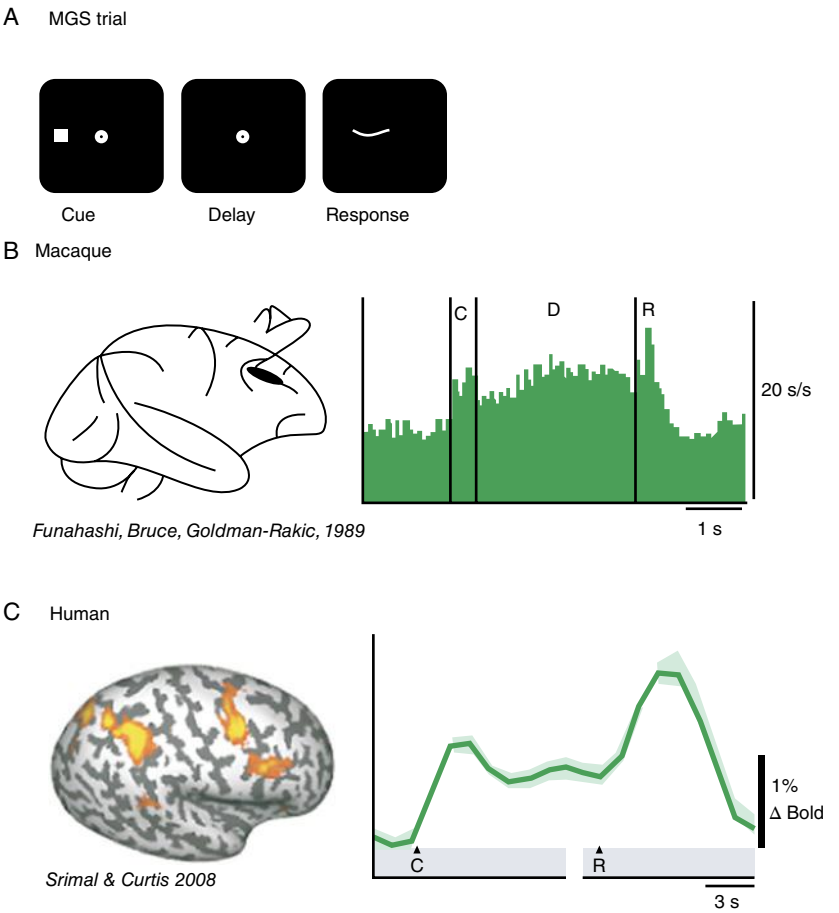

FIGURE 8.4 The delayed-saccade task (also referred to as "memory-guided saccade (MGS)" or "oculomotor delayed-response (ODR)"). Panel **A** illustrates a trial, as described in the text. Panel **B** illustrates the recording location in a monkey, in this instance at the caudal end of the principal sulcus, near the rostral boundary of the FEF, and activity recorded from a neuron at this location. Panel **C** illustrates regions with elevated delay-period activity in a human performing this task in an MRI scanner. The anatomical image has been "inflated," so that sulci (darker gray) are visible. The fMRI time series data are interrupted between cue (C) and response (R) because the researchers used variable-length delay periods in this study. Source: Ikkai and Curtis, 2011. Reproduced with permission of Elsevier.

Human attentional control

Recall from *Chapter 6* that we restricted ourselves at that time to a detailed consideration of the *site* of the effects of attention, pushing off consideration of the *source* of attentional control to a later chapter. Well, *source*, your time has come.

From experimental psychology, we have long known that there are two general modes by which attention can be directed: the exogenously triggered *capture* of attention vs. the endogenously controlled *allocation* of attention. An example of an exogenously triggered capture of attention would be that you are sitting quietly, reading this book, when suddenly someone loudly, unexpectedly, bursts into the room. Your reflexive reaction is to turn your head in the direction of the noise. An example of the endogenous control of attention is when you are sitting quietly, pretending to

METHODOLOGY BOX

8.1 Block designs vs. event-related designs in fMRI (Zarahn, Aguirre, and D'Esposito, 1997)

A challenge for both PET and fMRI studies is how to design tasks that allow for the isolation of a cognitive mechanism of interest. Recall from *Methodology Box 6.1* that the relatively low SNR of PET necessitates minutes-long periods of data acquisition. To isolate activity associated with speaking, therefore, the researchers subtracted images corresponding to blocks of time when subjects were viewing words from blocks from when they were reading the words aloud. Because the logic underlying this approach is that both blocks contain activity associated with the same cognitive operations (e.g., visually perceiving the letters, reading the word), except one (speaking the word aloud), it is called "cognitive subtraction."

"Block designs," in which several trials of the same type are blocked together, and the resultant neuroimaging signal is averaged across the entire block, are also common with fMRI studies, as illustrated in *Figure 6.19*. One of the most compelling reasons for employing a block design is that it features higher SNR than event-related designs, by virtue of the inherent "sluggishness" of the hemodynamic response function (HRF; *Figure 6.8*). (The technical reasons for this are nicely explicated in *Further Reading* by Aguirre and D'Esposito (1999).) A downside of block designs, however, is that assumptions underlying the logic of cognitive subtraction can sometimes fail. *Figure 8.5*, for example, illustrates this with data from a *delayed-recognition* task (similar in some respects to the delayed-response task from *Figure 8.4*, but different in that subjects can't know what the appropriate response is until the memory probe (labeled "discrimination/response") is presented at the end of the trial (*Tip 8.6*)). *Figure 8.5.B* illustrates two hypothetical response patterns, one in which the brain region responds to stimulus presentation, but not during the delay period, the second in which the brain region responds during all three.

Figure 8.5.C illustrates the actual response of two adjacent regions of right PFC during this task, from Zarahn, Aguirre, and D'Esposito (1999). In these panels, we are privy to the moment-to-moment evolution of activity in these regions, because they were analyzed in an event-related manner. But let's pretend for a moment that they were analyzed in a block design. Were there to have been, say, 10 consecutive D trials in a block, in both regions these would clearly have produced a significantly greater "area under the curve"

FIGURE 8.5 Event-related fMRI. Source: Zarahn, Aguirre, and D'Esposito, 1999. Reproduced with permission of Elsevier.

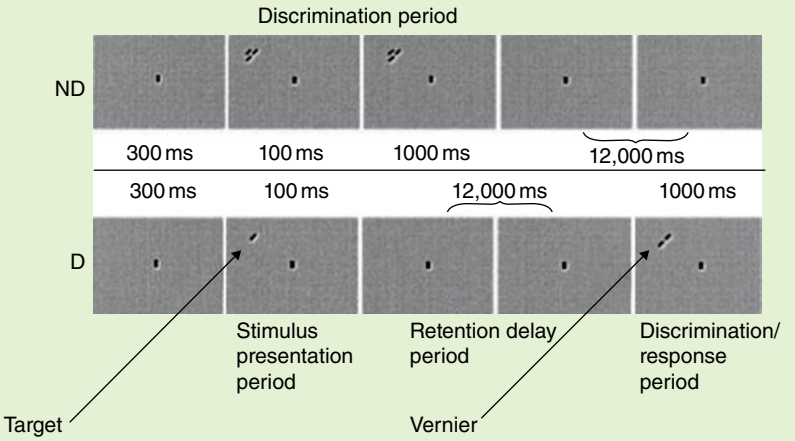

FIGURE 8.5.A The "no delay" (ND) and "delay" (D) variants of the task, in which the subject indicated whether the vernier array was further from, or closer to, fixation than the target bar.

Methodology Box 8.1 *Continued*

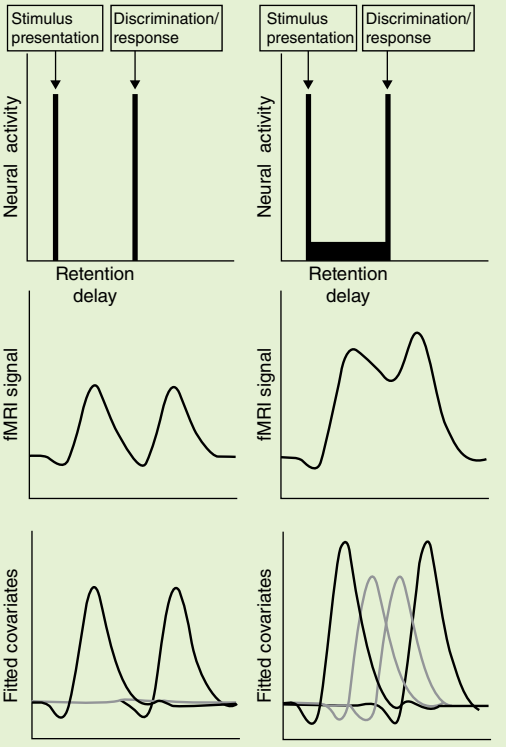

FIGURE 8.5.B A simulation of how two brain areas might respond during "D" trials on this task. The region in the left-hand column shows elevated activity during the "stimulus presentation" and "discrimination/response" epochs of the trial, but not during the delay period; the region in the right-hand column shows elevated activity across all three components of the trial. The middle row illustrates what the (noise-free) fMRI response would be in these two regions. The bottom row shows loadings on regressors ("fitted covariates") in a regression model set up to analyze these event-related data.

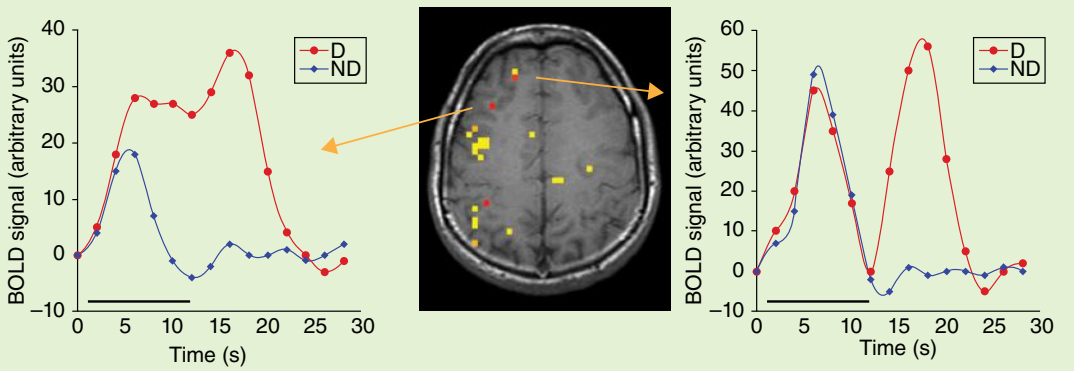

FIGURE 8.5.C The responses of two regions in the right PFC during ND and D trials. The black horizontal bar denotes duration of delay period. Note that, in the more anterior voxels, even though there is no elevated delay-period activity during D trials, discrimination/response activity is markedly higher in D trials than in ND trials. Thus, averaging across several trials of both types, as one would do in a block design, would yield a higher signal intensity value for these voxels for D trials than for ND trials, even though these voxels evince no elevated delay-period activity. Replotted by the author from Zarahn et al. (1999).

Methodology Box 8.1 *Continued*

value than a block of 10 consecutive ND trials. The interpretation, therefore, would be that both these regions exhibit significantly elevated delay-period activity. This is because the two trial types are matched with regard to the amount of time that stimuli are on the screen, the decision and response that are required, and so on. They only differ in that D trials include a delay period and ND trials do not. For the region plotted on the right, however, this interpretation would be incorrect. The activity of this region illustrates a failure of the assumption of "pure insertion," in that insertion of a delay period did *not* leave the other epochs of the task unaffected. Specifically, "discrimination/response" activity increases when a delay is inserted. (Perhaps a memory-based decision is more difficult than a perceptually based one, leading to greater probe-related activity on D vs. ND trials?)

be reading this book, but are, in fact, waiting for the imminent arrival of your roommate/boyfriend/girlfriend/spouse/whomever, and so are attending to the door, waiting for it to open at any moment.

The endogenous control of attention

Researchers at Washington University in St. Louis, led by neurologist Maurizio Corbetta and experimental psychologist Gordon Shulman, have used event-related fMRI to separate, for example, transient responses associated with a cue instructing the subject where to attend (i.e., to a location in the periphery) – or what to attend to (e.g., motion in a particular direction) – from the sustained activity associated with the top-down maintenance of attention at the cued location – or to the cued direction of motion. Importantly, on perceptually identical "passive viewing" trials, when subjects were told that there was no task to perform, and that they merely had to watch the screen, the transient responses in posterior occipitotemporal regions were only slightly attenuated relative to what they had been in attention-demanding trials (*Tip 8.8*). This was consistent with what we have already seen at the site of attention. Activity in rostral IPS and caudal superior frontal cortex, including the FEF, however, showed a very different pattern.

Tip 8.8

In the human, MT is located on the lateral surface of the occipital lobe, at the dorsoventral level of the inferior temporal gyrus.

It was markedly attenuated, almost to baseline levels, on passive trials. Also noteworthy was the fact that virtually identical patches of cortex in superior parietal and frontal regions showed elevated activity regardless of whether the sustained attention required by a task was location-based (e.g., *Detect the onset of a target at this location*) or feature-based (e.g., *Indicate when you see motion in this direction*). This led to the conclusion, consistent with results from many, many other studies performed in many other laboratories, that coordinated activity in this dorsal frontoparietal network acted as a *source* of top-down attentional control (*Figure 8.6*).

How might the dorsal frontoparietal system work? At the network level, projections from deep layers of cortex in these regions likely deliver the signals that manifest in sensory processing areas as baseline shifts (in the case of location-based attention) and/or as the biasing signal that influences the competition for representation of a behaviorally relevant stimulus vs. irrelevant items. (Revisit *Chapter 6* if the phenomena of the baseline shift and of biased competition aren't fluently springing to mind.) And at a functional level, many researchers have proposed that elevated activity in dorsal frontoparietal cortex (*Figure 8.6*) may reflect the operation of a **salience map**. The idea is that the brain maintains a representation of what's currently the most relevant for behavior, whether that be a representation of the location of potentially important stimuli in the environment, a representation of current behavioral priorities, or both.

Let's say you're new to Madison, WI, and are driving in a rental car from the airport, trying to navigate

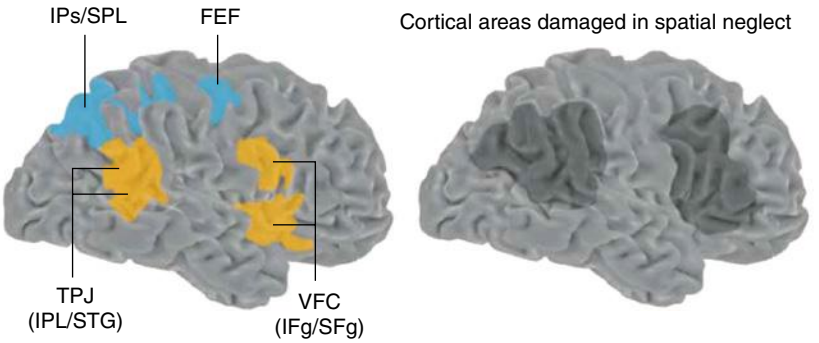

FIGURE 8.6 Two networks for the control of attention. At left, the networks associated with the endogenous (blue) vs. the exogenous (yellow) control of attention. At right, regions whose damage is often associated with hemispatial neglect. Source: Corbetta and Shulman, 2002. Reproduced with permission of Nature Publishing Group.

to my house. As you drive along Old University Avenue your eyes are, of course, on the road ahead of you. In your "mind" however, are additional representations: (1) you are searching for street signs at each intersection, so as to identify "Forest St." (which should be coming up on the left); (2) you are aware of a group of people standing near a crosswalk in front of you (*What's the law here? Do I have to stop and let them cross in front of me, or will they wait for a break in the traffic?*); and, (3) what's up with the car behind you that is driving right up on your @&&? Thus, although your eyes are, indeed, on the road, your salience map will prioritize (1) the detection of green street signs; (2) movement into the crosswalk by a pedestrian; (3) evidence of trouble in the rearview mirror.

SUDDENLY, A BALL ROLLS OUT ONTO THE STREET HALFWAY DOWN THE BLOCK!

This unexpected event triggers what Corbetta and Shulman (2002) call the "circuit breaker," which overrides the influence of your current salience map on your behavior.

The exogenous control of attention

Exogenous control of attention occurs when something salient and unexpected occurs in the world around you, and the source is, therefore, *outside* of your brain. (The endogenous control of attention, in contrast, is considered more "cognitive," in the sense that your thoughts/plans/priorities/motivations are controlling your behavior. They are endogenous in the sense that they are generated within and maintained *inside* you, in your brain.) Corbetta and Shulman have documented that exogenous shifts of attention are associated with a right hemisphere-dominant ventral network that includes the temporoparietal junction (TPJ) and the inferior frontal gyrus of the ventral prefrontal cortex (which they abbreviate as VFC). The ventral network is illustrated in yellow in the brain image on the left of *Figure 8.6*. Intriguingly, Corbetta and Shulman (2002) have noted that "the anatomy of neglect … matches the ventral TPJ–VFC system" (p. 212). (For one independent dataset generally consistent with this view (and generated several years later), revisit *Figure 6.4*; *Tip 8.9*.)

Next, we turn our attention to something that many readers will have already noticed, which is that the dorsal regions highlighted in panel C of *Figure 8.4*, which illustrates activity associated with a delayed-saccade task, show remarkable overlap with the dorsal frontoparietal system illustrated in *Figure 8.6*.

Tip 8.9

Perhaps some of you, like me, now find it easier to reconcile *Chapter 6*'s account of unilateral neglect, and particularly Figure *6.4*, with the rest of that chapter's decided emphasis of more dorsal regions of PPC.

THE CONTROL OF ATTENTION VIA THE OCULOMOTOR SYSTEM

Covert attention

There are many times in our lives when we want to attend to something, or someone, without directly looking at it (or him, or her). In high school math class there was that cute classmate on whom you had a crush, but at whom you didn't want to stare (Never happened to me, but I've heard stories …). At the dinner table, one wants to convey to one's kids that one trusts them, and so doesn't want to appear to be closely monitoring their every move, yet one also wants to closely monitor their every move (Again, so I'm told …). These are examples of covert attention. (Overt attention is when you look directly at the object of your attention.) Sustained covert attention may be related to another attentional phenomenon, which is that there is considerable data from experimental psychology, and from neurophysiology, that the focus of attention jumps to the target of the next saccade tens of milliseconds prior to the initiation of the saccade itself. Therefore, might it be the case that the sustained allocation of covert attention to a location/object amounts to a presaccadic shift of attention, but one that does not culminate in the saccade itself?

Covert attention as motor preparation?

This last bit of speculation is essentially what is posited by the **premotor theory of attention**, articulated in the 1980s by Giacomo Rizzolatti (of mirror neuron renown). It argues, in summary, that attention may fundamentally be "nothing more" than motor planning [my quotes]. Here's how Rizzolatti and Craighero (2010) have summarized it:

> Classically, spatial attention was thought of as a dedicated supramodal control mechanism, anatomically distinct from the circuits underlying sensorimotor processing … [However] there is no need to postulate two control mechanisms, one for action and one for attention. According to [the premotor theory,] spatial attention does not result from a dedicated control mechanism, but derives from a weaker activation of the same frontal-parietal circuits that, in other conditions, determine motor behavior toward specific spatial locations.

Empirical evidence for covert attention as motor preparation

The first of a series of studies by Tirin Moore and colleagues that have put this theory to the test is described in *Research Spotlight 8.1*. Learning the details and implications of this experiment may bring one, in the humble estimation of this author, the closest that one could hope to get, in a textbook such as this, to Nabokov's "aesthetic bliss" (*Tip 8.10*).

A second study from Moore offers an even more direct demonstration of the premotor theory, and is even more technically breathtaking than what's described in *Research Spotlight 8.1*. Additionally, it shows that this scheme can generalize to object-based attention, such as investigated by Chelazzi, Duncan, Miller, and Desimone (1998) and detailed in *Chapter 6*. In it, Moore and graduate student Kathleen Armstrong (2003) matched up FEF neurons and V4 neurons whose motor fields and receptive fields, respectively, overlapped (*Tip 8.11*). In this way, they were able to stimulate at a putative source of attentional control while simultaneously recording from a site. *Figure 8.8* illustrates that the effect of FEF stimulation was to boost the V4 visual response in a manner similar to the effects of selective attention that we considered in *Chapter 6*. Importantly, in this study *the monkey was not performing a task*, other than maintaining central fixation. Thus, as with Moore and Fallah (2001), it is a remarkable demonstration of the ability to control the focus of attention via a decidedly exogenous intervention in the activity of a neural source of the endogenous control of attention (*Tip 8.12*)!

Where's the attentional controller?

Remember in *Chapter 7*, when a skeptic chimed up about "some little homuncular mathematician perched up on the precentral gyrus … calculating vector sums prior to each movement"? At this point s/he might chime in again, asking sarcastically if we've placed the spotlight operator (from *Chapter 6*'s "Day at the Circus") in the FEF. Stilted rhetorical devices aside, this is actually a legitimate concern, and one about which the cognitive neuroscientist and, particularly, the theoretician, needs always to be mindful. *Where does this control come from? Does assigning it to the dorsal frontoparietal system make it any less mystical, any more material?*

RESEARCH SPOTLIGHT

8.1 Controlling the spotlight of attention with microstimulation of the FEF (Moore and Fallah, 2001)

In previous chapters we have emphasized the value of being able to causally influence the system under study in systematic ways. This is illustrated very elegantly in the study highlighted here, which tests a hypothesized mechanism of attentional control. The first step of the experiment is to find a neuron in FEF whose microstimulation produces a repeatable saccade to the same location (e.g., *Figure 8.3*). This is referred to as the cell's motor field (by analogy to the sensory receptive field; panel A in *Figure 8.7*). Next, the layout of the covert attention task – on which the monkey has already been trained – is arranged so that the target stimulus is located in that neuron's motor field (panel B in *Figure 8.7*). In the task, the monkey is to depress

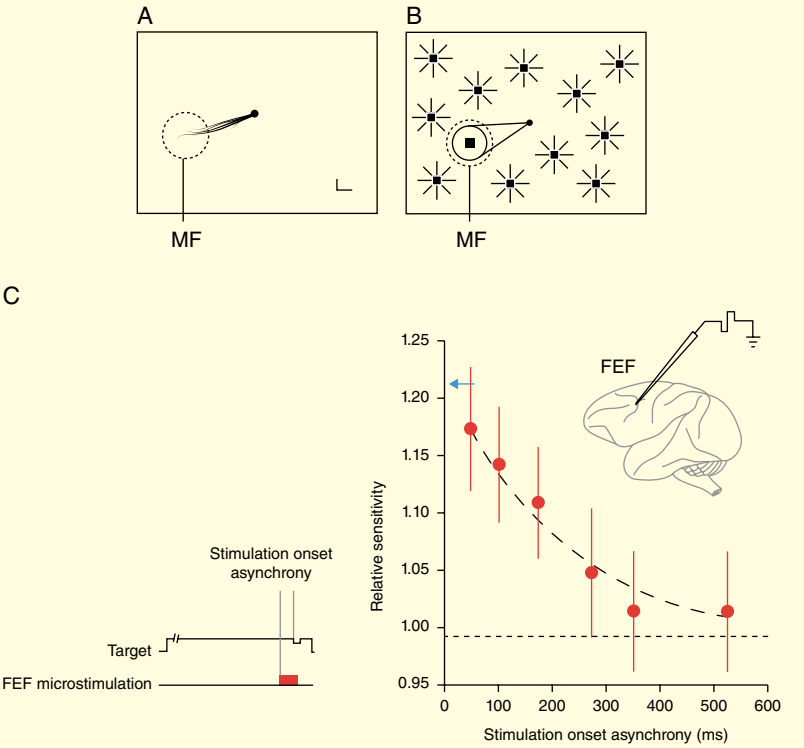

FIGURE 8.7 Electrically controlling the source of endogenous attentional control. **A.** Microstimulation of a location in the FEF of a monkey produces reliable vector saccades into a motor field (MF). **B.** The array of the visual search task is customized for that neuron, such that the target location falls within this MF. **C.** Microstimulation-related improvement in performance varied monotonically with the stimulation onset asynchrony (see text). Source: Awh, Armstrong, and Moore, 2006. Reproduced with permission of Nature Publishing Group.

Research Spotlight 8.1 *Continued*

a lever to start the trial, and maintain central fixation while attending to the target. When the target dims, ever so slightly, the monkey is to release the lever in order to receive its reward. The dimming of the target is made difficult to detect by making the luminance change very small and very brief, by making the timing of the event unpredictable, and by including catch trials in which the target never dims. Finally, as if that wasn't already enough, most trials also feature the concurrent presentation of flashing distractors throughout the trial. (Distractors flashed on and off, one at a time, in the locations illustrated with "sunbursts" in panel B in *Figure 8.7*.)

Now for the really cool part. Recall that the target is positioned within the motor field of the FEF neuron. This means that, if the investigator wanted to, he could deliver a 100 ms train of ~ 50 μA of current and drive the eyes to the target location. Instead, they did what Tirin Moore sometimes characterizes as "just tickling" the neuron, by stimulating at half the amplitude that had been determined to produce a saccade 50% of the time. That is, the stimulation was subthreshold, in that it never produced an eye movement.

But would it – drumroll please – *produce a covert shift of attention*?

To assess this, the investigators varied the latency between when microstimulation was applied and when the target event occurred, a parameter called the "stimulation onset asynchrony." The findings suggest that "tickling" an FEF neuron does, indeed, produce a covert shift of attention, and that it does so in a timing-dependent manner: the closer in time the microstimulation was to the target event, the stronger were its attention-like effects. The effects, plotted in panel C in *Figure 8.7*, express the effect of microstimulation on the animal's "relative sensitivity," which is a measure of how much smaller the magnitude of the dimming needed to be made for the monkey to perform at the same level as it did on no-stimulation trials. Thus, in effect, what the plot shows is that delivering microstimulation with a stimulation onset asynchrony of 50 ms improved the animal's performance by roughly 17.5%. For comparison, the blue arrow shows that turning off the distracting stimuli improved the monkeys' performance by ~ 21%.

In effect, instead of overtly cuing the animal about an impending target event, a la Posner (1980), Moore and Fallah "reached into its brain" and directly shifted the animal's attention!

Tip 8.10

For Nabokov, the best fiction afforded "a sense of being somehow, somewhere, connected with other states of being where art (curiosity, tenderness, kindness, ecstasy) is the norm" (Nabokov, 1955, p. 286). For me, the best science does this, too.

Tip 8.11

Tirin Moore had been a doctoral student of Charles Gross, of hand- and face-cell fame (*Chapter 5*), left to do a postdoc, then returned to Gross's lab (at Princeton University) for three additional years as a research scientist. Both Mazyar Fallah and Kathleen Armstrong were graduate students of Gross, collaborating with Moore, when this research was performed.

Have we accomplished anything more than assigning a more specific address to the ghost in the machine? The answer is that, in fact, in the domain of attentional control, and in many others, cognitive neuroscience has made important strides toward developing very explicit accounts of how control emerges as a consequence of network interactions. In these models, there is no one place from which control signals are generated de novo, no figurative "corner office" from which a neural executive spontaneously decides to issue commands.

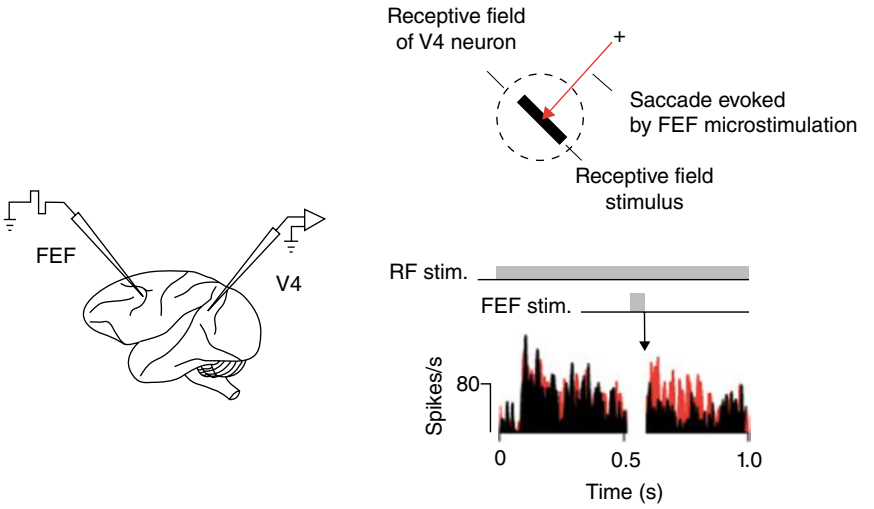

FIGURE 8.8 Generating the cause and measuring the effect of attentional control. Simultaneous stimulation of an FEF neuron and recording from a V4 neuron with overlapping motor field and receptive field, respectively. Effect of visual stimulus (oriented bar) on activity in the V4 neuron is illustrated on trials without FEF stimulation (black histogram) and with FEF stimulation (red histogram). Source: Awh, Armstrong, and Moore, 2006. Reproduced with permission of Nature Publishing Group.

Tip 8.12

If what you attend to determines what you think about, then what Moore and colleagues are doing may be, in effect, controlling what their monkeys are thinking about!

Such models are often most convincingly achieved via computational simulations. Although such simulations invariably include some idiosyncratic choices made by the modeler that may not account for every detail that we (think we) know about how the brain works, their value is in demonstrating that it is possible, in principle, to understand, to describe, and to measure every step, every connection, every causal factor in a system capable of producing complex behavior. For the question of the source of attentional control, we can consider a computational model by the German theoretician Fred Hamker.

The reentry hypothesis

Research Spotlight 8.2 details this computer network model, the goal of which was to simulate the findings of Chelazzi et al. (1998) that provide an important empirical foundation for the biased-competition model of visual attention (*Chapter 6*). In particular, this model provides an explicit account for how the FEF (and IT) act as sources of the top-down attentional control that provide the biasing signal. Additionally, by specifying and explicitly modeling the activity of different classes of neurons in the FEF, it provides a plausible scenario for how a saccade plan might be prevented from actualization in a saccade, and, thus, could also serve as the basis for sustained covert attention. (Hint, it would involve the fixation neurons.)

Another clue to the question of *Who controls the controller?* comes from the research presented in *Web Clip 8.2*, which details the influence of the modulatory neurotransmitter dopamine on the attentional-control functions of the FEF.

ARE OCULOMOTOR CONTROL AND ATTENTIONAL CONTROL REALLY THE "SAME THING"?

So, we've seen evidence that FEF can act as a source of attentional control when it's electrically stimulated, and we've considered a model describing how it

RESEARCH SPOTLIGHT

8.2 A computational account of the control of visual attention (Hamker, 2005)

Figure 8.9 illustrates the architecture of the model (panel B), and the anatomical connections on which it is based (panel A). One of the features of this model, which we haven't considered up to this point, is the heterogeneity of neuronal classes within FEF. Not unlike the deep layers of the superior colliculus, within FEF one can find visually responsive neurons, fixation neurons, visuomotor neurons, and movement neurons. Three of these are explicitly represented in the model (panel B in *Figure 8.9*), as are their temporal dynamics:

> Visuomovement cells in deep layers are active from stimulus onset until saccade execution. Typically their initial response does not distinguish between distractor or target, but the activity decays when a distractor is in the receptive field. Movement cells are active prior to saccades and do not show any response to stimulus onset. Fixation cells decrease their activity before a planned eye movement. (Hamker, 2005, pp. 433–434)

Now, how does this network implement attentional control? We can watch it happen, in *Figure 8.10*, on a (simulated) millisecond-by-millisecond basis:

0 Not shown is the beginning of the trial, when initial presentation of the target object establishes the search template for that trial, which is retained in "working memory" in recurrent connections between PF and IT (between "Working memory" and "Target" populations) until the search array is presented. (For a cartoon of the task, see *Figure 6.20*.)

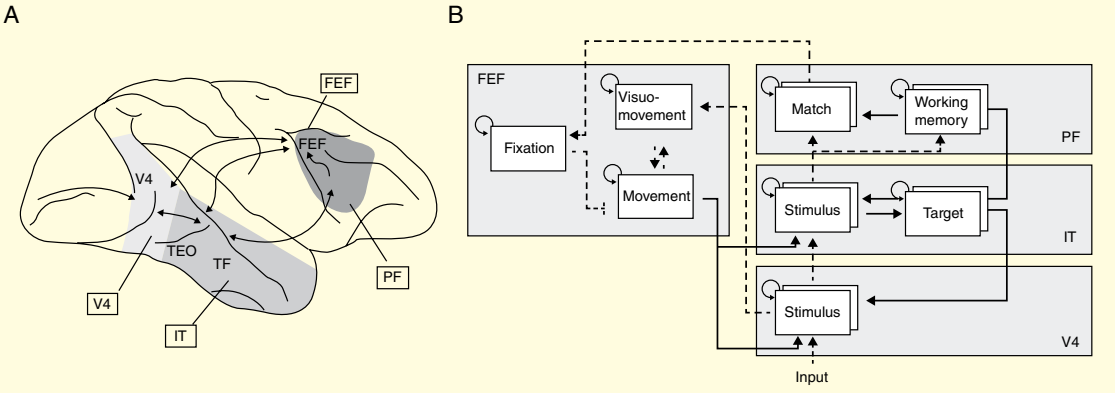

FIGURE 8.9 Illustrations from Hamker's simulation of the source of top-down control. Panel **A**, the known neural connections included in the model. Panel **B**, a schematic of the implementation of the model. Bottom-up connections are indicated in dotted arrows, feedback ("reentrant") connections in solid arrows. Each white box represents a population of neurons, "stacked" boxes indicating populations processing two stimulus features (color and form) in parallel. The "turning back on itself" arrow at the upper-left corner of each of the boxes indicates that individual units within a population exert lateral inhibition on each other. Source: Hamker, 2005. Reproduced with permission of Oxford University Press.

Research Spotlight 8.2 *Continued*

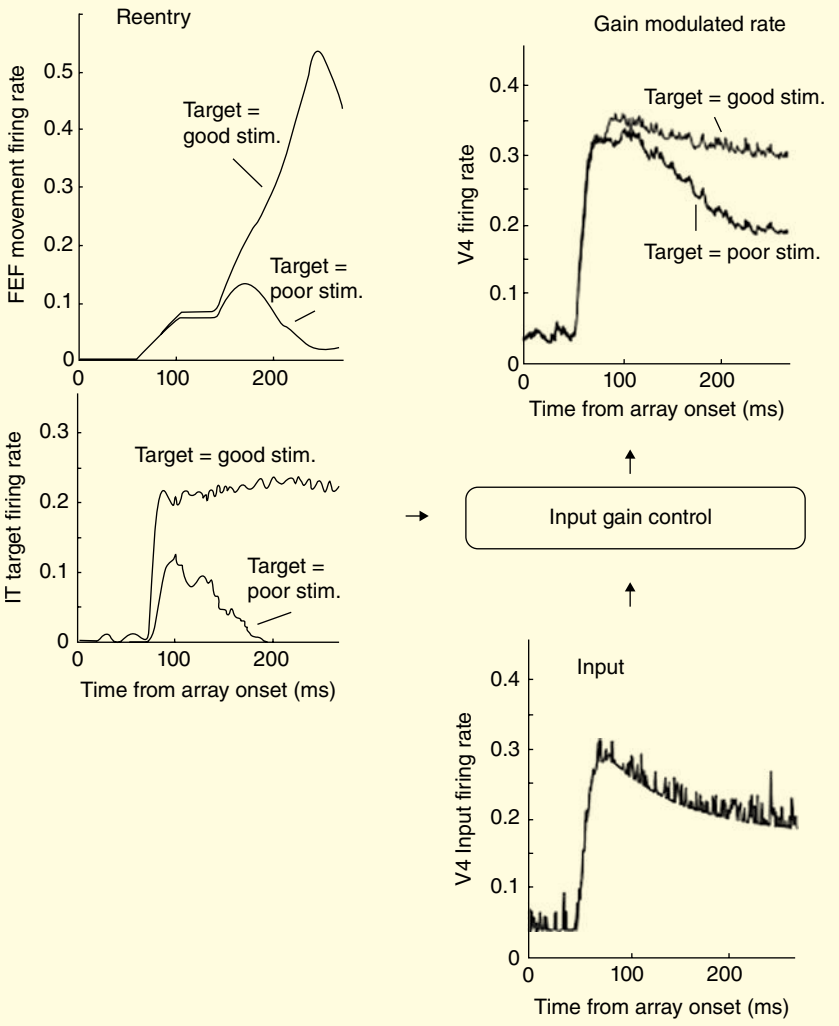

FIGURE 8.10 Multiple windows on the temporal evolution of activity occurring simultaneously at multiple stages of the simulated oculomotor system from *Figure 8.9*, for a V4 receptive field/FEF movement field processing information about a stimulus that is the V4 neuron's "good" stimulus (definitions in *Figure 6.20* and associated text). The "Input" box shows the stimulus-driven input to a V4 neuron from visual cortex. The "Input gain control" implements the **convolution** of this Input signal with the "Reentry" signals from FEF and IT (i.e., it's the operation in the model that computes the effects of the reentrant signals on the Input) and the "Gain modulated rate" shows the resultant activity in V4 (*Tip 8.13*). Source: Hamker, 2005. Reproduced with permission of Oxford University Press.

Research Spotlight 8.2 *Continued*

1 When one stimulus of the search array appears within the simulated receptive field, increased input elevates activity within V4 neurons tuned for particular stimulus features, and this feeds forward to FEF, producing the initial ramp-up in the *FEF movement firing rate* that begins ~ 75 ms into the trial.
2 Meanwhile, neurons in IT have knowledge (from working memory) about the target, and so their early activity to the stimulus reflects whether it is the target (*Target = good stim.*) or not.
3 [First wave of *reentry*:] Feedback from IT to V4 produces the initial little bump in *Target = good stim.* activity in the "Gain modulated rate" of V4 that begins prior to 100 ms.
4 Although the receptive field of any one V4 neuron is relatively large, the aggregate result of feedforward activity from a large population of them is that visuomovement neurons in FEF that encode the region of the visual field containing the target will receive the strongest feedforward drive (a phenomenon reminiscent of population coding in M1). The visuomovement neurons in FEF receiving the strongest feedforward drive will, in turn, activate the FEF movement neurons whose movement field overlaps the location of the target. The accumulating effect of the small differential feedforward drive from V4 begins to emerge in FEF activity at ~ 130 ms.
5 [Second wave of *reentry*:] Increasing activity in FEF movement neurons is experienced by V4 neurons as top-down feedback, and those representing features of the target, which already have a slightly elevated level of activity, benefit from this top-down boost to further increase their activity and, in turn, apply lateral inhibition to V4 neurons representing features of the nontarget stimulus.
6 These processes continue, in parallel, until a threshold is reached and a saccade to the target triggered.

(*Tip 8.14*.)

Tip 8.13

The Hamker (2005) paper presents several side-by-side comparisons of model outputs of simulated "Gain Modulated Rate," from various conditions, with the comparable empirical data from Chelazzi et al. (1998; such as those illustrated in *Figure 6.21*).

might come to serve this function. But is there evidence that this *is* how attention is controlled in the human? That is, can the endogenous control of attention truly be reduced to "nothing more" than covert oculomotor control? Although this is a difficult question to answer unequivocally, we'll conclude this chapter considering one way of addressing it.

Tip 8.14

Note that this model only takes into account firing rates, and how an increase (or decrease) in an area affects the region(s) that it projects to. There is, however, a growing body of electrophysiological evidence that dynamic changes in oscillatory phase synchrony between regions also plays an important role. *Web Clip 2.3*, for example, presented evidence for IT alpha-band oscillations having a pacemaker role for V4 alpha-band oscillations. Other research suggests that the efficacy of FEF influence on posterior regions may depend, in part, on long-range synchrony between these regions in the beta band.

The "method of visual inspection"

As noted previously, there is a striking similarity between the dorsal frontoparietal regions activated in the delayed-saccade task of Srimal and Curtis (2008; *Figure 8.4*), and the endogenous control network of Corbetta and Shulman (2002; *Figure 8.6*). Indeed, there have been many demonstrations in the literature that tasks engaging these two categories of behavior activate overlapping regions of the brain. However, there's only so far that one can get by, in effect, saying, *See? These two patterns of activity look the same.* The reason for this is that brain activity – whether considered at the level of a single neuron, a cortical column, a population of 100 or so neurons, a 3 mm^3 fMRI voxel containing tens of thousands of neurons, or a whole brain fMRI volume comprising ~30,000 such voxels – is simply too complex to be interpretable by visual inspection. Thus, for example, one cannot compare the patterns of activity illustrated in *Figure 8.4* and *Figure 8.6* and conclude, with any certainty, that the brain is "doing the same thing" during the two tasks that produced these images. Why? First, one can find many, many examples of experiments in which these same regions are active, but subjects are not performing either a spatial attention task or an oculomotor task. The IPS, for example, also shows elevated activity during tests of mental arithmetic and numerosity, of learning sequences of finger movements, of "understanding the goals of others," of perceptual decision making, and so on. Second, what if activity that you can't see determines the function that IPS carries out? Perhaps there's different "unseen" activity happening in *Figure 8.4* vs. in *Figure 8.6* (recall, from *Methodology Box 6.1*, the uncolored parts aren't inert, their activity simply isn't exceeding a statistical threshold). Simply put, there is no brain area whose activity is specific for any single cognitive function. ("Take that, *Thought Box 1.1* and *Methodology Box 1.1* …")

Another way to think about this is that a limitation of the "visual inspection" approach is that it is inherently univariate. That is, we look at the blob in IPS in *Figure 8.4* and can only watch it vary along one dimension: its signal intensity either increases or decreases. This can lead to thinking of it as one "activation" doing only one thing (*Tip 8.15*).

Tip 8.15

Now I know that "*hate* is a strong word"; I frequently remind my kids of this. But this author hates the term "an activation," because it implies a physical (and physiological) reality for something that doesn't actually exist. One can measure *the activation* of a system, or *activity in a region*, but *an activation* implies the existence of a thing, like a cloud or a shadow. They do exist on the figures that we use to illustrate the results of our studies, but not in any biological sense. "Blob" would be a better term, because it can be used interchangeably, but the inherent silliness of the word keeps us from overinterpreting the significance of "an activation."

Finally, *These two patterns of activity look the same* isn't really what we care about. To address the question posed in the title of this section, what one would really want to be able to ask is *Is the brain actually doing the same thing during the performance of these two tasks*? One approach that offers a way to get closer to being able to ask this question is to apply one of the relatively recently developed methods of multivariate pattern analysis (MVPA) to one's neuroimaging data. After first introducing some of the principles of MVPA in *Methodology Box 8.2*, we'll wrap up this chapter by considering how one study has used it to ask this fundamental question.

"Prioritized Maps of Space in Human Frontoparietal Cortex"

This is the title of a study from the laboratory of Clayton Curtis, the same group that produced the delayed-saccade data illustrated in *Figure 8.4*. In it, Jerde et al. (2012) first employed a method similar to the one illustrated in *Figure 6.9.A* to identify cortical regions supporting retinotopic maps of space (*Figure 8.12.A*). Within the resultant ROIs, which included the dorsal frontoparietal regions associated with oculomotor control and with the endogenous control of attention, they applied MVPA to data from three tasks: oculomotor delayed response

METHODOLOGY BOX

8.2 Multivariate pattern analysis (MVPA) of fMRI data

Let's consider the hypothetical example illustrated in *Figure 8.11*, in which there are two voxels in a reduced dataset, voxel *F* and voxel *P*. On 10 trials our subject attends to the left, and on 10 trials she attends to the right. The first analysis that we run is to "collapse across" all the voxels in our ROI. That is, to average their signal together, then compare the spatially averaged signal from the 10 rightward trials vs. the spatially averaged signal from the 10 leftward trials. The bar graph in panel A of *Figure 8.11* illustrates that one can detect no appreciable difference between the two conditions (*Tip 8.16*). If we consider the two voxels separately, i.e., we determine the average signal from voxel *F* separately for the 10 leftward trials and the rightward trials, and do the same for voxel *P*, we seem to do a little bit better: keeping in mind that these are made-up data, paired *t*-tests indicate that voxel *F* shows significantly greater activity for leftward than rightward trials, and voxel *P* the converse. Nonetheless, each voxel also shows robust activity for its "nonpreferred" direction.

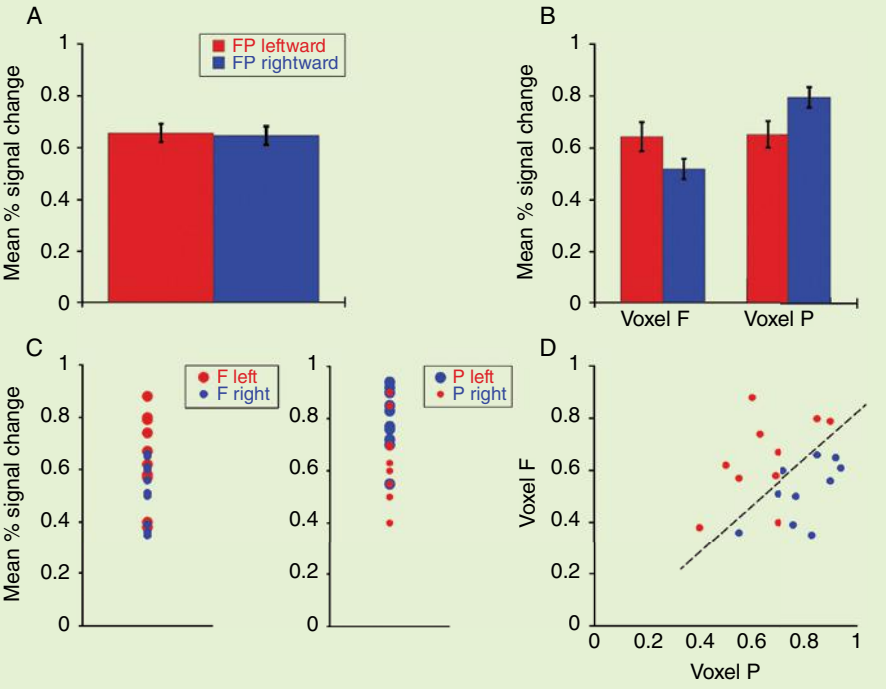

FIGURE 8.11 Improved sensitivity via MVPA. Plot **A** illustrates the data for the two conditions, averaged across voxel F and voxel P. In plot **B**, the data are broken out by voxel. The plots in **C** illustrate the signal intensity from each trial, for each voxel. Plot **D** is a multivariate re-presentation of the data from **C**, with a discriminant line added to illustrate how one would classify a 21st trial, depending on which side of the line it falls. Source: © Bradley R. Postle.

Methodology Box 8.1 *Continued*

A consequence of this is that, if we were presented with data from a 21st trial, we'd have difficulty classifying it as a leftward or a rightward trial if we only knew that voxel *F*'s activity on this trial was, say, 0.6. To illustrate this, consider panel C, which shows each voxel's response on each of our 20 trials (these are the data that produced plots A and B): where, on the voxel *F* plot, would you draw the line above which you'd be confident that a trial was a leftward trial?

The two "stacks" of data points plotted in panel C are 1-D values – they only vary along a line. Panel D illustrates how the same data used to generate the plots in A, B, and C can be re-depicted as data that vary along *two* dimensions. To create the plot in *Figure 8.11* D, all we've done is rotate the plot of voxel *P*'s activity – from panel C – from vertical to horizontal. That permits us to "project the data from each trial into a higher dimensional space." (Sounds high-falutin', I know, but it *is* what we are doing; 2 is higher than 1 …) Put more simply, it permits us to plot what each trial's values were at the two voxels simultaneously. As a result, it's easier to decide "where to draw the line": if the hypothetical 21st trial falls above and to the left of the discriminant line that cuts through the cloud of data points, it's probably a leftward trial.

With real fMRI datasets, this same principle is applied, but the assertion that one is "projecting the data into a high-dimensional space" no longer elicits snickers, because, with, say, 300 voxels in an ROI, one is working in 300-D space. Within this space, **machine learning** algorithms find the high-dimensional hyperplane that, like our dotted line in *Figure 8.11* D, provides the best discrimination between conditions A and B. The higher sensitivity afforded by MVPA will be exploited in many experiments that we will consider from here on out.

Tip 8.16

Note that this type of averaging across large numbers of voxels has been a bread-and-butter approach to cognitive neuroimaging, from its earliest days up through the day that these words are being typed. In this book, it has been illustrated in, e.g., *Figures 8.4* and *8.5*; and *6.8* and *6.18*.

(ODR; "intention"; see, e.g., *Figure 8.4*); spatial "attention" (see, e.g., *Figure 8.7*); and spatial delayed recognition (short-term "memory"; e.g., *Figure 8.6*). (That is, after acquiring the retinotopic mapping data in one scanning session, the researchers had their subjects return for follow-up sessions in which they performed these three experimental tasks.) To address the question of *Is the brain actually doing the same thing during the performance of these* three *tasks?*, they employed a clever variant of MVPA, and produced a remarkable result.

A common use of MVPA is as a tool for "decoding" brain activity. Consider *Methodology Box 8.1*. After plotting the results of 20 trials of leftward vs. rightward attention, we felt reasonably confident that we would be able to classify the identity of a 21st trial, based solely on the neural activity associated with this trial. That is, we felt reasonably confident that we could *decode* the neural activity so as to know what the subject was thinking about. (Well, at least, so as to predict whether the subject was attending to the left or to the right.) What Jerde et al. (2012) did with their data is a step beyond "just" predicting the identity of a 21st trial drawn from the same task that produced the first 20 trials. Rather, what they did was train a classifier to discriminate between *attend-left* and *attend-right* trials, then asked it to classify trials from the intention task. That is, they defined the high-dimensional space into which *attend-left* and *attend-right* trials fell, established the high-dimensional plane that best discriminated *attend-left* from *attend-right* trials, then introduced trials from the ODR task and tried to predict, based on where the ODR data fell

in this attention-defined high-dimensional space, whether these were *saccade-left* or *saccade-right* trials. The only way that so audacious an approach could work would be if, in fact, *the brain was actually doing the same thing during the performance of both of these tasks.*

And it did work. Figure *8.12.B.* illustrates that in sPCS (i.e., the FEF), and IPS2 (i.e., superior IPS), a multivariate classifier trained on only *intention* trials could decode data from *attention* trials, and vice versa. This finding provides some of the strongest evidence to date that "oculomotor control" and "spatial attentional control" may be two verbal labels that we use in different behavioral contexts but that, in fact, engage the same underlying neural activity. (This study and its implications are taken up in more detail in *Web Clip 8.3.*)

OF LABELS AND MECHANISMS

As humans, we like (need?) to categorize things. I just traded in my son's "hybrid" bicycle (with properties of a "street bike" and of a mountain bike) for a (expensive!) mountain bike. To a nonexpert, the two would seem to be only superficially different from one another, but to my 11-year-old expert, they're as different as are a minivan from a sports car. To him, and to the biking community, they belong in different categories. To our intuitions, the act of moving one's eyes and of choosing to focus one's attention on object A vs. object B may feel like two different kinds of behavior. Certainly, up until quite recently, psychology and neuroscience have treated these as two different domains. But what if, as the data that we reviewed in this chapter would suggest, it is the very same underlying neural systems and processes that produce these two putatively different kinds of behavior? Surely, at least on the neural level, they can no longer be assigned to different categories. This is one of the reasons why so much attention, and often debate, is focused on seemingly arcane questions such as *Are PPC neurons encoding* intention *or* attention*?*, or *Are Moore's attention-like results attributable solely to the activation of sensory neurons in the FEF, or do movement neurons also contribute?* This tension between different levels of description will reoccur often during the remainder of our time together. Just one example is manifested in *Figure 8.12*, which impels us to include spatial STM in this discussion of neural mechanisms vs. psychological categories, even though we're not "supposed" to discuss it in detail until *Chapter 13* …

FIGURE 8.12 Is the brain "doing the same thing" during saccade planning and attentional orienting? Source: Jerde, Merriam, Riggall, Hedges, and Curtis, 2012. Reproduced with permission of the Society of Neuroscience.

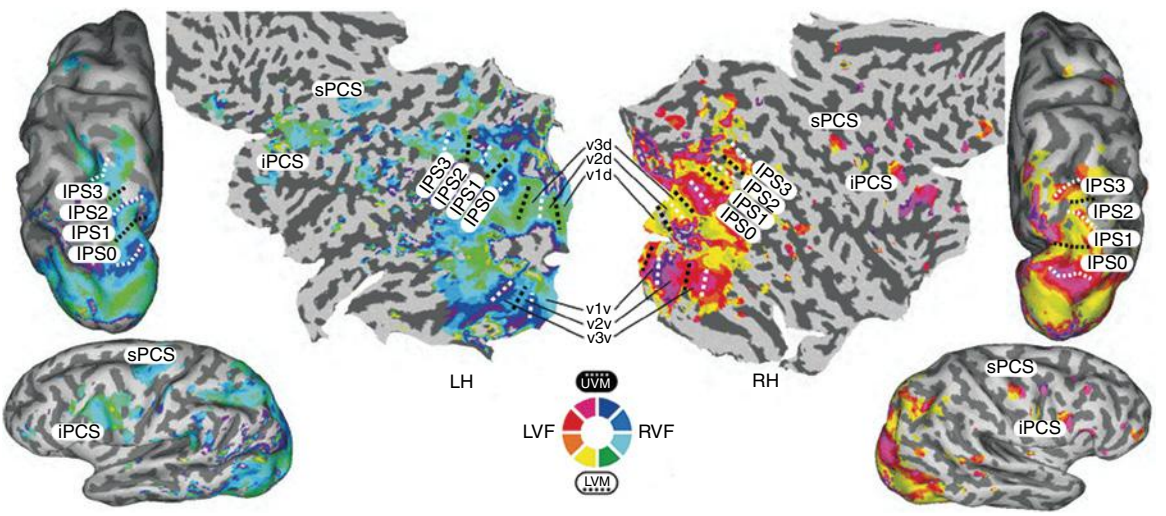

FIGURE 8.12.A The results of retinotopic mapping (compare with *Figure 6.9*), used to define the ROIs to which MVPA was applied to data from tests of "intention," "attention," and "memory."

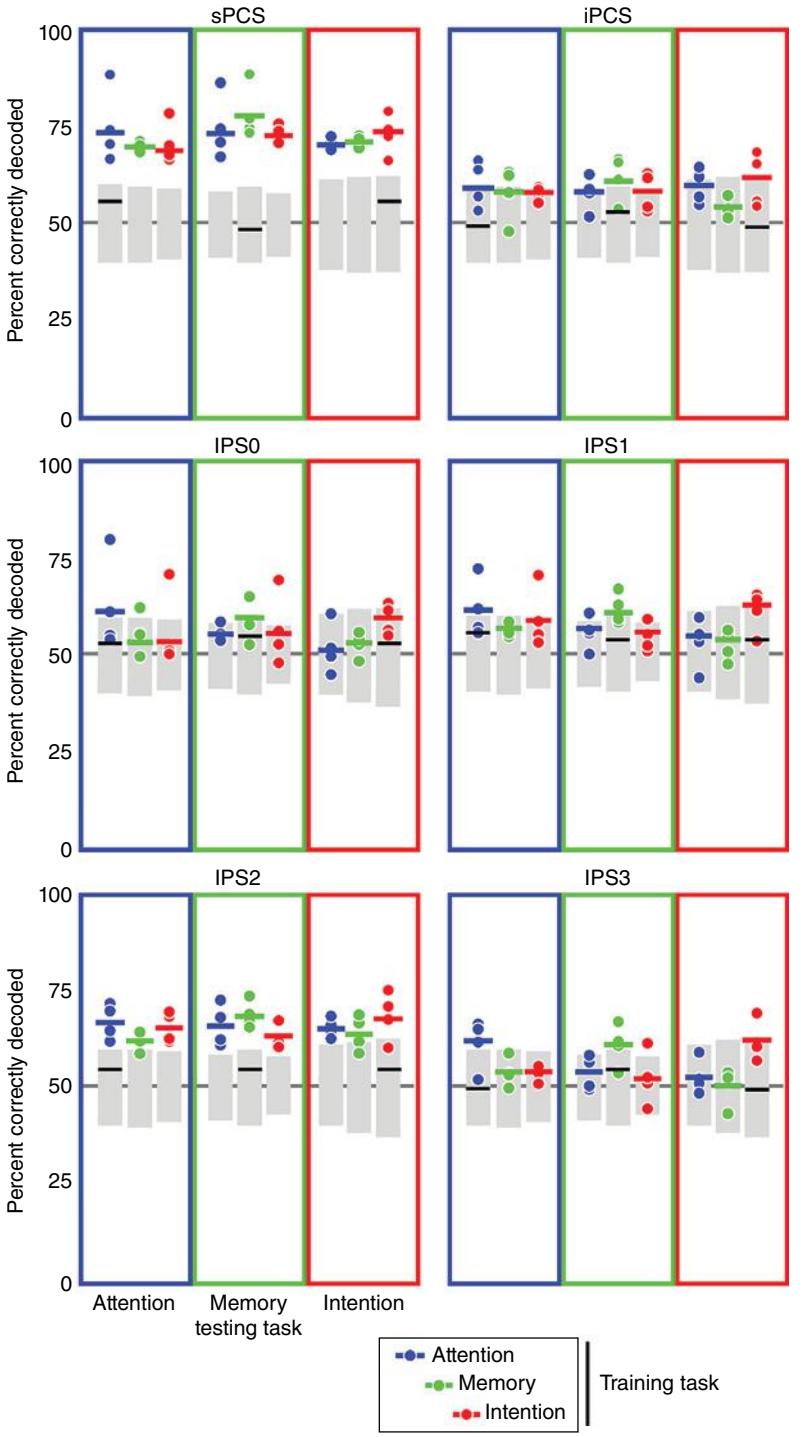

FIGURE 8.12.B Classification of data from tests of "intention," "attention," and "memory." Each dot corresponds to a subject. The color of each set of dots indicates the task used to train a classifier; the color of each rectangle around a set of dots indicates the task used to test the classifier. Thus, e.g., the upper-left rectangle illustrates that, within sPCS, not only were *attention* trials decodable by the attention-trained classifier, but so, too, were *memory* trials and *intention* trials.

END-OF-CHAPTER QUESTIONS

1. What are some important differences between the posterior and anterior oculomotor control systems?
2. There are at least three different neural codes used to encode saccade metrics at different stations of the oculomotor system. Name the three codes, and the region(s) associated with each.
3. Define the exogenous vs. the endogenous control of attention. What cortical networks are associated with each?
4. How has the development of event-related methods for fMRI been critical for the study of oculomotor control and of attention? Describe one example from each domain.
5. Summarize the data that implicate the FEF as an important *source* of attentional control.
6. How does explicit computational modeling of the network interactions underlying visual search dispel the need to locate an attentional control homunculus in dorsal frontoparietal cortex?
7. How does MVPA differ fundamentally from neuroimaging data analysis methods that predominated during the decades of the 1990s and the 2000s? What advantages does MVPA offer?
8. Are "oculomotor control" and "attentional control" just two different labels that we have given to the same underlying neural mechanisms depending on the behavioral context in which they are used, or are there fundamental differences between the two? Defend your answer with data.

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