CHAPTER 14

Two Visual Streams: Neuropsychological Evidence

DAVID P. CAREY

The science of goal-directed movement has always been multidisciplinary. Recently, scientists from motor control and kinesiology have developed substantial interest in the neurobiology of reaching in nonhuman primates and in patients with brain damage and deficits in looking and reaching. The two-visual systems model of posterior neocortex has enjoyed considerable popularity outside of clinical neuropsychology and certainly has generated substantial praise (in parallel with criticism) within it. After more than 15 years, a reappraisal of one variant of the model is well overdue. This particular variant is especially relevant for scientists interested in sensorimotor control and its interface with cognition. In this chapter I argue that early concerns about the overreliance of the model on one patient with visual agnosia have been addressed through new work on optic ataxia. Some of this latter research on dorsal stream function and dysfunction, inspired by the two-visual systems theory, is now extending beyond the limits of the model.

Two Visual Pathways in the Cerebral Cortex

Work in the 1960s by Trevarthen, Ingle, Schneider, and others anticipated the heavily cited two visual systems hypothesis of Ungerleider and Mishkin (1982) and its subsequent revision by Milner and Goodale (1995; Goodale & Milner, 1992, Milner & Goodale, 2008).[1] In fact, several works from clinical

[1] Jeannerod (1994) also suggested a variant of the model that differs from that of Milner and Goodale mainly in how much action representation is represented in the dorsal stream (see Jeannerod, 1994). In addition, Glover and Dixon have a variant that subdivides motor functions into planning and control (see Glover, 2004; Glover & Dixon, 2001).

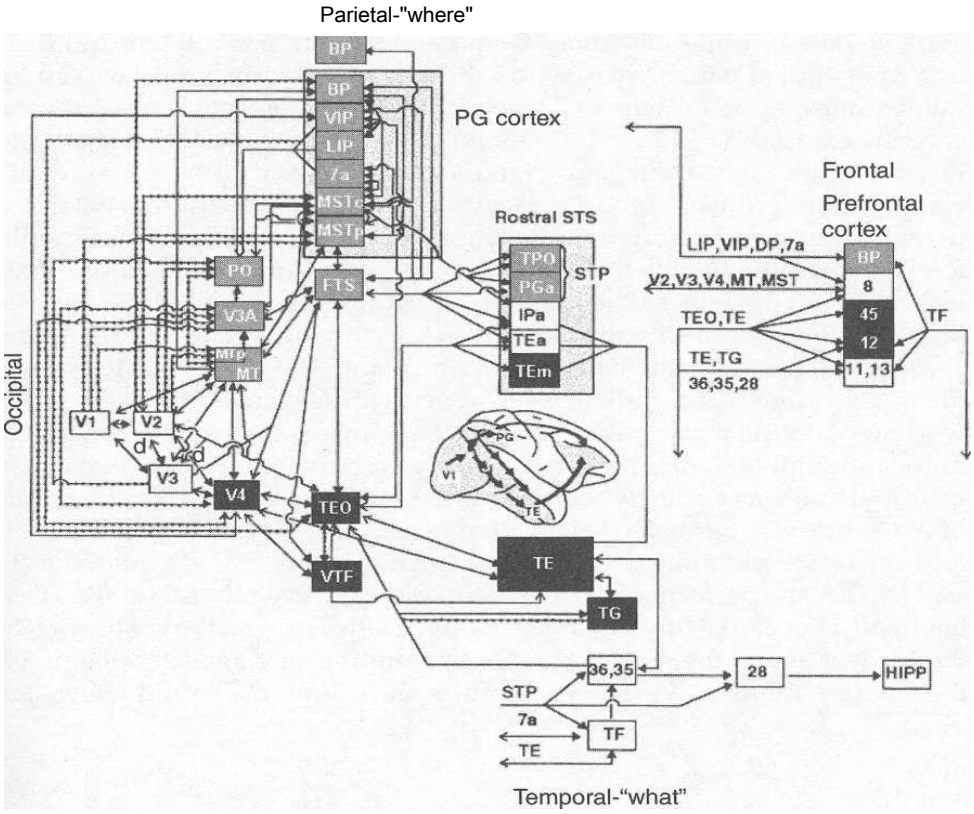
neurology (e.g., Kleist, 1934, cited in Grusser & Landis, 1991) and neuroanatomy also suggested a distinction between spatial and object vision (e.g., Glickstein, May III, & Mercier, 1985). Nevertheless, the Ungerleider and Mishkin and in particular the Milner and Goodale models have become the most influential. The purpose of the present chapter is to review these alternatives in terms of the two decades of research on patients with brain damage that have passed since the initial formulations.[2]

The two visual streams (see figure 14.1) have been discussed so extensively in visual neuropsychology that only a brief precis is provided here. The ventral chain of areas in the occipitotemporal cortex plays crucial roles in the processing of perceptual information about form, faces, color, and other visual attributes crucial for recognition and perception of visual stimuli. The distinction between the two variants of the two-visual systems hypothesis is in what the dorsal stream of the occipitoparietal cortical regions is specialized for. In Ungerlieder and Mishkin, spatial vision, including judgments of spatial attributes (such as which of two targets is closer to a landmark) and the use of spatial information to guide movement, is crucial, where as in the Milner and Goodale account, the use of any visual attribute (not just spatial position such as distance or orientation) for the guidance of sensorimotor acts is crucial.

Early Arguments Against the Milner and Goodale Account

Critics of any two-visual systems account frequently argue that cross talk between visual cortical areas in both streams invalidates these simple dichotomies. However, these critics often ignore much of the excellent anatomy performed by Ungerleider and colleagues (e.g., Boussaoud, Desimone, & Ungerleider, 1992; Ungerleider & Mishkin, 1982), which has stood the test of time. The two pathways are relatively independent of one another—they are interconnected much more heavily within stream than across stream (e.g., Catani, Jones, Donato, & ffytche, 2003; Felleman, Xiao, & McClendon, 1997; Galletti et al., 2001; Shipp, Blanton, & Zeki, 1998) and mainly communicate with one another via the superior temporal polysensory area, the frontal eye fields, and a few other subdivisions of the premotor and prefrontal cortex (Boussaoud, Ungerleider, & Desimone, 1990; Young, 1992). The two streams also have relatively distinct targets in the frontal lobes (Grol et al., 2007; Petrides & Pandya, 2007; Tomassini et al., 2007).

[2]Many neuroimaging and behavioral studies that speak to these issues will not be addressed in this chapter (see Culham, Gallivan, Cavina-Pratesi, & Quinlan, 2008; Culham, Cavina-Pratesi, & Singhal, 2006; Konen & Kastner, 2008), but in my eyes the key issues are related to imaging studies that show *superior* parietal activation in the absence of covert or overt motor responses. Behavioral studies on the dissociations between illusion and action as well as the effects of delay on sensorimotor responses are also important but cannot be addressed here.



✾ **Figure 14.1** An anatomically detailed variant of the primate two-visual systems schematic. The back of the brain is to the left of the figure. Lighter gray boxes represent distinct cortical areas of the dorsal stream (labeled by this group as *where);* darker gray boxes represent areas of the ventral stream *(what).* Areas of integration in the superior temporal sulcus (STS) and prefrontal cortex are also depicted.
"Pathways for motion analysis: Cortical connections of the medial superior temporal and fundus of the superior temporal visual areas in the macaque," D. Boussaoud, L.G. Ungerleider, and R. Desimone, *Journal of Comparative Neurology* Vol. 296,1990, pgs. 462-495. Copyright 1990. Adapted by permission of John Wiley & Sons, Inc.

A second critique of two-visual systems accounts is that any idea involving only two visual streams must be grossly oversimplified and is at best a crude heuristic. It is difficult to argue with the former statement, but the latter (the crudeness of the heuristic) can be addressed by empirical evidence. In fact, in spite of all of the caveats associated with generalizing from damaged to neurologically intact brains, the evidence suggests that the heuristic is anything but crude. Furthermore, the staunchest critics of two- visual systems models must acknowledge that these models have inspired several interesting, challenging, and productive lines of experimentation, whatever the precise veracity of the original ideas that drove them.

Much of the most compelling evidence cited in support of the Milner and Goodale formulation comes from patients with optic ataxia and from one well-studied patient, D.F., who exhibited visual form agnosia. In effect, numerous experiments with D.F. suggested that her perceptual systems were severely compromised by carbon monoxide poisoning, such that she could no longer read (alexia), recognize familiar faces (prosopagnosia), or identify common objects (visual agnosia). Her variant of this latter disorder extended to making discriminations among simple rectangles of equal areas but different shapes, the so-called *visual form agnosia* (Benson & Greenberg, 1969). In 1991, we (Goodale, Milner, Jakobson, & Carey) described how, despite serious difficulties with size judgments and with judgments of orientation of a slot that could be rotated in the picture plane, D.F. demonstrated movements that were still completely sensitive to these visual attributes. When working with the rectangles that were equal in area but different in aspect ratio, although D.F. could not tell them apart perceptually, her maximum grip aperture was perfectly scaled to the size of the object while still on the move to grasp it. Similarly, when we required D.F. to use a handheld card to show us the orientation of a slot in a picture plane that we rotated from trial to trial, she performed extremely poorly. However, if instead we asked her to post her card in the slot as she would a letter in a postbox,[1] she rotated the card to match the orientation well before the card made contact with the slot (see figure 14.2). These examples show how D.F could use visual

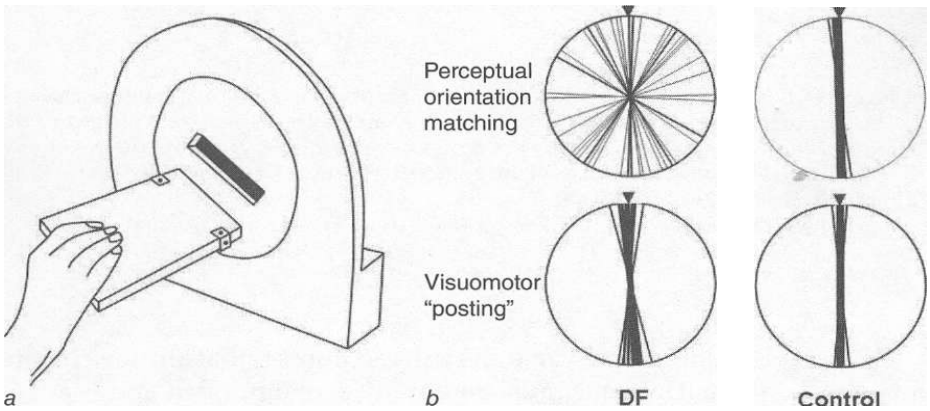


Figure 14.2 The perception-action dissociation in D.F., a visual agnosic patient, (a) The stimulus, which was a handheld card, and a sample visuomotor posting trial. For perceptual orientation matching, participants rotated the handheld card to match the slot's orientation. (b) D.F.'s performance versus a control performance on the two tasks, with correct performance normalized to the vertical (indicated by the arrow).
Adapted, by permission, from A.D. Milner and M.A. Goodale, 1998, "Visual brain in action," *Psyche* 4(12). Available: http://psyche.cs.monash.edu.au/v4/psyche-4-12-milner.html.

attributes such as orientation and size to control her hand actions and yet, when asked to show us what she perceived, could not function normally.

Some critics of the two-visual systems account of Milner and Goodale have suggested that the model is overly reliant on data from the single case of D.F. (e.g., Ungerleider & Haxby, 1994). Of course, if D.F. is a valid instance of a patient with a compromised ventral stream,[2] then evidence for the double dissociation with deficits and spared abilities of patients with compromised dorsal streams is logically possible. In a nutshell, double dissociations show that task A isn't failed in a single case patient because it is more difficult than task B (which patient A can do), because a different patient passes task A and fails task B (see Dunn & Kirsner, 2003).

Double Dissociations in Perception and Action

The first claim for a double dissociation between impairments after lesions to the dorsal and ventral streams appeared in 1994. Goodale and colleagues (Goodale, et al., 1994) contrasted the ability of D.F. (compromised ventral stream, intact dorsal stream) to reach and grasp irregularly shaped pebbles (see figure 14.3 for examples) with that of patient R.V. (originally described in Jakobson, Archibald, Carey & Goodale, 1991) experienced optic ataxia as part of Balint-Holmes syndrome. Optic ataxia is a disorder of reaching to visual targets that cannot be explained by low-level visual deficits. It is thought to be a consequence of damage in the superior parietal lobe (De Renzi, 1982; Husain & Stein, 1988; Ratcliff & Davies Jones, 1972; Rondot, De Recondo, & Ribadeau Dumas, 1977; although see Karnath & Perenin, 2005). The parietal lobe regions damaged in patients with optic ataxia include several important dorsal stream areas implicated in the control of eye and hand movements. R.V. had a compromised dorsal stream but a largely intact ventral *st*ream.

Goodale and colleagues (Goodale et al., 1994) found that D.F. was extremely poor at making same-different judgments (under conditions of free viewing) about irregularly-shaped "pebble" stimuli (see figure 14.3), while R.V. was virtually flawless at this task. In other words, R.V.'s visuoperceptual performance was relatively intact, while D.F.'s was not. The opposite pattern was obtained when the participants were required to grasp the pebble stimuli between their index fingers and thumbs. Figure 14.3 is a schematic representation of the performance of R.V. and D.F. in terms of selected grasp points (as depicted by grasp lines that show the orientation of the index finger and thumb before contact). As can be seen, D.F.'s grasp

[1]The James, Culham, Humphrey, Milner, and Goodale (2003) fMRI study of D.F. suggests that some portionsf her ventral stream are intact, more than a decade after her accident. The most extreme structural damage is to the lateral occipital cortex (area LO), which recent functional image studies (e.g., Kourtzi, Erb, Grodd,1 Biilthoff, 2003) have implicated as being crucial for perceptual encoding of shape.

points tended to intersect the pebble's center of mass,[1] a finding that suggests that her dorsal stream was perfectly capable of generating effective grasps using visual information that could not be used for perceptual judgements (Goodale et al., 1994).
The second type of double dissociation between optic ataxia and visual agnosia that supports the two-visual streams model depends on a theoreti-

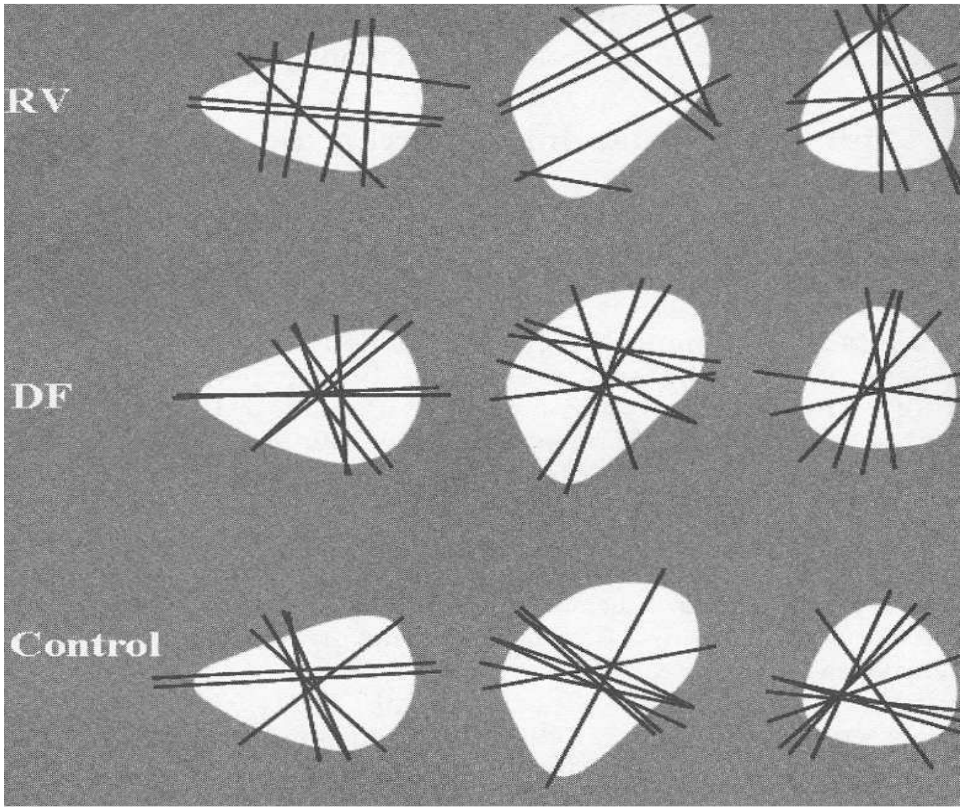


Figure 14.3 Grasp lines preceding object contact by D.F., who demonstrates visual agnosia; R.V., who demonstrates optic ataxia; and a matched control. Grasp lines of the control and D.F. tend to intersect near the center of mass of each object. While R.V. had no difficulty making same-different judgments about these objects, she clearly did not pick them up normally. On the contrary, D.F.'s grasps were indistinguishable from the control's, in spite of her difficulty in telling any two pebbles apart.

[1]Monika Harvey, David Milner, and I have argued elsewhere that using the principal axis to guide grasping (i.e., grasping the perpendicular axis) would produce roughly the same results, at least with these types of stimuli (Carey, Harvey, & Milner, 1996).

cal property of the dorsal stream: Its default modus operandi is to work in real time with visual information for the guidance of eye, head, and hand movements. In D.F., for example, sensitivity to both target size (her grip scaling was eliminated; Goodale, Jakobson, & Keillor, 1994) and location (her pointing accuracy dropped appreciably; Milner, Dijkerman, & Carey, 1998) was dramatically reduced when we required her to move after a delay. Paradoxically, these same delays may improve sensorimotor responses for patients with optic ataxia.

Milner and colleagues (Milner, Paulignan, Dijkerman, Michel, & Jean- nerod, 1999) investigated the effects of delay on sensorimotor responses of patients who misreach due to optic ataxia. In one study, patient A.T., who experienced bilateral parietal lobe damage 12 years previously (Jeannerod, Decety, & Michel, 1994) was required to point to peripheral targets immediately or 5 s after they were extinguished. Five control subjects performed somewhat worse in the delayed condition; A.T. performed substantially better. In a second study, patient I.G. (who also demonstrated optic ataxia) was tested on her ability to scale the size of her grip to different targets in immediate conditions versus a delay of 5 s, which required a pantomimed grasp (as used by Goodale and colleagues with D.F.) or a delayed real grasp. Like A.T., I.G. demonstrated an improvement after delay. The authors also found that I.G. was better when she pantomimed grasps than when she had to wait 5 s and then grasp (delayed real grasping). During delayed real grasping, presumably some dorsal stream impairments attenuated the good skills that could be driven by ventral stream mechanisms. Additional evidence for this interpretation is that I.G. tended to scale her grip to the originally presented target when, in delayed real grasping trials, the target was replaced with an object of a different size. Control participants never did this (Milner, Dijkerman, Mcintosh, Rossetti, & Pisella, 2003). The authors concluded that A.T. and I.G. could rely on a representation of target position or size mediated by the ventral stream to guide their actions after the compromised dorsal system response was not utilized. D.F., the previously described patient with visual agnosia, had no such representation to fall back on to guide her motor responses. Therefore, her motor skills were substantially compromised by even relatively short delays (Goodale, Jakobson, & Keillor, 1994). This line of research was reviewed by Milner and colleagues in 2003 (Milner, Dijkerman, Mcintosh, Pisella, & Rossetti, 2003).

Later Controversies: Diagnosing Optic Ataxia

The use of double dissociation between perception and action in optic ataxia and visual agnosia to support two-visual systems models is not without controversy. First, diagnosis in neuropsychology is not based exclusively on a particular neurochemistry, neuropathology, genetic marker, or other

incontrovertible piece of hard medical evidence. The diagnosis of visual agnosia, and more specifically visual form agnosia, can depend on interpretation of the magnitude of recognition dysfunction juxtaposed with the degree of associated low-level visual disturbance. How poor does a patient have to be at recognizing Snodgrass and Vanderwart line drawings (a standard set of drawings whose psychometric properties are well described; Snodgrass & Vanderwart, 1980) before the diagnosis is made? If patients fail low-level visual subtests of standardized instruments such as the Visual Object and Space Perception Battery (VOSPB; Warrington & James, 1991) or the Birmingham Object Recognition Battery (BORB; Riddoch & Humphreys, 1993), could we reasonably expect them to be capable of normal object recognition? If not, visual agnosia should not be diagnosed.

For optic ataxia, how exaggerated does misreaching have to be (particularly in peripheral vision) before we can be confident in a diagnosis? Many of the studies examining this disorder, with only a few recent exceptions, include no control subjects matched in age or visual status. When control subjects are included, the control sample is small (and there are some serious statistical issues as a consequence; see Crawford & Garthwaite, 2005) and the selection mechanism is unspecified—often the controls are highly functioning individuals from a university community who might be not be comparable with patients with stroke, even when matched for age and sex. In some cases hand- or field-specific deficits (which of course must be consistent with lesion laterality) may allow patients to serve as control participants for themselves. However, these studies usually only describe the relatively intact performance in the unaffected arm or field—they don't typically measure it.

A second caveat for experimental work on patients with optic ataxia is that the most florid form, in which misreaching is observed even for fove- ated targets, occurs as part of Balint-Holmes syndrome, a complex disorder with at least a triad of symptoms. These other symptoms, including atten- tional and gaze difficulties, could be indirectly responsible for the misreaching, or the ataxia could be an independent symptom that depends on a different component of the large bilateral occipitoparietal lesions. Accurate eye tracking in the elderly is difficult, and when it has been done, details on calibration (which requires accurate eye movement and good control of fixation), recalibration frequency, system accuracy, and drift correction are suspiciously absent from the method sections of the reports.

The third caveat regarding optic ataxia is a critical but typically omitted demonstration: The misreaching deficits in patients who have been diagnosed with optic ataxia by definition are restricted to deficits in moving to visual targets. As I have argued elsewhere (Carey, 2004), auditory and proprioceptive guidance of movement are rarely assessed in these patients in any formal way For example, magnetic misreaching (a very rare disorder in which patients slavishly reach to the fixation point rather than an

extrafoveal target; Carey, Coleman, & Delia Sala, 1997; see also Buxbaum & Coslett, 1997,1998; Jackson, Newport, Mort, & Husain, 2005; van Donkelaar & Adams, 2005) has been labelled as a variant of optic ataxia by Buxbaum and Coslett as well as Jackson and colleagues, but we have evidence that, in our patient at least, poor guidance of limb movement is not restricted to the visual modality. Blanghero andcolleagues (2007) have made similar observations in two patients previously described in the literature as demonstrating optic ataxia.

The fourth caveat relates to the distinction between foveal and nonfoveal variants of optic ataxia, which is becoming blurred or even ignored (perhaps because the former are so rare, it is becoming convenient to discuss the extrafoveal variant as the prima facie case). Misreaching in the full-blown foveal variant is so fascinating because the errors happen in spite of accurate gaze, which also in a sense serves as a crude control of visual status: The patient sees the target well enough to fixate on it. The same cannot always be said when targets are presented well into the visual periphery, which is one of the reasons why I worry about the visual status of control participants. Of course, conducting clinical experiments with the more common extrafoveal cases means ensuring fixation compliance, with all of the problems related to monitoring eye position briefly outlined earlier.

How do these concerns constrain the usefulness of any particular patient for evaluating cortical models of perception-action dissociation? I have little doubt that, in spite of her diffuse brain damage, D.F.'s recognition problems are restricted to the visual domain (for example, I tested her haptic recognition abilities in the 1990s and they were effectively normal). In spite of several attempts, other patients with a similar agnosia and perception-action profile have been difficult to find. A patient who had experienced meningo-encephalitis in childhood and subsequently demonstrated relatively intact visuomotor skills in conjunction with poor visuoperceptual abilities has been described (Le et al., 2002). Nevertheless, he differs from D.F. in several important respects, including visuoperceptual matching skills and a lifetime of development that was not afforded to D.F., who acquired her disabilities in adulthood. Similarly, a few patients with agnosia have shown, similarly to D.F., dependence on binocular vision for grasping, although these people do not share many features of D.F.'s unusual profile. Recently Yang, Wu, and Shen (2006) described an anoxic patient who may have demonstrated a similar perception-action profile, but that report focussed on implicit processes related to shape and not sensorimotor skills per se. Goodale, Wolf, and others (Goodale et al., 2008; Wolf et al., 2008) have recently described a patient with visuoperceptual problems even more severe than those of D.F., who has intact grasping. Very recently, Karnath, Riiter, Mandler, and Himmelbach (2009) have found a patient with a ventral stream lesion who shows a very similar perception-action profile to D.F. using Efron square matching and grasping, as well as orientation posting and matching.

The difficulty in finding other patients with visual agnosia and such a pure dissociation has not hindered research inspired by the Milner and Goodale variant of the two-visual systems theory Much of the recent work in support of their model has come from studies of neurologically intact samples. Work on dissociations in perception and action has proven to be fruitful as well as contentious (see Bruno, 2001; Carey, 2001; chapter 13 of this volume for reviews of dissociations in perceptual and action systems in response to visual illusions; although see Biegstraaten, de Grave, Smeets, & Brenner, 2007, for a contrary view). In parallel, claims about the rather short-term memory of sensorimotor systems (Goodale, et al., 1994) mediated by dorsal stream mechanisms have remained rather unchallenged.

One patient study (Himmelbach & Karnath, 2005) attempted to evaluate whether the effects of delay in patients represent a step function (the strongest support for a switch in processing mode from a sensorimotor representation to a more enduring, less metric perceptual representation) or a continuous drop (or improvement) in performance. These authors found some evidence for improvement with delay in two patients with optic ataxia, although this effect was relatively continuous and therefore did not suggest a step change indicative of a switch from impaired dorsal representation to intact perceptual representation. However, the two cases described in this study are not without controversy. First, no details beyond mean age were provided on the elderly control participants. Given the acuteness of the patients, a nonataxic control group with similar visual status or neglect might have been appropriate. Second, one of the patients (U.S.) had profound right-sided neglect, but we were not told of her error rates in right and left sides of space. In addition, monitoring fixation in these patients must have been extremely difficult (how was the infrared eye tracker calibrated in patients with potential difficulties in eye movement and hemispatial neglect?). Third, these cases were tested in a very acute phase of the patients' illness (6 and 8 d poststroke). In neuropsychological syndromes such as hemispatial neglect, patients with left-brain damage can show large right neglect (e.g., De Renzi, 1982) that often resolves very quickly and is made of very little by researchers. Perhaps optic ataxia, like neglect after left-brain lesions, is easier to diagnose in acute patients (although Himmelbach & Karnath do not say so explicitly). If this is the case, a more specific control group is probably more appropriate.

Optic ataxia and delay have also been linked in experiments by Schndler and coworkers (2004) and Rice and colleagues (2008). In the first study, Schindler and coworkers found that two patients with optic ataxia were impaired in adjusting their reach trajectories when two obstacles were moved in the work space from trial to trial. In the follow-up study, Rice and colleagues (2008) showed that introducing a delay between stimulus presentation and response abolished this failure. In other words, the patients showed trajectories that were appropriate for the obstacle locations in the

same manner as control participants did in immediate and delayed conditions. These effects represent a third example of how optic ataxic patients behave in ways consistent with the Milner and Goodale account.

Recently colleagues of David Milner have changed their tune somewhat by criticizing the use of patients with optic ataxia as the appropriate counter to patient D.F. (Pisella, Binkofski, Lasek, Toni, & Rossetti, 2006). One of their claims is that the deficits studied in peripheral vision in the optic ataxia cases are not strictly comparable to D.F., who performs with full foveal vision in conditions where perception and action responses are contrasted with one another. The critique is not strictly true of course: D.F. shows similar perception-action dissociations when forced to work with stimuli in peripheral vision. We have shown that D.F. reaches to target positions in peripheral vision with accuracy similar to that of controls (although interposing short delays before the response seriously impairs this skill; Milner, Dijkerman, & Carey 1998). Figure 14.4 (from Milner & Goodale, 2008) shows data contrasting the performance of D.F. with that of A.T. (data from Milner et al., 1999). And there are examples of patients with optic ataxia operating in central vision who show the perception-action distinction, such as V.K., who we described in 1991 (Jakobson, Archibald, Carey, & Goodale, 1991).

A final study worthy of note questions the assumptions that the superior parietal lobule is the crucial site of the lesion that produces optic ataxia in humans (Karnath & Perenin, 2005). The authors of this study used a lesion overlap method (Rorden & Brett, 2001) with a large sample of patients screened for optic ataxia and found that the crucial regions of overlap did

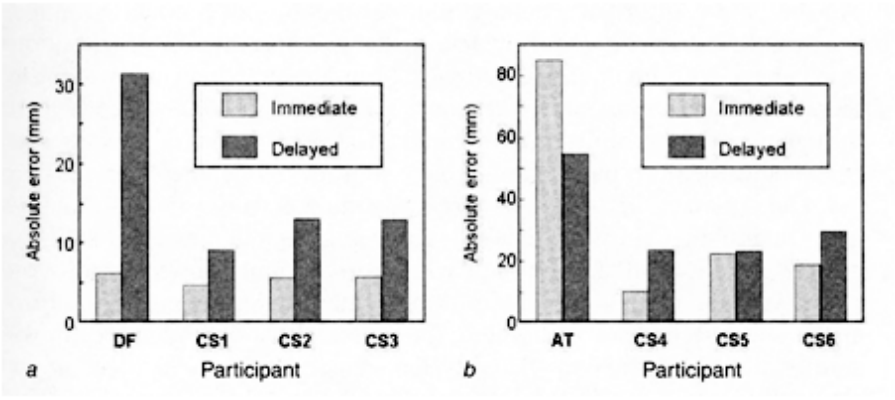


Figure 14.4 Reaching into the periphery in *(a)* D.F., a patient with agnosia (from Milner, Dijkerman, & Carey, 1998), and *(b)* A.T., a patient with optic ataxia (from Milner et al., 1999). Note that worse performance is higher on the y-axis (absolute error = unsigned error of end point from actual target position). The scales have been modified to show the patterns from the two separate studies.

not center on the superior parietal lobule, as suggested by early studies (e.g., Ratcliff & Davies-Jones, 1972) and the Milner and Goodale account. Instead, they found that lesion overlap centered on a region around the border of the inferior parietal lobe with occipital cortex. The precise anatomy of optic ataxia is not crucial for the Milner and Goodale model in a functional heuristic sense, but there are suggestions in the literature from neuroimaging and patient work that this region may be utilized in nonperceptual operations. This controversy warrants additional research.

Summary and Future Directions

In spite of these unresolved issues, the identification of several patients with extrafoveal optic ataxia, such as R.V., A.T., and I.G., and the first round of experiments inspired by the two-visual systems model of Milner and Goodale have reintroduced this category of dysfunction to contemporary neuroscience. For example, neurophysiological models of eye-hand transformations, inspired by data from single-unit studies in nonhuman primates (e.g., Andersen, Snyder, Batista, Bueno, & Cohen, 1998; Chang, Dickinson, & Snyder, 2008), have inspired recent experiments on the nature of eye- hand coordination deficits in neurologically intact participants as well as in patients with optic ataxia. These latter studies may never have occurred if the two-visual systems debate on dorsal and ventral streams had not reintroduced this interesting type of patient to the mainstream literature.

For example, studies by Crawford, Khan, and colleagues (e.g., Khan et al., 2007) have supported the idea that hand movements may be coded in eye-centered coordinate schemes. Some of these models may be limited because they do not necessarily acknowledge a specific role of foveation in eye-hand coordination (and retinal coordinates will do in many of these hierarchical accounts; Carey, Ietswaart, & Delia Sala, 2002). Nevertheless, the eye-centered accounts have inspired additional studies of patients and neurologically intact participants from a sensorimotor control perspective (c.f., Dijkerman et al., 2006; Scherberger, Goodale, & Andersen, 2003;Verha- gen, Dijkerman, Grol, & Toni, 2008). These studies will inevitably suggest greater specifications for simple two-stream accounts. Indeed, the two- visual systems models may fade from the neuropsychological literature as the specifics become elaborated. Of course, the indebtedness of those studies of the future to two-visual systems theory will remain. Ideas about a monolithic multi-feature representation of the visual world (see Goodale, 1983) have gradually been laid to rest, thanks in no small part to the literature referred to in this chapter.

In conclusion, the patient work related to the two-visual systems account of Milner and Goodale has grown dramatically in the past 15 years or so since the model first came to the attention of neuroscientists. Although

contentious elements remain and some of the details have required minor rethinks (c.f., Milner & Goodale, 2008), for the most part the model has stood the test of time. What is of little debate is the amount of directed research that it has driven or at least inspired. The growing recognition of sensorimotor processes and their rightful place in cognitive neuroscience has been a long time coming.

VISION AND GOAL-DIRECTED MOVEMENT

Neurobehavioral Perspectives

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