

Motion Processing in Human Visual Cortex

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Acknowledgments. The authors thank David Heeger, Alex Huk, Nestor Matthews and Mark Nawrot for comments on sections of this chapter. David Bloom helped greatly with manuscript formatting and references. During preparation of this chapter RB and EG were supported by research grants from the National Institutes of Health (EY07760) and the National Science Foundation (BCS0121962) and by a Core Grant from the National Institutes of Health (EY01826) awarded to the Vanderbilt Vision Research Center.

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During the past few decades our understanding of the neural bases of visual perception and visually guided actions has advanced greatly; the chapters in this volume amply document these advances. It is fair to say, however, that no area of visual neuroscience has enjoyed greater progress than the physiology of visual motion perception. Publications dealing with visual motion perception and its neural bases would fill volumes, and the topic remains central within the visual neuroscience literature even today.

This chapter aims to provide an overview of brain mechanisms underlying human motion perception. The chapter comprises four sections. We begin with a short review of the motion pathways in the primate visual system, emphasizing work from single-cell recordings in Old World monkeys; more extensive reviews of this literature appear elsewhere.^{1,2} With this backdrop in place, the next three sections concentrate on striate and extra-striate areas in the human brain that are implicated in the perception of motion. These sections draw on experimental work from brain imaging studies, from studies employing transcranial magnetic stimulation, and from psychophysical studies in patients with localized brain damage. These three methodologies have generated complementary characterizations of multiple neural mechanisms underlying different aspects of human motion perception: perception of translational motion (including direction and speed), perception of 3D shape from motion, perception of heading from optic flow and perception of biological activity. To isolate these different aspects of motion perception, researchers have utilized a variety of different motion stimuli, each designed to challenge the visual system in some characteristic, revealing fashion; examples of some of those stimuli are illustrated schematically in Figure 1.

Insert Figure 1 about here

Because of space limitations, our chapter will not cover the extensive psychophysical literature on motion perception, except for selected phenomena that are related directly to brain activation studies or lesion studies. For up-to-date reviews of the psychophysical literature, readers may consult either of two recent chapters dealing with human motion perception.^{3,4} Nor will we deal with visual motion processing as it applies to the generation and control of eye

movements; for work on this topic, readers are referred to other sources (for example, Horowitz & Newsome⁵). Also omitted from coverage are studies of motion processing that use visual evoked responses⁶ or magnetoencephalography⁷ as the dependent variable. Finally, our coverage of deficits in motion perception will be limited to neuropsychological studies of patients with restricted brain lesions — we will not cover the important and potentially revealing literature on deficits in motion perception that can accompany psychopathology,⁸ reading disorders,⁹ amblyopia,¹⁰ Alzheimer’s disease,¹¹ and developmental disorders including William’s syndrome¹² and autism.¹³

Visual Motion Pathways in Non-Human Primate

It is generally agreed that explicit analysis of visual motion is inaugurated in the primary visual cortex. While it is true that cells in the retina and in the thalamus respond vigorously to moving contours, spots of light and gratings, those responses are *not* selective for the direction or the speed of motion and, hence, those cells are not representing anything about velocity (a fundamental value when specifying information about motion). The foundations for motion processing in the primate visual system are the direction-selective (DS) cells first encountered in primary visual cortex, here after termed V1. First described by Hubel and Wiesel¹⁴ in the anesthetized monkey, DS cells in V1 are characterized by their vigorous response to stimulus motion in a given direction, with other directions of motion giving little or no responseⁱ. It is estimated that about one-third of cells in primary visual cortex of the macaque monkey fall in the DS category,¹⁸ and these cells tend to congregate in the upper portions of cortical layer 4 (which receives strong input from the magnocellular layers of the lateral geniculate nucleus) and in layer

ⁱThe idea of individual brain cells responsive to given directions of motion can be traced to Reichardt’s seminal chapter¹⁵ in which he employs simple logic devices to construct a circuit for registering the presence of motion in a given direction. For that matter, an even earlier version of a “motion detector” can be found in the monograph by Exner,¹⁶ who proposed a simple neural network involving comparisons of successive stimulation arising at different points of the retina at different points in time. (See Smith and Snowden¹⁷ for a more detailed explication of this earliest of neural models of motion detection.)

6.¹⁹ Within large samples of DS cells there is no obvious bias toward a given direction of motion, and the breadth of directional tuning varies considerably among cells. DS cells are also found in the superior colliculus of the primate visual system, but here their responses depend importantly on whether moving stimuli are presented against a background of stationary contours or against a background in which contours move in a direction different from that of the ‘target’ stimulus — the latter condition produces activity in many more superior colliculus cells than does the former.²⁰

While varying in the details of their behavior, DS cells in V1 have in common a receptive field structure that allows them to register the presence of a pattern moving through the receptive field. These cells can be modeled as spatio-temporal filters that extract motion energy within local regions of the retinal image.^{21,22} One useful way to portray a DS cell’s receptive field is by depicting its preferred stimulus in a plot of the variation of that stimulus in spatial position over time. Portrayed in this way, it is easy to see that DS cells respond to events embodying visual energy oriented in space/time. And just as one can model spatial orientation selectivity based on the interplay between excitatory and inhibitory regions arrayed spatially, one can model direction selectivity as the product of an interplay between excitation and inhibition arrayed in space and time.

Much more could be written about DS cells, including their vulnerability to early visual deprivation, their selectivity for spatial frequency and their relative insensitivity to the wavelength composition of moving stimulus. For our purposes, however, we will concentrate on an inherent limitation in the responses of DS cells, a limitation arising from the “local” nature of the motion extraction performed by DS cells. It is important to understand this limitation and the subsequent computational steps needed to overcome it, for this understanding sets the stage for appreciating the neural operations performed in extrastriate visual areas.

As a point of departure for considering this limitation of DS cells, we can construe their receptive fields as spatially restricted “windows” within which motion is “measured.” When an extended contour, such as a bar, moves through that window, the DS cell’s receptive field may

be too small to “see” the ends of the bar and, hence, the cell cannot unambiguously respond to the bar’s direction of motion independent of its orientation. Instead, the cell will respond as though the contour were moving perpendicular to the orientation imaged within the cell’s receptive field.²³ Thus, for example, a vertical bar moving smoothly up and to the right may traverse a series of small DS receptive fields over time. Those cells, however, can only signal the motion component perpendicular to the bar’s orientation which, in the case of a vertical bar, means the rightward motion; the bar’s upward component of motion produces no measurable change within the receptive fields of those cells. In other words, the DS cells “see” only what transpires within their local receptive fields, which means that their responses are inherently ambiguous with respect to the actual direction of motion of an extended contour or border. Think about it this way: for any given DS receptive field there exists a family of directions of motion that could generate the same spatio-temporal luminance changes within the cell’s local receptive field and, hence, generate the same response in the cell. How, then, can the actual direction of motion be recovered from these ambiguous responses?

For reasons just explained, the local signals generated by V1 DS cells must be processed further by subsequent stages of the nervous system, where overall, global direction of flow specified by local, overlapping motion can be extracted. And it is widely believed that this second stage of processing transpires within the brain area known as MT (for “middle temporal” area). Some of the DS cells in V1 project to the so-called “thick” stripes in V2, and from here projections innervate MT, or V5 as it is sometimes termed. Other DS cells in V1 project to area V3 and then on to MT,²⁴ while still other DS cells in V1 project directly to MT.²⁵ Moreover, a fraction of MT cells can be activated even when area V1 has been surgically removed, implicating subcortical inputs to this extrastriate area.²⁶

Middle Temporal Area

In the monkey, the middle temporal gyrus (MT) is a visual area near the parietal-occipital juncture. Similar to primary visual cortex, MT contains a retinotopically organized

representation of visual space, and the receptive field sizes of cells increase with eccentricity.²⁷ It is estimated that the latency between photoreceptor stimulation and MT activation may be as brief as 20-25 msec,²⁸ which befits MT's role in registering information about events that may require immediate behavioral reaction. In MT, unlike V1, essentially all neurons are direction-selective,²⁹ with this receptive field property being topographically organized in a columnar system.³⁰ The distribution of preferred directions is isotropic among a large sample of cells,³¹ which dovetails with the uniform sensitivity to all directions of motion found psychophysically in humans.^{32,33}

Several lines of evidence imply that MT plays an important role in motion perception. For one thing, some MT neurons are as sensitive to weak motion in random dot cinematograms (RDC – see Figure 1C) as is the alert, behaving monkey from whom MT recordings are being made – there exists a close correspondence between cell firing rates and psychophysical performance.^{34,35} For another, microstimulation applied to MT neurons can bias the perceived direction of motion reported by the animal.³⁶

But why is MT thought to embody the second-stage process discussed above, i.e., the stage that disambiguates direction of motion? The answer is because a substantial fraction of MT neurons respond to a given direction of motion independently of the orientation of the moving stimulus pattern.²⁵ These cells behave as if their receptive fields are combining multiple, appropriate outputs from V1 to provide an explicit representation of velocity disambiguated from contour orientation. The discovery of such cells represents a remarkable advance in our understanding of motion computations, and these findings have had a marked influence on computational modeling of human motion perception.³⁷⁻³⁹

Besides their sensitivity to motion, some MT cells are also disparity selective, i.e., selectively responsive to the depth plane in which motion appears.⁴⁰ Moreover, activity in this class of cells fluctuates coincident with reversals in the perceived depth ordering of motion-defined surfaces,⁴¹ as predicted by Nawrot and Blake's⁴² neural model of the kinetic depth effect. There is also evidence that activity evoked by stimulation within MT neurons' receptive fields

can be modulated by stimulation falling outside those ‘classic receptive field’ ,i.e., within areas of the visual field where stimulation on its own does not evoke neural responses.⁴³ This response property could provide a mechanism for registering motion-defined boundaries within complex optic flow fields. Finally, it has been shown that MT responses can be modulated by the attentional state of the animal.⁴⁴

Closely allied with MT is a companion area, the medial superior temporal area (MST), that receives input from MT. Neurons in area MST have even larger receptive fields organized in ways that endow them with selectivity for complex optic flow such as expansion or contraction (e.g., Saito et al⁴⁵). MST neurons are also sensitive to non-retinal information about eye movements.⁴⁶ This information could be especially important to an organism that is trying to extract its direction of heading from optic flow, while its eyes are moving about.⁴⁷ In fact, Bradley et al⁴⁸ demonstrated that neurons in the dorsal part of MST behave in just the manner needed to accomplish this extraction: a large fraction of dorsal MST neurons responded to optic flow centered at a given region of the visual field independent of the direction in which the eyes were pointed. These cells, in other words, can signal the direction in which the animal was headed even as it turned its eyes from side to side while maintaining a constant direction of self-movement.

Superior Temporal Area

Anatomically situated just anterior to MT and its satellites on the superior temporal sulcus (STS) of the monkey brain is the anterior superior temporal polysensory region (STPa); for a review on the debate regarding this region’s exact location, see Cusick.⁴⁹ Unlike cells in MT+, cells in STPa are characterized by their multi-modal responses. This region receives inputs from visual, auditory and somatosensory cortices, but the most dominant modality is vision.⁵⁰ Receptive fields of the visually responsive neurons usually cover the entire visual field, and nearly all cells prefer moving stimulation over stationary stimulation.⁵¹ While most cells have no size or shape preferences for the object in motion and half have no preference for

direction of motion, many STPa cells prefer particular types of motion such a movement in depth, radial motion or optic flow.⁵² Generally STPa cells respond well to smooth motion over a range of speeds, but many prefer jerky movements and respond maximally at the onset of each discrete step.⁵¹

Cells in STPa, which appear to respond indiscriminately to form but very selectively to movement, have been implicated in perception of body movement. Hietanen and Perrett⁵³ measured responses of cells in the dorsal bank of the anterior superior temporal sulcus (STS) of the macaque brain, the region corresponding to STPa. They found a majority of the cells to be sensitive to viewing external body movements; only a few cells responded when the animal viewed movements of it's own body. Others have found cells in this area that respond selectively to a human form that is walking, rotating, moving in and out of occlusion and flexing the limbs.⁵⁴ Oram and Perrett⁵⁵ found a small population of cells (about one third of all cells sampled) in the upper bank of STPa that responded to whole body movement (such as walking) portrayed in animations consisting of a dozen or so "points" of light attached to the major joints of the body (i.e., the "biological motion" animation technique illustrated schematically in Figure 1F). Many of these cells were selective for a given direction of movement implied by the gait of the biological figure, even when there was no net translational motion because the figure was walking in place (as if on a treadmill). Most of the cells did not respond well to "jumbled motion" consisting of the same component motion vectors as biological motion but scrambled to disrupt their spatial coherence. Also found were cells that responded when the biological motion depicted some form of articulation (e.g., bending of the arm). Oram and Perrett noted that cells with similar response selectivity (such as a preference for head rotations towards the animal) tended to be found in close proximity to each other, with these cells clustered according to motion and viewpoint preference.

With this overview of the non-human, primate visual system, we now are ready to turn to three categories of research studies whose results bear on the issue of brain mechanisms underlying human motion perception. As will become evident, the stimulation conditions used

in these studies have been guided by the monkey physiology just reviewed.

Human Brain Areas Responsive to Motion Revealed by Brain Imaging

Based on functional magnetic resonance imaging (fMRI), it is estimated that the human brain contains as many as 17 anatomically distinct areas that can be activated by visual motion of one sort or another.⁵⁶ Figure 2 illustrates the approximate locations of most of the motion-responsive areas that we will focus on in this section. Unless otherwise noted, results described here were obtained from normal adult humans in whom brain activity was inferred either from metabolic maps obtained from positron emission tomography (PET) or from the blood oxygen level-dependent (BOLD) signals recorded while the observers viewed visual animation sequences inside the bore of a magnetic resonance image scanner (fMRI); details of the scanning procedures (field strength, sequence type, image plans, spatial resolution, concurrent task) are not included in the discussion unless specifically relevant for a given finding.

Insert Figure 2 about here

Visual Area V1

In the human brain, V1 can be readily identified using well established retinotopic mapping procedures that clearly delineate the boundaries and layout of this primary sensory cortex located on the medial bank of the calcarine fissure.⁵⁷ Targeting this region of interest, it is well known that strong, reliable signals can be evoked by a variety of forms of dynamic stimulation, including flickering or drifting gratings (Figure 1A), rotating wedges, expanding and contracting rings (including “second-order” rings defined exclusively by contrast modulation similar to that illustrated in Figure 1B), moving dots (Figure 1C) and flashing lights. These responses to dynamic stimulation, however, are not unequivocal evidence for explicit representation of motion information in the activity of DS neurons. Indeed, V1’s indiscriminate responses to a wide variety of dynamic visual stimuli can all be accounted for by the amount of

spatio-temporal energy in the display sequence, irrespective of the motion quality *per se*.^{58,59} So, to obtain unequivocal evidence for DS neurons in V1, one must employ strategies for producing BOLD response attributable to DS cells. One such strategy, described in the next several paragraphs, was developed by Heeger and colleagues.⁶⁰

A hallmark property of nearly all models of motion processing is “motion opponency.” In a neural network exhibiting motion opponency, DS cells with opposing preferred directions of motion suppress one another’s firing rate, the aim being to generate a net motion signal for given local regions of the visual field. Thus when opposite directions of motion of equal strength are present simultaneously at a given location in the visual field, motion opponency produces equal, relatively low levels of activity within DS cells tuned to those two directions. But when one direction of motion is stronger than the other, the balance of activity within the opponent DS cells tips in favor of those cells whose preferred direction corresponds to the stronger motion stimulus.

Now, there is good psychophysical evidence for the existence of motion opponency in human vision,⁶¹ and Heeger and colleagues used fMRI to identify specific brain areas exhibiting BOLD responses consistent with motion opponency. In the condition we shall focus on, observers viewed two different motion sequences that alternated over time: a single grating drifting in a given direction interleaved with presentation of two identical gratings drifting in opposite directions (“counterphase” grating). In V1 the fMRI BOLD signal increased during presentation of the counterphase grating and decreased during presentation of the single direction of motion. This outcome implies that the two opposite directions of motion produced greater neural activity than did either grating on its own; there was not strong evidence for motion opponency, in other words. The opposite pattern of BOLD signal responses was found in MT: counterphase gratings produced weaker responses than did single moving gratings, implying strong motion opponency.

From that study it would be premature to conclude that DS cells are not present in human V1, for the interpretation of the findings depends on assumptions about the nature of the

inhibitory interactions among DS cells. A more direct brain imaging test for DS cells in V1 was recently performed by the Heeger laboratory using an adaptation procedure which will be described in more detail in the next section. In brief, the procedure produces estimates of the strength of direction-selective adaptation by comparing (1) BOLD responses to repetitive stimulation with visual motion in a single direction to (2) BOLD responses to repetitive stimulation by visual motion that changes direction from moment to moment. The extent to which BOLD signals are smaller in the former case provides an index of the extent of DS processing in a region of interest. Using this technique, Huk et al⁶² found evidence for relatively weak but reliable direction-selective adaptation in V1, reminiscent of the relatively modest proportion of DS cells in V1.

Before moving to subsequent, extrastriate cortical areas, we should say a word about V1's response to apparent motion of an illusory figure created by rotating the sectors in an array of sectorized disks ("pacmen") forming an illusory square – appropriately sequenced, this kind of animation can create the vivid impression that the illusory square is moving back and forth over a set of black disks.⁶³ When observers view these sequences in the fMRI scanner, robust activation is evoked in V1 as well as in other visual areas including MT.⁶⁴ This does not mean, however, that V1 directly computes motion from illusory contours, for the V1 responses could reflect feedback from extrastriate areas. (For a discussion of the interpretation of V1 BOLD signals, see Logothetis et al.⁶⁵)

Visual Area 3A

A portion of the output from V1 destined for dorsal portions of the brain, in the parietal lobe, innervates a retinotopically organized brain region located near the junction of the transverse occipital sulcus and the intraparietal sulcus (see Figure 2). This dorsal extrastriate area, called V3A, was shown by Tootell et al⁶⁶ to be motion sensitive. In fact, subsequent studies have shown that V3A exhibits the strongest direction selectivity of any of the retinotopically defined areas,⁶² and it responds much more strongly to coherent motion of random dots than to

incoherent, random motion.⁵⁸ Culham et al⁶⁷ discuss possible homologies between human V3A and monkey visual area V3.

Visual Area MT+

Everyone agrees that the human analog to monkey MT is located within a relatively large expanse of tissue anterior to early, retinotopically organized visual areas. This putative MT is situated posterior and inferior to the superior temporal sulcus on the ascending branch of the inferior temporal sulcus and lateral temporal sulcus.⁶⁷⁻⁷⁰ In neuroimaging studies, human MT is most commonly isolated by comparing activity levels while observers view a moving pattern (often, moving dots) to activity levels while observers view a stationary pattern or one in which stationary elements flicker on and off (Braddick et al;⁷¹ but see Orban et al⁷²). Simply comparing moving to stationary conditions, however, lacks the specificity needed to distinguish MT and its surrounding satellite areas, and so the entire motion responsive region in humans is often referred to as MT+; in some publications, this brain area is identified as V5.

BOLD signal levels from MT+, unlike signals from any other motion responsive area, are linearly proportional to the degree of motion coherence present in random-dot cinematograms evoking those BOLD signals (Rees et al;⁷³ but see McKeefry et al⁷⁴ for conflicting results obtained using PET); with a few simplifying assumptions, this linear relation between BOLD signal and motion coherence can be nicely modeled based on population responses from single neurons in area MT of alert, behaving monkeys.⁷⁵ When activated using gratings varying in contrast, MT+ shows reliable responses to very low contrasts, below the threshold for evoking activation in V1; those MT+ responses, however, saturate at relatively low contrast values.^{70,76} Besides luminance defined gratings and RDCs, MT+ is also strongly activated by expanding and contracting rings whose borders are defined exclusively by contrast-modulation, which constitutes a form of second-order motion.⁵⁹ As an aside, no fMRI study has uncovered a brain area that responds exclusively to second-order motion (recall Figure 1B), although visual area V3 and its ventral counterpart VP respond more strongly to second-order motion than they do to

first-order motion.⁵⁹

There is disagreement concerning the extent to which MT+ responds selectively to more complex fields of motion depicting spatial structure (e.g., curvature) or motion-defined forms. Some laboratories report evidence for shape-specificity⁷⁷ while others find no differential responsiveness to simple translation vs structured flow fields (e.g., Reppas et al⁷⁸, Grossman et al⁷⁹). It remains to be learned whether these seemingly conflicting results arise because different laboratories are measuring BOLD signals from different subregions within the MT+ complex, subregions containing neurons that differ in their functional properties. At present we do not have MT localization techniques comparable in spatial precision to those used to identify boundaries between early visual areas V1, V2 and V3a.

Activity in MT+ varies in several interesting ways all of which point to the involvement of this region in the actual perception of motion. For one thing, signal levels from this area are reliably higher when observers genuinely experience motion and not just flicker. This result was found by Muckli et al⁸⁰ who performed an event-related study in which observers viewed repetitive, two-frame animation sequences which sometimes yielded the strong impression of apparent motion (AM) and other times an impression of on/off flicker with no hint of AM. During brain scanning, observers tracked transitions between these perceptual states, and the resulting BOLD signals were analyzed contingent on the observers' perceptual reports. Activation levels from MT+ covaried over time, with perceptual fluctuations being strongest when observers reported seeing motion and weakest when flicker was seen. In a related study, Sterzer et al⁸¹ utilized a bistable motion stimulus — a “spinning wheel” — whose apparent direction of rotation alternated irregularly over time between clockwise and counterclockwise. While in the scanner observers tracked these changes in direction of perceived rotation, which allowed the experimenters to identify brain regions in which activation coincided with switches in perceptual state. Sterzer and colleagues found reliable event-related activations in MT+, as well as in several brain regions in parietal and frontal areas; no activations coincident with perceptual fluctuations were found anywhere in temporal lobe areas generally thought to be

involved in object recognition. Based on these two studies, then, it seems clear that neural events underlying perceptual fluctuations when viewing bistable motion displays arise within the same brain region — MT+ — that responds reliably to physical changes in the motion characteristics of the retinal image.

Also consistent with the involvement of MT+ in the actual experience of motion, Blakemore et al⁸² found that simple animations implying causation produced greater activation in MT+ than did essentially the same animations in which the perception of causality was absent. While in the scanner, observers viewed brief animations in which a colored circle resembling a “ball” moved horizontally across the visual field. On some trials this ball appeared to collide with another, previously stationary ball, with the collision “launching” this second ball into motion (the “causal” event); on other trials, the initially moving ball appeared to move underneath the stationary ball without collision, thereby continuing its path of motion (“non-causal” event); these animations are similar to those devised by Michotte⁸³ in his classic work on perception of causality. In Blakemore et al’s study, the actual motion trajectories in both conditions were essentially identical, with only a change in the color of the balls controlling whether or not collision was perceived. Still, the “causal” event produced significantly greater MT+ activation than did the “non-causal” event, even on trials where attention was directed to the motion trajectory itself and not to the implied causality. Incidentally, this same pattern of activations was also observed in portions of the superior temporal sulcus (a brain region we shall discuss in the next subsection). Blakemore and colleagues interpret these results as evidence that sensory areas of the human brain registering the presence of visual motion are responsive to the causal relations implied by that motion.

Another strategy for studying the relation between MT+ activation and perception exploits the well-known motion aftereffect (MAE), wherein prolonged viewing of motion in one direction causes a subsequently viewed, stationary stimulus to appear to move in the opposite direction. Several laboratories have reported that BOLD signals recorded from MT+ are larger when observers view a stationary figure undergoing illusory motion, compared to when they

view the same stimulus after adaptation conditions that do not produce illusory motion.⁸⁴⁻⁸⁷ Moreover, several of these studies have found that the initially elevated BOLD signal associated with the MAE drops to its baseline level over the same period of time that the MAE itself dissipates (e.g., Culham et al⁸⁵). This temporary elevation in MT+ activation during MAE but not during control trials was interpreted as the neural concomitant of illusory motion. However, that conclusion was questioned by Huk et al,⁶² who showed that differential levels of attention during MAE and control trials contribute to the differences in BOLD signal differences between these two conditions. The compelling nature of illusory motion during the test phases, in other words, may have unwittingly boosted MT+ activation, a likely possibility in view of the well-established finding that explicit attentional manipulations modulate MT+ BOLD signals (e.g., Buchel et al⁸⁸).

Huk et al⁶² went on to show that the MAE could be attributable to temporary reductions in activity among neurons stimulated during the adaptation phase. To arrive at this conclusion, they compared BOLD signals measured in blocks of trials during which a single direction of motion was repeatedly presented (“same” direction trials) to BOLD signals measured in blocks of trials during which motion directions varied from presentation to presentation (“mixed” trials). During both trial types, observers performed a challenging speed discrimination task requiring sustained attention. MT+ signals were considerably smaller during “same” trials compared to “mixed” trials, presumably because repeated stimulation of DS cells tuned to a given direction of motion depressed their responsiveness when there was no opportunity for recovery, which the “mixed” trials on the other hand did afford. Based on these and other results, Huk et al concluded that the MAE results from a temporary imbalance in activation within populations of DS cells . This theory of the MAE, incidentally, was advanced by Mather⁸⁹ two decades ago based on psychophysical evidence. Huk and colleagues were able to index the “strength” of direction-selectivity by computing the extent to which adaptation reduced BOLD signals, relative to baseline response; the resulting “direction selectivity” indices for the six different brain areas studied are shown in Figure 3. Obviously, this index is highest in MT+, befitting its putatively

strong role in motion perception.

Insert Figure 3 about here

Recall that one hallmark property of many cells in monkey MT is their selectivity for given directions of motion independent of the orientations of the moving contours – these cells respond to the overall *pattern* of motion within their receptive fields, not to a given component of motion. Huk and Heeger⁹⁰ found compelling evidence for the existence of such “pattern-motion” responses in human visual cortex, including MT+. In their study, activation levels in multiple visual areas were measured while observers viewed a pair of superimposed gratings, differing in orientation and drift direction. In all cases, the pair of gratings cohered to form a “plaid” that appeared to drift in a direction dependent on the orientations of the components. During one block of trials (“same direction” trials), the component directions of the two gratings varied from trial to trial, but the resulting plaid itself appeared always to drift in the same direction throughout the block of trials. During another block of trials, the same component gratings were arranged in pairings in which the direction of coherent motion of the plaid varied from trial to trial (“mixed direction” trials).ⁱⁱ Huk and Heeger reasoned that the “same direction” condition, wherein the plaid always moved in a given direction, should produce strong adaptation in “pattern motion” cells, compared to adaptation of those cells in the “mixed direction” condition. Thus in a brain area containing “pattern motion” cells, the fMRI response should become periodically weaker and stronger as viewing conditions alternated between “same” and “mixed” trials. Brain areas with only component-motion cells would respond the same to both conditions. Huk and Heeger calculated a “pattern motion” index reflecting the extent to which the fMRI response was modulated, and the values of that index for different visual areas are shown in Figure 4. Here it can be seen that MT+ generates the highest degree of

ⁱⁱ The perceived direction of a plaid composed of two superimposed component gratings is governed by the orientations of the two components, by their drift directions and by their relative spatial frequencies. Thus it is possible to produce plaids from different components all of which yield the same global direction of drift, and it is possible to produce plaids from different combinations of the same components each of which yields a different direction of global motion.

“pattern motion” adaptation, implying the existence of a substantial portion of “pattern motion cells. V1, on the other hand, shows evidence for component motion cells only, with intermediate visual areas showing intermediate amounts of pattern motion responsiveness. So it appears that the human brain, like that of the macaque monkey, solves the ambiguity problem inherent in V1 cell responses by the addition of a second stage of processing in which “pattern motion” neurons extract the global direction of motion independent of the local contours signaling that direction.

Insert Figure 4 about here

MT+ also appears to receive input from other sources besides earlier visual areas. Two studies^{91,92} report that human MT+ can also be activated when observers view stationary photographs of an object in motion (e.g., a person engaged in an athletic maneuver), with activation being greater than that produced by viewing the same object without implied motion (e.g., the same person standing still). Activation associated with implied motion could be construed as evidence for “top-down” influences on MT+. Consistent with the existence of such “top-down” influences, activity levels in human MT+ are significantly boosted when observers voluntarily attend to a set of moving dots seen against a background of stationary dots; shifting attention to the stationary dots yields weakens activation even though the physical stimulation conditions remain identical.⁹³ Even a stationary object (e.g., an arrow pointing in a given direction) can produce significant activation in MT+ when that object cues the direction of motion to be portrayed in a subsequently viewed animation.⁹⁴ For that matter, simply imagining an array of moving dots with eyes closed is reported to yield activation in MT+ compared to a condition involving imagination of stationary dots.⁹⁵ Also pointing to top-down influences, Zacks et al⁹⁶ found transient spikes in MT activation time-locked to perception of transitions between two discrete activities portrayed in an extended video sequence (e.g., a person ironing a shirt and then playing a musical instrument). This finding can be interpreted as the consequence of knowledge-based monitoring of a sequence of events, with the “boundaries” between events being particularly salient. Tong⁹⁷ provides additional speculations on the implications of these intriguing results. Considered together, these intriguing findings imply that BOLD signals

originating within MT+ are not modulated solely by the physical characteristics of visual motion stimulation: mental set, imagination and intentions all influence activity within this intermediate stage of the motion pathway.

Before leaving MT and moving into neighboring visual areas responsive to motion, it is worth saying a word about the responsiveness of MT+ to motion defined by color exclusively. As discussed elsewhere in this volume, an overarching model in visual neuroscience assumes the existence of two visual pathways coursing throughout the human brain, the so-called P-pathway originating in the parvocellular layers of the lateral geniculate nucleus and the M-pathway originating in the magnocellular layers of the lateral geniculate nucleus. When this dual pathways theory was first being developed,^{98,99} great emphasis was placed on the functional segregation of color processing (P-pathway) and motion processing (M-pathway). In more recent years, however, it has become abundantly clear that vivid impressions of motion can be created by moving chromatic contours in the absence of luminance contrast (e.g., ffytche et al,¹⁰⁰ Dougherty et al¹⁰¹), although perceived speed can appear slower for chromatically defined stimuli. Likewise, single-unit recording experiments have documented the existence of neurons in monkey MT responsive to chromatic motion (e.g., Thiele et al,¹⁰² Seidemann et al¹⁰³), again undermining the notion that color and motion are strictly segregated visual processes. It is not surprising to learn, then, that human MT+ can be activated by chromatic contours defined exclusively by chromatic stimuli that produce only S-cone signals.⁷⁶ These activations are relatively weak, mimicking those generated by very low contrast luminance contours; this contrast equivalence may account for the apparent slowing of motion defined exclusively by color. It remains to be learned just how these color signals get transmitted to MT+, with possibilities including the koniocellular pathway which receives “on” signals from S cones or the P-pathway which receives “off” signals from S cones (see chapter # in this volume by Casagrande). Whatever their route to MT, it is important to keep in mind that the existence of color-driven responses in MT+ does not necessarily mean that those signals influence color appearance, a point underscored by Wandell et al.⁷⁶

Kinetic Occipital Area

Posterior to the MT+ region and lateral to V3A is another putative motion-selective area, dubbed the kinetic occipital area (KO) by Orban and colleagues;^{77,104} it is thought by some that KO may overlap with visual areas LOC/LOP identified and named by Malach et al,¹⁰⁵ although Dupont et al¹⁰⁴ argue otherwise. In brain imaging studies using PET and fMRI, KO has been distinguished by its enhanced responsiveness to contours defined by differences in velocity.^{77,104,106} Figure 1D shows a schematic example of a “kinetic” grating in which alternating “bars” are defined solely by differences in direction of motion of dots. Area KO responds more vigorously to kinetic gratings than it does to coherent dot motion in a single direction or to multiple, superimposed planes of dots. In addition to kinetic gratings, KO also responds selectively to curved motion-defined contours and to alphabetic letters defined by velocity differences between figure and background.⁷⁷ One wonders whether the neural interactions endowing KO with this ability to signal form from motion might arise from center/surround antagonism of the sort that has been described for monkey MT neurons.⁴³

Whatever the underlying neural architecture, we know that KO’s responsiveness to motion-defined forms does not extend to point-light animations depicting a human body engaged in some form of biological activity.¹⁰⁷ For brain areas responsive to this class of novel, compelling motion stimuli, we must shift our attention to yet another brain area located in the dorsal portion of the superior temporal sulcus, a location that could be construed as the confluence of the so-called parietal lobe pathway situated in dorsal portions of the cortex and the temporal lobe pathway situated in more ventral regions of the cortex.

Superior Temporal Sulcus

Using PET and fMRI, several different laboratories have sought to identify brain regions selectively activated when one views someone else engaged in various familiar activities, a form of motion perception dubbed biological motion. Results from this work point to the unique

activation of a neural area on the STS, adjacent to human MT+. We will start by summarizing a study from the Vanderbilt laboratory, for those results are highly representative of other findings.

Grossman et al¹⁰⁷ had observers view alternating presentations of point-light animations (see Figure 1F) and spatially scrambled versions of the same animations. The biological motion sequences differentially activated a region on the posterior end of the STS, just anterior to the dorsal bend (see Figure 2). This region is anterior and superior to MT+, which itself was localized by Grossman et al on the ascending limb of the lateral occipital sulcus. Neural activity in MT+ increased relative to resting baseline at the onset of the point-light animations, indicating that MT+ certainly registers the presence of motion contained in these animations. However, the levels of MT+ activation were not different for the point-light motion compared the scrambled motion; MT+, in other words, does not respond selectively to biological motion. Grossman and colleagues also compared neural activity associated with biological motion to that evoked by kinetic gratings (Figure 1D), the stimulus used to reveal visual area KO (e.g., Van Oostende et al⁷⁷). They found reliable activation to kinetic gratings in regions posterior and superior to MT+ along the lateral occipital sulcus, which dovetails closely with the published landmarks of KO. However, this region, like MT+, did not respond selectively to biological motion sequences. Nor did STS — the biological motion area — respond to the kinetic gratings. Thus STS is anatomically and functionally distinct from other brain regions putatively involved in registering shape information from motion.

In a similar demonstration of unique patterns of brain activity associated with point-light stimuli, Howard et al¹⁰⁸ compared neural activity while viewing coherent motion, optic flow, and point-light biological motion. Observers in this study first viewed a high contrast checkerboard either stationary or moving coherently. The resulting areas with higher neural activity during the coherent motion condition consisted of a large region extending over the ascending limb of the lateral occipital sulcus and into posterior parietal cortex, the region corresponding to area MT+. Observers also viewed optic flow displays and point-light biological motion displays, and the resulting neural activity was compared to when the same dot motions for each display were

randomized. Howard and colleagues found that viewing optic flow resulted in increased neural activity in a region overlapping and adjacent along the superior boundary of MT+. Biological motion sequences also produced increased neural activity superior to MT+, and this region overlapped with the cortical regions active during viewing the coherent motion and optic flow animations. Howard and colleagues interpreted their results as evidence for specialization of motion perception within MT+. Outside of the MT+ region, they also found activation on the superior temporal gyrus (STG) during the biological motion and optic flow conditions. Anterior regions on STG are known to be involved in auditory perception,^{109,110} and the site of activation on STG during biological motion perception overlapped with neural areas active during listening to a book-on-tape. Howard and colleagues suggest either that MT+ is connected to auditory cortex associated with interpretation of language, or that the overlapping region on STG corresponds to a polysensory area active during visual or auditory stimulation.

This is not the only study to find neural activity associated with auditory perception in regions quite close to those associated with visual perception. An fMRI study on voice-selective regions in cortex found two loci on human STS that had higher levels of activation when listeners heard speech sounds than when they heard energy matched non-speech sounds.¹¹¹ Speech sounds included isolated words and strings of words connected together and presented in various languages. Energy-matched non-speech sounds included natural sounds, mechanical noises and animal cries. The most posterior area of activation on STS corresponds quite closely to reported site of activation during viewing biological motion.^{107,108} Thus it is quite possible that the posterior region of STS, which is situated between primarily visual and primarily auditory responsive cortex, may in fact be a polymodal region.

Point-light animations not only convey movements such as running and walking, but can also convey specific goal oriented actions, such as picking up a box or grasping a glass.¹¹² Bonda, Petrides, Ostry and Evans¹¹³ compared neural activity while observers viewed point-light perception of goal-oriented hand actions to that found when observers viewed whole body movement. A point-light hand and arm conveyed goal-oriented activity by reaching for an

invisible glass and bringing it to the (invisible) mouth. Body movements comprised video segments of a point-light walker moving in various directions. Like the previous studies, Bonda and colleagues found that viewing body movements resulted in higher activity levels on posterior STS than when observers viewed random motion or when they viewed meaningless dot patterns moving rigidly. Viewing hand movements also produced increased activity levels on posterior STS. Based on these findings, plus those from single-unit recordings (discussed earlier), Bonda and colleagues concluded that posterior STS may be involved in the planning and comprehension of goal-directed reaching. Interestingly, during the body movement condition significantly higher levels of activity were measured in the amygdala than during the hand movement conditions, as if observers experienced emotional responses to the whole body sequences.

It is interesting to note, too, that brain activity during perception of biological motion depends on whether the perceived visual motion is biologically possible or not. In a study by Stevens et al¹¹⁴ observers viewed successive images of whole-body motion that gave the impression of smooth “apparent” motion of limbs either around the body (possible movement) or passing through the body (impossible motion). Following the lead of Shiffrar and Freyd,¹¹⁵ Stevens and colleagues manipulated the perceived path of the limb by changing the inter-frame interval (IFI): observers see the biologically plausible path of motion when IFI is long (approximately 450 msec) but see the impossible motion path when IFI is short. Stevens and colleagues identified a region on STG that responded preferentially to the possible movement over the impossible movement, as did primary motor, premotor cortex and the cerebellum.

Even simple changes in facial features — movement of the eyes or opening and closing of the mouth — are sufficient to produce increased activity levels in posterior STS.¹¹⁶ BOLD signal levels in posterior STS are higher during both the eye and mouth movement animations than during conditions in which the eyes or mouth are replaced with a low contrast checkerboard that give the impression of random movement.

In addition to activating posterior STS, viewing biological motion sequences in point-

light animations also activates four brain areas thought to be involved in object recognition: the extrastriate body area (EBA), the lateral occipital complex (LOC) and the occipital and fusiform face areas (OFA and FFA).¹¹⁷ In only two of these areas, however, the FFA and OFA, were the evoked neural signals capable of differentiating biological from non-biological motion. It seems likely that activity in FFA and OFA is consequent to the “shape” generated by the motion sequences, although this speculation remains to be tested. In any event, these results imply that the perception of biological motion engages a network of distributed neural areas in visual pathways specialized for form perception (the ventral stream) and for motion perception (the dorsal stream).

Before moving to the next section, we should point out that very recent brain imaging studies have begun to focus on visual perception of “intention” and object “affordance”.^{118,119} While not explicitly manipulating motion information, these studies seek to identify brain areas that become active when people plan activities (e.g., grasping) in response to presentation of pictures of objects or when people think about imitating the activity of another person. Results from these studies are highlighting possible links between between the visual perception of actions and the execution of those actions; these studies, thus, are extending the scope of “motion perception” to the realm of goal-directed behavior.

Motion Processing Revealed by Transcranial Magnetic Stimulation

A very promising technique for relating motion perception and brain activity is provided by transcranial magnetic stimulation (TMS), wherein a brief magnetic pulse applied to a restricted region of the scalp induces a local electric field in the immediately underlying brain tissue.¹²⁰ Because of its high temporal resolution and moderately good spatial resolution, TMS provides a unique means for producing a temporary, reversible “lesion” (functional disruption) within the brain area near the cortical surface. Rather than destruction of neural tissue, this “lesion” actually results from the brief disruption of ongoing, coherent neural activity; TMS, in

other words, transiently introduces “noise” into the underlying neural processes.

Several research groups have examined the consequence of TMS applied to the scalp region overlaying visual area MT while observers view visual animation sequences.¹²¹⁻¹²⁴ In general, TMS directed to this brain area disrupts motion perception, as evidenced by disruptions in performance on a direction discrimination task and/or a speed discrimination task; performance on control tasks (e.g., form acuity) is unaffected by TMS-induced disruption of MT activity. There is some disagreement about exactly how this degradation in motion perception varies with the relative timing of TMS and visual stimulation (e.g., compare Hotson et al¹²³ with Beckers & Zeki¹²⁵), but these disagreements could arise from methodological differences, such as the exact placement of the coil used to induce TMS.

It is illuminating to compare the effects on motion perception of TMS applied to visual areas V1 and MT. Beckers and Zeki¹²⁵ applied brief TMS pulses just before, during or just after presentation on a video monitor of a small array of dots moving coherently either leftward or rightward, with direction varying randomly over trials. (The dots were imaged to one side of fixation, so that the TMS pulse could be directed to the contralateral hemisphere in which those dots would normally evoke activity.) Observers were instructed to report the direction of motion, with TMS pulses applied to regions of the scalp coinciding as closely as possible with area V1 or area MT (Beckers and Zeki acknowledge the imprecision of targeting these areas exclusively). When delivered within ± 50 msec of the visual stimulus, TMS stimulation targeted on V1 had essentially no effect on motion perception, as evidenced by accurate performance on the direction discrimination task. Only when delivered about 60 msec after the onset of the visual stimulus did TMS stimulation of V1 disrupt performance on this task, and then by only a modest amount. TMS stimulation targeting MT, however, produced pronounced masking, lowering task performance to chance levels when TMS and visual stimulation fell within about 20 msec of one another. Based on this pattern of results, Beckers and Zeki concluded that at least some motion-related neural signals arise within MT before reaching V1, which they take as evidence for a visual input to MT that bypasses V1. This possibility will be discussed further in a subsequent

section of this chapter, when we consider the consequences of selective brain damage on motion perception.

In another, related study, Matthews et al¹²⁴ applied brief TMS pulses to the scalp region overlaying V1 or the region overlaying MT, and they required observers to judge perceived speed or perceived direction of motion associated with brief presentations of translational RDCs. TMS stimulation at both sites produced reliable slowing of perceived speed but no shift in perceived direction of motion. In a second experiment, these differential consequences of TMS applied at these two locations were replicated using forced-choice testing procedures (although there were intriguing differences in the timing relationship between RDC presentation and TMS for speed discrimination and speed perception). The failure of TMS to alter perceived direction of motion is not necessarily in conflict with the results of Beckers and Zeki¹²⁵, for that study did not directly measure perceived direction of motion but, instead, asked observers to indicate in which one of four cardinal directions an array of semi-coherent dots was moving.

In the studies just summarized, TMS was used to disrupt motion processing instigated by presentation of real motion signals. But through appropriate adjustment of stimulation levels, TMS can also be used to generate visual sensations (“phosphenes”) of motion in the absence of external stimulation; deployed in this way, TMS transiently induces illusory motion when applied to visual area MT.^{126,127} Two experiments using TMS to trigger phosphenes have produced intriguing results bearing on the question of V1’s role in motion perception.

Cowey and Walsh¹²⁶ created TMS-induced phosphenes in normally sighted individuals, in an adult male with complete blindness owing to bilateral optic nerve damage (peripheral blindness) and in an adult male missing his left striate cortex (blindsight patient GY, who will be discussed in more detail in the following section). In all observers — sighted and blind — magnetic pulses were applied over the occipital scalp at points targeting primary visual cortex as well as at more dorso-lateral points targeting MT+. The normal, sighted individuals all experienced phosphenes, sometimes colored but never moving, when stimulation was directed to regions presumably activating neural tissue around the calcarine sulcus and, by inference, the

primary visual cortex; the visual field location of the phosphenes depended on the specific site of stimulation and, of course, on the hemisphere stimulated (phosphenes always appeared in the visual field contralateral to TMS stimulation). When the site of pulse delivery was moved to the more dorso-lateral site (presumptive area MT+), all normally sighted observers experienced moving phosphenes in the visual field contralateral to the stimulated hemisphere; the overwhelming majority of phosphene descriptions characterized them as greyish and sparkly; faint color sensations were rarely reported. When TMS stimulation was applied to the same regions of the occipital lobe of the man blind from optic nerve damage, he described the same two categories of phosphenes (stationary, colored vs. uncolored, moving). Incidentally, this individual also expressed surprise and pleasure upon experiencing visual phosphenes. Patient GY experienced normal phosphenes — stationary and moving — when TMS was delivered to the appropriate sites over his right hemisphere. But when pulses targeted the equivalent sites in his left hemisphere, GY experienced no phosphenes whatsoever. The absence of stationary phosphenes with more posterior stimulation is not surprising, since GY's damage encompasses the entire striate cortex in the left hemisphere. However, the absence of visual phosphenes when area MT+ is stimulated is rather surprising, since this region is intact in GY as evidenced by successful activation of MT+ in both hemispheres in fMRI studies.¹²⁸ From this pattern of results, Cowey and Walsh conclude that activity in area MT+ alone is necessary but not sufficient for generation of visual motion perception.

This same conclusion has been reached based on results from a clever experiment in which TMS was used both to evoke motion sensations and to disrupt them. Pascual-Leone and Walsh¹²⁹ applied TMS to both V1 and MT in close temporal proximity; importantly, the MT pulse was set to a level yielding perception of moving phosphenes when delivered on its own and the V1 pulse was set to a level too weak to yield phosphenes of any sort. Results from this intriguing study are reproduced in Figure 5. When TMS was applied to V1 before MT, moving phosphenes were reliably reported by observers, as if the V1 TMS pulse had not been delivered at all. But when TMS was delivered to V1 5 - 45 msec after TMS stimulation to MT, perception

of moving phosphenes was abolished. This outcome is remarkable because the disrupting, V1 event is occurring later in time than the evoking, MT event. The authors interpret their findings to indicate that V1 activation is necessary for conscious awareness of visual motion, even though the signals critical for signaling that motion arise in MT. Obviously, this conclusion dovetails nicely with the findings of Cowey and Walsh.¹²⁶

Insert Figure 5 about here

TMS applied to the scalp overlying MT+ can also neutralize the effect of prior exposure to a motion stimulus that, ordinarily, would facilitate or “prime” subsequent performance on a motion task.¹³⁰ In this study, observers were administered a series of brief trials separated by short intervals. During each trial the observer viewed four arrays of moving dots presented around a central fixation point. In three of the arrays all dots moved in the same direction and in the fourth array the dots moved in a direction orthogonal to the other three; observers indicated by manual response which one of the four arrays portrayed the “odd” direction. Ordinarily, observers are faster at making this judgment when the direction of motion of the “odd” target is the same on consecutive trials, regardless whether it appears in the same location. This perceptual priming effect implies that visual information during one exposure is briefly retained in visual memory and utilized during processing in the following exposure. Campana et al¹³⁰ showed that a brief TMS pulse delivered during the intertrial interval, when no motion stimulus was present, was sufficient to neutralize this priming effect. This disruption of processing was specific to motion stimulation: when the same task structure required the use of color for the “odd” discrimination, TMS applied to MT had no deleterious effect on priming. These results add weight to the idea that visual memory engages some of the same areas involved in actual visual perception, and they further highlight the potentially powerful marriage of psychophysics and TMS.

Visual Motion Perception and Brain Damage

In this final section we focus on deficits in motion perception consequent to accident-induced or disease-induced lesions of restricted regions of the brain, another, complementary strategy for identifying motion-responsive areas of the human brain. Our survey of this literature will be highly selective, and readers seeking a more comprehensive overview will want to consult any of several available sources.^{131,132}

Cerebral motion blindness. Certainly one of the most dramatic examples of motion impairment is the “motion blindness” or “akinetopsia” suffered by LM, a woman who sustained bilateral damage to the occipito-parietal region, consequent to cerebral infarction;¹³³ subsequent brain imaging confirmed that the damage included (but was not limited to) visual area MT in both cerebral hemispheres.¹³⁴ⁱⁱⁱ This fascinating individual has been extensively studied by a number of labs. While maintaining serviceable spatial vision, reasonably good object recognition and normal color vision, LM denies experiencing any sense of visual motion. Video clips of LM reveal that she pours water into a glass past the point where it overflows, presumably because she cannot see the rising water level, and she experiences anxiety when attempting to cross the street or navigate environments replete with moving objects. Even with forced-choice testing in the laboratory, LM experiences great difficulty telling which of two objects is moving at a higher speed¹³⁶ or the direction in which a single stimulus moves.^{134,137} For instance, LM sometimes confuses upward motion and downward motion, especially when coherent, translational motion is presented against a background of static, random noise. When asked to identify letters defined by moving dots presented against a stationary background (motion-defined form), LM experiences great difficulty.¹³⁷ LM has also been tested on the “Ternus” ambiguous motion display in which three evenly spaced bars shift positions back and forth by one cycle from frame to frame.¹³⁸ Normal observers perceive apparent motion with this display, with the particulars of

ⁱⁱⁱAs an aside, LM is almost certainly not the first case of akinetopsia to be described in the neurological literature — Zeki¹³⁵ describes several other, similar cases dating back to the early part of the 20th century, and he also describes cases in which motion perception is preserved in the face of losses in visual object recognition and color vision.

the motion depending on the interval separating successive frames. Upon viewing the Ternus display, LM denies *any* experience of motion and, instead, describes the outer bars as blinking on and off regardless of the timing of the frames.¹³⁷

It is perhaps not so surprising that LM, because of her brain damage which encompasses visual area MT, suffers impaired motion perception. After all, monkeys with lesions in the homologous region of the brain show pronounced deficits in detection and discrimination of motion,¹³⁹⁻¹⁴¹ although recovery can lessen the perceptual impact of such lesions.¹⁴² (LM's impaired motion perception, by contrast, showed little recovery over the 15 years or so following her stroke.) Furthermore, the neural consequences of LM's damaged MT most likely adversely affected other brain regions innervated by MT and normally involved in motion processing (e.g., the frontal eye field).^{iv} Finally, we must keep in mind that LM's lesion encompasses tissue outside of area MT, and it encroaches on white matter as well. In fact, some of her perceptual difficulties may be related to the large cerebellar lesion that extends to some midline structures. Consistent with this possibility, Nawrot and Rizzo¹⁴³ have shown that patients with midline cerebellar lesions exhibit elevated thresholds for detecting coherent motion in random-dot cinematograms. These impairments were measured at least 2 years after the incident leading to cerebellar damage, and they are not attributable to unstable fixation or poor tracking eye movements, for both of these oculomotor functions were normal in this sample of ten patients. Nawrot and Rizzo (1998)¹⁴³ speculate that the cerebellum may play a crucial role in the perceptual interpretation of retinal image motion, which can arise from object motion, head movements and postural adjustments.

So, given the large extent of LM's bilateral damage, it is simply impossible to attribute her motion disorders to the loss of any single visual area such as MT. There are, however, other neuropsychological studies that point to the crucial involvement of MT in normal motion

^{iv}In the Zihl et al¹³³ paper, LM's pursuit movements were characterized as normal when tracking a relatively slowly moving target, but at higher speeds the tracking movements were jerky and saccade-like. To our knowledge, no other, detailed eye movement records were published on LM (she died in the spring of 2002).

perception. In one large sample of 50 stroke patients with unilateral brain damage, deficits in direction discrimination using RDCs were reliably observed in those individuals whose damage included the MT+ complex but not in patients whose lesions were confined to more ventral structures in the temporal lobe.¹³² Two studies^{144,145} have examined motion perception in patients with parietal lobe lesions, finding no deficits in speed perception or detection of moving gratings. On the other hand, patients with damage to the right parietal lobe do evidence deficits in their ability to track the motion of a subset of targets within an array of moving elements,¹⁴⁶ a deficit that probably involves an inability to attend to transient events, not necessarily a deficit in registration of motion information *per se*.

As well, there are some single case studies in which specific motion deficits are associated with highly restricted, unilateral lesions, and these results also bear on the role of MT in motion perception. Several of those studies are summarized in the next section.

Brain damage and perception of first-order vs second-order motion. An intriguing selective deficit in motion perception has been identified in patient FD, an adult male with a small, shallow lesion located dorsal to, but not including, area MT; unlike LM's lesion, FD's damage is confined to one hemisphere, making it possible to compare visual performance in the two visual fields, contralateral and ipsilateral to the brain damage. Patient FD has been described in some detail by Vaina and Cowey,¹⁴⁷ whose findings can be summarized as follows. FD has normal visual acuity, normal visual fields, good color vision and stereopsis and normal contrast sensitivity. He can readily judge 2D and 3D shape from motion (recall Figure 1E), and he has no trouble discriminating the direction or speed of moving stimuli. His coherence thresholds for detection of translational motion in RDCs (Figure 1C) are within the normal range, regardless in which visual field the moving dots are imaged. So in these respects, FD's motion perception is very different from LM's. However, FD does show pronounced impairments in perception of "second-order" motion in which movement is signaled by displacements of boundaries defined solely by contrast variations or by texture discontinuities, not by luminance differences (compare

Figures 1A and 1B). This deficit in perception of second order motion was found on several different tasks.

In a related study, Vaina et al¹⁴⁸ identified a male patient, TF, with a very small, unilateral lesion that involved visual area V2 only, with MT completely spared. In contrast to FD, TF experienced difficulties on motion tasks involving “first-order” motion (e.g., direction and speed discrimination in random-dot cinematograms) but he exhibited normal motion sensitivity to “second-order” motion. This distinction between the consequences of brain damage on first vs second order motion is buttressed by Greenlee and Smith’s¹⁴⁹ study of a large sample of patients with unilateral brain damage to regions of the temporal, lateral occipital and/or posterior parietal cortex. All patients exhibited essentially normal performance on a grating orientation task, but all performed below normals on several motion discrimination tasks; the magnitude of the motion deficits were dependent on the nature of the task (direction vs speed discrimination) and the nature of the motion stimuli (first-order vs second-order). This pattern of results, too, supports the idea that motion perception is not a unitary ability arising from a single set of neural mechanisms. Considered together, these three neuropsychological studies, along with others by Plant and colleagues,^{150,151} lead to the conclusion that the human brain contains multiple motion pathways that implement different computational operations, pathways whose components are differentially vulnerable to brain damage.

Brain damage and perception of biological motion. Next we turn to neuropsychological studies relating brain damage to performance on “shape from motion” tasks, with particular emphasis on perception of biological shapes defined by motion. Vaina et al¹⁵² examined an individual, patient AF, with bilateral lesions of the temporal-parietal-occipital junction extending into the posterior parietal cortex of the left hemisphere (this pattern of damage was consequent to a series of strokes that occurred over an unspecified period of time). AF’s ability to see shape from luminance or from color was intact, but he was unable to see shapes defined by retinal disparity or by motion. AF also had elevated thresholds for detecting coherent motion signals in RDCs.

Nonetheless, AF could easily identify biological motion in Johansson's point-light displays (Figure 1F). He readily described animations of a man climbing the stairs or riding a bicycle, and he had no problem reporting the direction in which the "walker" was headed. So despite obvious difficulties on more basic motion perception tasks, AF experienced no problem seeing the more complex forms of motion specifying human activity in point-light animations.

Another interesting patient, AL, suffered two strokes that affected her temporal lobe and posterior parietal cortex.¹⁵³ Immediately following her second stroke AL was unable to recognize friends and family members by looking at their faces or watching their gaits, but she could readily identify them by their voices. She soon regained her ability to recognize familiar faces when people remained still, but complained that she could not recognize those same faces when the people were moving. During psychophysical testing AL was almost perfect at discriminating luminance defined geometric shapes, but she could not detect objects defined by flicker nor could she discriminate objects defined by motion. She failed to tell the difference between a virtual cylinder created from moving dots and a spatially unstructured display of the same moving dots. AL also utterly failed to identify any human activities depicted in point-light animations, even after having been told that the display showed a person engaged in a routine action. So unlike patient AF, AL's motion deficits included perception of biological motion.

Schenk and Zihl^{154,155} measured motion and form perception in two patients with bilateral lesions in the parietal lobe and extensive damage to the underlying white matter. These patients had normal coherent motion thresholds and were able to segregate sketches of objects superimposed on top of each other. These patients also recognized human activities depicted in point-light animations when a single biological actor was animated; however, when the actor appeared embedded in a field of static or randomly moving dots, the patients, unlike normal observers, were unable to recognize the point-light figure.

Considered together, results from these four studies imply that perception of one particular form of shape from motion — the human form defined by the kinematics in point-light animations — can be spared in the face of deficits in other aspects of motion perception and,

conversely, can be impaired even when other aspects of motion perception are normal. In the neuropsychological field, these kinds of double dissociations are typically taken as evidence for the involvement of multiple, anatomically distinct neural mechanisms.

Conclusions

It was the noted comparative anatomist Gordon Walls¹⁵⁶ who poetically described the significance of visual motion perception to behaving organisms:

"If asked what aspect of vision means the most to them, a watchmaker may answer 'acuity', a night flier, 'sensitivity', and an artist, 'color'. But to the animals which invented the vertebrate eye, and hold the patents on most of the features of the human model, the visual registration of movement was of the greatest importance."

As Walls knew, the ability to detect and recognize visual events in our environment is crucial to our survival, and motion perception is the key to event perception. Motion specifies where objects are headed (direction) and how soon they'll get there (speed). Motion aids in the perception of the shapes of moving objects (shape from motion) and their relative positions in 3D space (depth from motion). In fact, motion can reveal the presence of an otherwise camouflaged object when that object moves. Motion also allows mobile organisms to get about in their environments, avoiding obstacles while at the same time intercepting objects of interest (heading from optical flow). And motion portrays important information concerning other people's body movements and facial expressions, allowing us to recognize who those people are and what their intentions might be.

Befitting this set of crucial visual abilities, it is not surprising to learn that considerable neural territory in the human brain is devoted to the registration and analysis of motion

information. Moreover, damage to portions of that neural hardware can produce serious perceptual problems that require substantial compensatory modifications to behavior. Our understanding of brain mechanisms underlying motion perception has advanced greatly during the last fifteen years, in part because of exciting technological advances in brain imaging and in brain stimulation. It is also worth noting that those advances were grounded in a solid psychophysical literature on motion perception and on sophisticated computational models that often guided the search for underlying neural mechanisms.

Figure captions

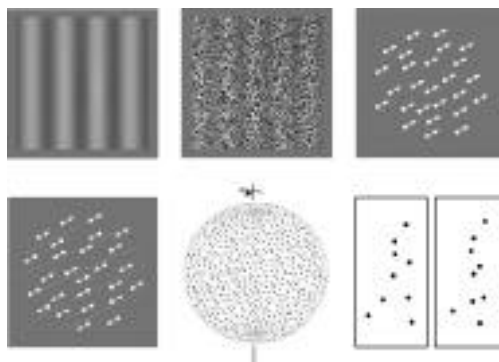


Figure 1. Schematic examples of some of the animation displays used in many brain imaging and neuropsychological studies of human motion perception. As discussed in this chapter, these different types of visual motion engage different neural mechanisms in the human brain. A. One-dimensional luminance grating (sinusoidal variations in luminance in the horizontal direction). When shifted in phase in small steps over time, the grating appears to drift in one of the two directions orthogonal to its contour orientation; this is one example of a first-order motion stimulus. B. One-dimensional, contrast-defined grating. The contrast of this random array of dots is sinusoidally modulated in the horizontal direction, producing visible “bars” defined by contrast modulation. The contrast envelope can be shifted over time in small steps in either direction orthogonal to its contour orientation, thereby creating an impression of motion; this is one example of a second-order motion stimulus. C. Array of small, randomly positioned dots that can move in any direction from frame to frame of the animation. Called a random-dot cinematogram (RDC), the ‘strength’ of motion can be varied by manipulating the percentage of total dots that moves in a given direction. In this example, 100% of dots move up and to the right. D. Subsets of dots within virtual regions of the array move in different directions, creating “boundaries” defined by differential optic flow. In this example, dots within alternating “bars” of a grating move in opposite directions, creating the vivid impression of oriented contours. This so-called “kinetic grating” (and other figures like it) can be employed to isolate regions of the human brain responsive to motion-defined form. E. Array of small, randomly positioned dots whose frame to frame motion corresponds to the optic flow associated with a real, 3D object, a sphere that rotates about its virtual vertical axis in this example; complex flow fields of this sort

create shape from motion (SFM). F. Two frames from a special type of SFM animation in which the motions of a small cluster of dots track the changing positions of the joints of a person engaged in some activity. In this example, the two frames (non-successive) depict a human walking in a rightward direction. Called “point-light animation” this technique was devised by Johansson¹⁵⁷ as a means for isolating the kinematics associated with biological motion. Brain imaging studies have compared activations associated with viewing examples of biological motion to activations associated with viewing scrambled versions of these same dot motions wherein the perception of biological motion is destroyed; results from those studies point to a region on the superior temporal sulcus as a reliable source of activation.

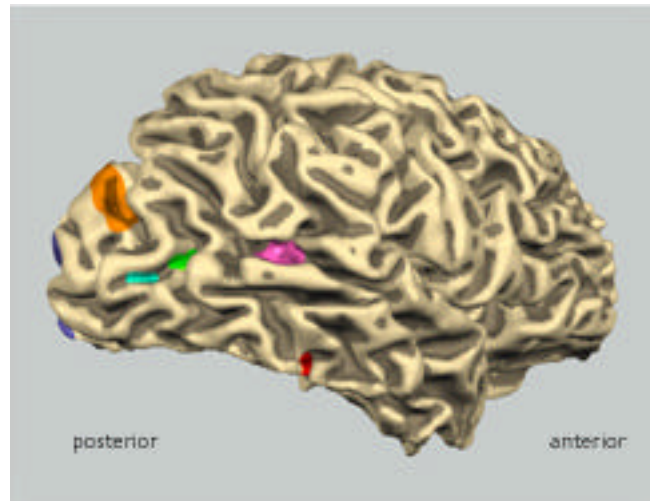


Figure 2. Human brain with colored regions denoting approximate locations of several well-studied motion-responsive areas of the human brain, areas that are discussed in this chapter. These regions are shown superimposed on a high-resolution surface map of the right hemisphere, reconstructed at the level of the white matter/gray matter junction. These motion responsive visual areas are color-coded as follows: purple – V1 (which is actually located on the medial surface of the hemisphere and, therefore, is largely hidden in this lateral view); orange – V3a; cyan – kinetic occipital region (KO); green – MT+ complex (sometimes referred to as V5); pink – posterior part of the superior temporal sulcus (STSp); red – fusiform face area (typically localized by contrasting two viewing conditions: faces vs objects). There are additional motion-

responsive areas that have been identified using fMRI, located in more dorsal and more anterior regions of the brain and, as well, in the cerebellum (not pictured). For a comprehensive map of these multiple motion areas, see Sunaert et al.⁵⁶

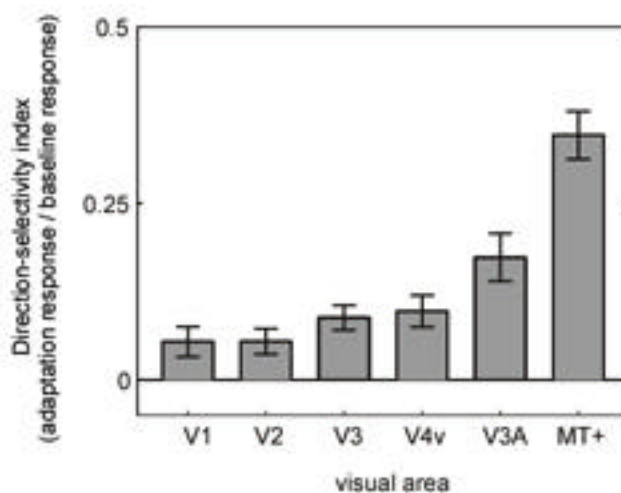


Figure 3. Histogram showing values of a ‘direction selectivity’ index measured from six different areas of the human brain responsive to motion. These indices were obtained using an fMRI adaptation procedure in which brief episodes of motion were interleaved over closely spaced trials; details of the procedure are given in the text. Reprinted with permission from “Neuronal basis of the motion aftereffect reconsidered” by A.C. Huk, D. Ress and D.J. Heeger, *Neuron*, 32, 161-172, Copyright 2002, Cell Press.

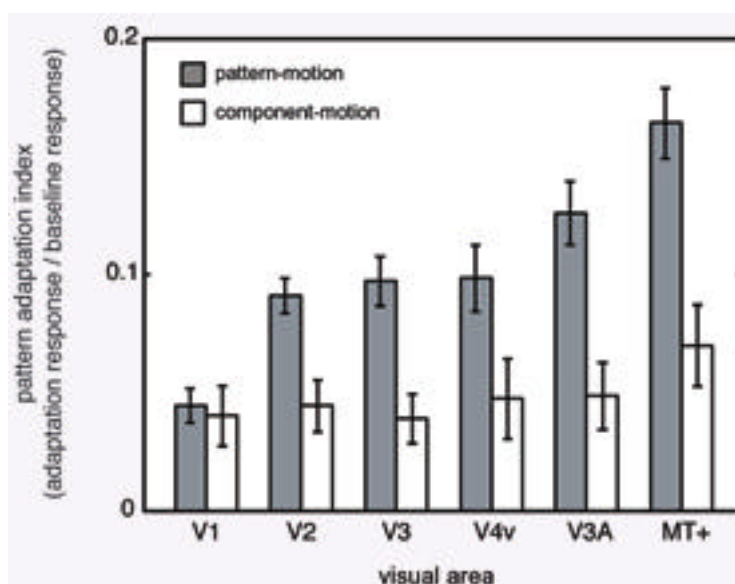


Figure 4. Histograms showing the extent to which brain activation in different visual areas fluctuates in coincidence with presentation of pairs of moving gratings that stimulate “pattern motion” neurons vs “component motion” neurons. Larger index values denote greater “pattern motion” responsiveness. Reproduced with permission from “Pattern-motion responses in human visual cortex” by A.C. Huk & D.J. Heeger, *Nature Neuroscience*, 5, 72-75, Copyright 2001, Nature Publishing Group.

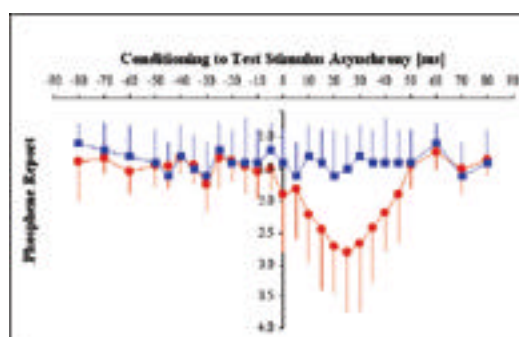


Figure 5. Graph showing the incidence of moving phosphenes elicited by the application of a brief TMS pulse to a region of the scalp overlaying visual area MT in the human brain. Human volunteers judged the quality of the phosphenes using a 4-category scale (1 = phosphenes present and moving; 2 = phosphenes present but uncertain whether they moved; 3 = stationary phosphenes; 4 = no phosphene observed). A second, brief TMS pulse was also applied at varying times relative to the MT pulse, with the interval between the two pulses plotted on the abscissa. For data shown with squares, both pulses were applied to area MT – this pairing had no effect on the high incidence of perceived motion regardless of the interval between pulses. For the data shown with circles, one TMS pulse of the pair was applied over visual area V1 at varying times relative to the MT pulse – when the V1 pulse closely followed the MT pulse, the incidence of moving phosphenes was reduced significantly. Reproduced with permission from “Fast backprojections from the motion to the primary visual area necessary for visual awareness” by A. Pascual-Leone and V. Walsh, 292, 510-512, Copyright 2001, American Association for the Advancement of Science.

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