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Disorders of motion and depth

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The perception of motion and depth is crucial to ambulating successfully through a cluttered route. Knowing the location of and detecting the relative movement of objects provide valuable navigation information about the spatial layout of the scene as one moves through the environment [1,2]. For instance, even in uncluttered environments, poor depth perception is an important risk factor for hip fracture in the elderly [3]. Deficits in motion and depth perception, however, often are difficult to detect in cursory examination, as patients may have normal visual acuity, contrast sensitivity, color vision, and visual fields. This article outlines what is currently known about disorders of motion and depth and the basic techniques used to examine more closely those deficits.

Motion and depth are closely related aspects of visual experience, as perception and neural processing of motion and depth invariably are linked. For instance, neurons in cortical area medial temporal (MT), initially identified and studied for motion selectivity [4–7], also show binocular disparity selectivity [8], suggesting a role in motion perception and depth perception. Psychophysical studies using aftereffects have demonstrated fundamental interactions between motion and stereopsis [9–11], and even without binocular stereopsis, motion remains an important source of depth information. The perception of depth from motion may be important for observers lacking normal binocular stereopsis.

The study of functional disorders and the location of brain damage producing the disorder reveal much about the processing architecture within the brain [12]. For instance, the study of injured soldiers during World War I led Holmes [13,14] to outline the orderly retinal representation in primary visual cortex and led Riddoch [15] to first suggest that the perception of movement may be dissociated from the perception of object form. This article looks at deficits in motion and depth perception and what these

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deficits might suggest about how the brain processes these important forms of visual information.

Akinetopsia—a motion-perception deficit

Zeki [16], in coining the term akinetopsia for a selective motion-perception deficit, provides a contrast between the histories of akinetopsia and achromatopsia. The acceptance of achromatopsia took a century of study and debate, whereas akinetopsia gained quick and easy acceptance based on a single case, that of LM [17]. Such quick and placid acceptance is surprising [8] and suggests that Zihl and colleagues' [17] finding was almost expected, even if it had not previously been reported. As Zeki points out, a century of brain research and the identification of cortical area MT/V5 in the monkey made an enormous difference in setting the stage for akinetopsia. Several events concurrent with the first report of LM's deficit helped usher quick acceptance, however. Newsome and Paré [18] demonstrated in the monkey that a lesion of cortical motion processing area MT produced a selective deficit in motion perception. At the same time, several visual motion sensor models were proposed, and these models were changing how researchers were thinking about motion perception [19–21]. For instance, the stimulus distinction between "real motion," such as an object moving in the environment, and "apparent motion," movement depicted by sequential presentation of static frames, was inconsequential. As suggested by Watson and Ahumada [19], "movement is not a physical property of the stimulus"; instead the perception of motion is the result of dynamic visual input activating specific visual spatio-temporal filters or motion detectors. "In short, all motion is apparent." At the same time, Nakayama [22] published an influential review of motion processing, giving one of the first citations of patient LM [17]. Perhaps one final influential event was the publication of Sacks' *The Man Who Mistook His Wife for a Hat* [23]. The extraordinary stories contained within this best seller were read widely by scientists and nonscientists, making the possibility of a patient showing a selective motion perception deficit comparatively easy to believe. These events helped put together the zeitgeist for akinetopsia and helped start a large-scale effort into understanding the neural mechanisms for motion perception.

The "motion-blind" patient—LM

The case of LM, first presented in by Zihl et al [17], has been the most influential case of a selective motion perception deficit in a human. LM's deficits are the result of somewhat symmetric, bilateral cortical lesions of the middle temporal gyrus, occipital gyrus, and occipital and occipitoparietal white matter, presumably resulting from venous sinus thrombosis [24]. The right posterior cerebellar lobe also was damaged by the event.

Although motion-perception deficits have been studied in many other patients, LM remains the single most studied case of a selective motion-perception deficit. Aside from motion perception, LM has much normal visual function [24], normal spatial and temporal contrast sensitivities [25], and normal visual cognitive abilities [26]. She does not exhibit characteristics of simulatanagnosia or Balint's syndrome [24,26].

Investigations involving several laboratories have produced a thorough overall picture of LM's deficit. LM perceives short-range motion (small spatial and temporal intervals) [17] if the velocity is less than approximately 6°/s [27] but has difficulty with long-range apparent motion [24]. LM can detect and indicate the direction of coherent motion in random-dot cinematograms but only with high-signal proportions, as her ability to perform this task quickly degrades with the addition of noise or stationary dots [26–28] (Fig. 1). Underscoring the extent of LM's motion-perception deficit, she reported no motion aftereffect to perceived real motion [17], and she was unable to use motion as a search criteria in a visual search task [29].

LM's ability to determine 2-D shape from motion was poor [26,28], but LM did perceive 3-D structure from motion (SFM), even reporting spontaneous perceptual reversals in the direction of figural rotation, although the perception of SFM also degraded when the form was presented amidst noise dots. Even more, binocular disparity information did not disambiguate the direction of figure rotation as it does for control observers [26]. LM also perceived biologic motion from the movement of dots representing joints on the walking figure [28].

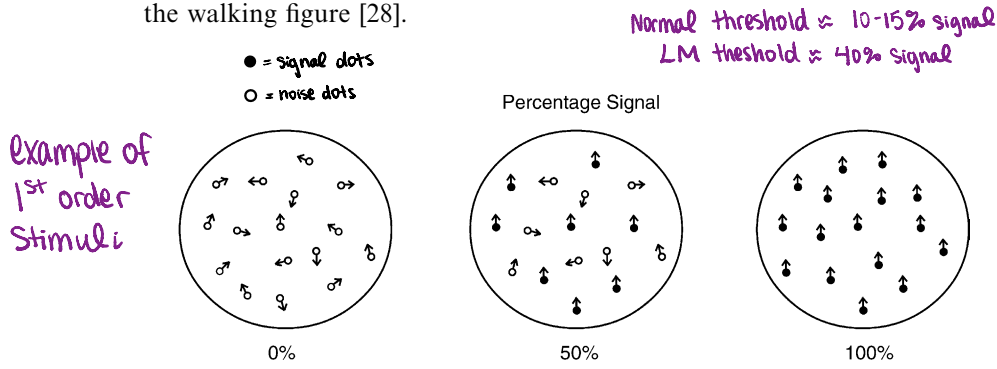

Fig 1. Motion perception may be assessed using random-dot cinematograms containing a mixture of signal dots (*filled dots*), which move in a particular direction (upward is shown), and noise dots (*open dots*), which are given a movement direction from a flat distribution of 360°. Typically, these stimuli are composed of hundreds or thousands of dots and individual dots vary between being signal and noise dots in successive frames so that each dot moves along a more-or-less random trajectory. The observer's task is to determine the direction of signal dot movement, which is usually perceived as a direction of global flow of all the stimulus dots. The proportion of signal dots and the direction of signal dots vary between trials in an effort to determine a threshold amount of signal for an individual to perform the motion direction discrimination task. The task is impossible at 0% signal and quite easy at 100% signal. Normal thresholds are approximately 10% to 15% signal, whereas the threshold for LM is approximately 40% signal [26].

Overall, the pattern of visual deficits and residual visual function is similar to those found in a monkey with area MT removed [28], underscoring the importance of a human homologue of area MT in the perception of motion. Given the enormous size of LM's lesion, localization of the human homologue of area MT was impossible in her case. LM sparked a great deal more study, however, of motion-perception deficits in humans with a variety of lesions where the goal was to better localize MT and better understand its role in the visual perception of motion.

Patient SF

An unusual set of circumstances allowed extraordinary experimental control in the study of the transient motion-perception deficit of patient SF [30]. This young patient with medically intractable epilepsy was tested in a variety of psychophysical motion-perception tasks before and after surgical resection of a small part of right lateral occipitotemporal cortex. The resection was a topectomy with a depth of only 5 to 6 mm, and the underlying white matter was spared as much as possible. The placement of a subdural electrode plate two weeks prior to surgical resection served as a "sham" surgery from an experimental perspective, thereby providing a control for the effects of the craniotomy. During surgery, four cortical tissue specimens were removed and processed for pathology and histologic characteristics. One specimen showed a region of dense myelination and SMI 32 staining consistent with the properties of area MT in the monkey. Together, these unusual circumstances lend support to the suggestion that the brain lesion, which likely damaged at least some portion of a human homologue of area MT, produced the specific effects on motion perception.

After the initial crainiotomy, but prior to cortical resection, SF's performance was normal on all psychophysical tasks [30]. After resection, most perceptual and cognitive functions remained unaffected. For instance, SF's visual acuity, contrast sensitivity, and performance in static control tasks were unchanged. SF's performance on direction discrimination in random-dot cinematograms was affected in the contralateral hemifield, however, but returned to normal within a few weeks. The perception of second-order motion was affected in both visual hemifields and also quickly returned to normal. SF's ability to recover 2-D shape from motion was impaired with stationary background dots but was normal when form and background dots moved in opposite directions. Overall, the psychophysical testing of SF shows that the topectomy produced a large, but short-lived, deficit in the perception of motion similar to the deficit reported with lesion of area MT in the monkey [18].

First-order and second-order motion

Real object motion, for instance of a dark object over a light background, activates motion detectors with elements that are sensitive to luminance

changes resulting from translation of the luminance difference between object and background. This often is referred to as first-order motion. Random-dot cinematograms, such as those described in Fig. 1, are first-order motion stimuli.

First-order motion detectors, however, are incapable of detecting motion of some other visual property, such as a region of local flicker or contrast change [31,32]. For instance, Fig. 2 illustrates a second-order motion stimulus where individual dots do not move; instead, a region of local

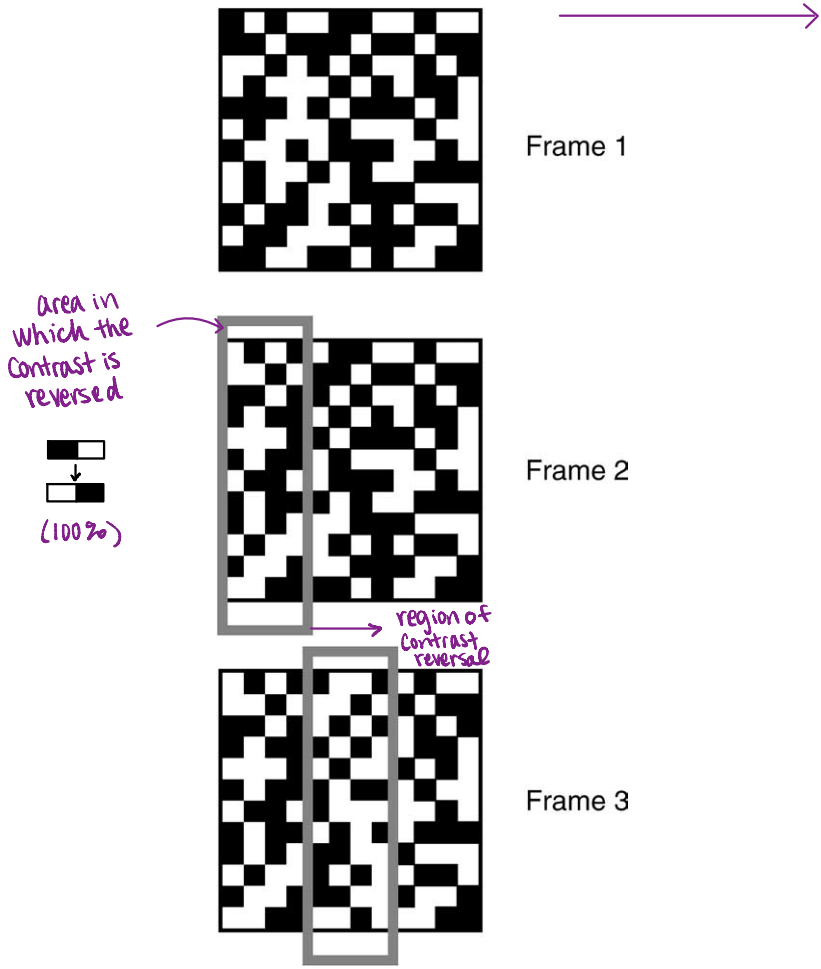

Fig. 2. Depicted is a second-order motion stimulus moving to the right. Individual dots do not move as in first-order motion. Instead, the contrast within the region delineated by the gray border is reversed (black to white, white to black). The region of this contrast reversal is moved across the stimulus. The proportion of squares within the region that actually reverses contrast (signal) can vary between 100% (shown) and 0%, where none of the squares change contrast.

contrast change is depicted moving from left to right. This second-order motion stimulus fails to activate first-order motion detectors, but is capable of generating a clear motion percept. O'Keefe and Movshon [33] found some neurons in monkey MT respond to, and show the same directional selectivity for, first- and second-order motion. It remains unclear, however, if this represents a small conjunction of separate first- and second-order processing streams or if these two types of motion are processed together.

The emerging human neuroanatomic understanding of first- and second-order motion processing is that unilateral cortical lesions near the occipito-temporal-pareital junction, including anterior occipital lobe, posterior medial or superior temporal gyrus, or inferior parietal cortex, can produce a deficit in the perception of first-order motion in the contralateral visual hemifield [30,34–37]. Reports have even implicated a medial occipital lesion with a selective first-order motion deficit [38]. Barton and colleagues [39,40] found a directional asymmetry in these motion perception deficits causing poorer performance of motion in the direction toward the side of the lesion. The variability of locations, illustrated previously, shows that damage to a single specific location produces a deficit, whereas sparing of this location spares first-order motion perception. Unilateral lesions, however, can produce bilateral deficits. Greenlee and Smith's [41] results show that first-order motion perception can be affected in both visual hemifields, and Vaina and colleagues [37] found similar results for patients with more dorsolateral and anterior lesions.

There is less consensus on the perception of second-order motion. If the perception of second-order motion is the product of high visual processing with greater interhemisperic connections and broader receptive fields spanning the visual midline, then deficits of second-order motion may affect both visual hemifields. Like their first-order deficits, Greenlee and Smith [41] found broad second-order deficits affecting ipsilateral and contralateral visual fields, whereas Braun et al's [35] deficits were confined to the contralateral hemifield. As discussed previously, patient SF [30], showing a unilateral first-order deficit, had a deficit of second-order motion that affected both visual hemifields. Finally, Plant et al [34] and Vaina and Cowey [42] report patients having a unilateral left hemisphere lesion that produced a selective second-order motion deficit in the contralateral visual field.

Uncertainty also remains concerning whether or not first-order and second-order motion are processed in the same cortical regions. Vaina and colleagues [42–44], studying two patients with selective deficits of first-order and second-order motion perception, propose that second-order motion is processed separately, relying on a more dorsal cortical region. Greenlee and Smith [41] are more equivocal: lesion analysis from a group of patients shows that first- and second-order motion are mediated by the same cortical regions, with a small indication that second-order motion may have greater posterior parietal involvement. In contrast, Braun et al [35] found no selective deficit of first- or second-order motion. Nawrot et al [30] found

that both types of motion were affected by a right lateral occipitotemporal topectomy, although the perception of second-order motion also was affected in both visual hemifields. A clear resolution of this will rely on more reports of either the dissociation or the coincidence of first-and second-order motion deficits and their accompanying lesions and whether or not second-order lesions are more closely associated with more dorsally located parietal lesions.

Shape from motion

Motion is a powerful cue for the segmentation of a 2-D shape or form from a background [45,46]. Differences in the direction of motion between a form and its background provide a potent cue to the shape of the form (Fig. 3). Using functional imaging, Orban and colleagues [47,48] found that the lateral occipital cortex in humans contains a region, which they labeled kinetic occipital (KO), specialized for the detection of motion-defined boundaries. This region, located posterior to human MT, also is called lateral occipital complex (LOC) and seems to have a broader role in object recognition [49]. As expected with damage to this region, patient SF [30] showed a deficit for motion-defined form, but not velocity-defined form. Consistent with the bilateral representation of this area, SF's deficit was more pronounced in the visual hemifield contralateral to the lesion. The "motion-blind" patient LM also has great difficulty in the perception of

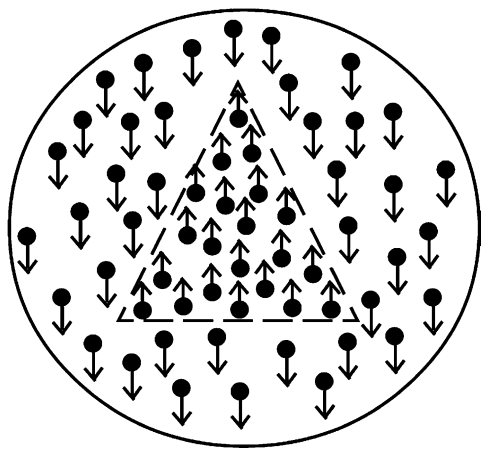

Fig. 3. Depicted is a 2-D shape-from-motion stimulus. A form is made visible, here a triangle, by having dots within the form move with a different direction or velocity as dots in the background. To assess ability to recover shape from motion, the proportion of dots differing from background dots can vary between 0% and 100%. It is important to eliminate nonmotion cues, such as edges and dot density cues to the shape.

shape from motion boundaries [26], although this finding is less informative considering her broad lesions and extensive motion-perception deficits.

Conflicting neuroimaging results, however, suggest that this process occurs much earlier. Using visually evoked responses in monkeys and humans, Lamme et al [50] found that primary visual cortex was involved in the perception of shape from motion. Also, Reppas et al [51], using functional MRI (fMRI), confirm that primary visual cortex is involved in this function and found little involvement of extrastriate regions including area MT.

Regan et al [52] found deficits in the recognition of motion-defined form from unilateral, left or right, parietotemporal lesions. Half these patients retained the ability to detect the form, and all these patients could discriminate leftward and rightward motion in 100% signal, low velocity cinematograms. Perhaps the large parietotemporal location of these patients' lesions impinged on or undercut connections with the lateral occipital location of KO.

Other motion perception deficits—subcortical regions

Motion perception deficits have been noted in patients with damage to other brain regions. These regions typically have strong connections and are involved in visuospatial processing and eye movement coordination and execution. Oscillopsia, perceived movement of the world when the subject moves, is one common visual feature of subcortical lesions. That is, during head and body movements, the visual scene usually is kept stationary on the retina by compensatory vestibular-ocular response (VOR) eye movements. If the eyes move, the movement of the scene on the retina is nulled by an efferent copy or corollary discharge [53,54] mechanism that cancels the perception of movement caused by eye movements. Eye movement and vestibular processing involve subcortical structures. Therefore, subcortical damage may disrupt motion perception by disrupting actual detection of movement or by disrupting visual-vestibular interactions or the corollary discharge mechanism. Most recently, there is evidence that poor motion detection is a compensatory mechanism for chronic oscilopsia: if the system cannot compensate for self-produced motion, then a reduction in the perception of all motion is a possible solution.

Cerebellar lesions

Nawrot and Rizzo [55] found a motion-processing deficit in a dozen patients with acute midline cerebellar lesions. Lesions confined to lateral cerebellar hemispheres did not produce a deficit. In follow-up, these deficits did not improve in testing two years later [56]. The investigators also note that the akinetopsic patient LM's extensive bilateral lesions also include a right cerebellar lesion affecting midline structures [24].

More diffuse cerebellar disease and degeneration also may impair motion processing. Ivry and Diener [57] found patients showed increased performance variability in a velocity discrimination task. Thier et al [58] also found elevated thresholds in several tests of motion perception.

A role for the cerebellum in sensory acquisition and discrimination is supported by a recent fMRI study. Gao et al [59] showed greater cerebellar activation during sensory tasks than during motor tasks. Underlying a cerebellar role in motion perception and discrimination are motion-selective neurons in the midline cerebellar vermis. Similar to neurons in cortical motion-processing areas MT and MST, cerebellar neurons have broad receptive fields and show direction, velocity selectivity, and even binocular disparity selectivity [60–63]. These neurons may interact with MT/MST neurons via the cortico-ponto-cerebello-thalamo circuit and may serve a role in perceptual stabilization during observer movement. As visual, vestibular, and eye movement information converge in the cerebellum, the role of each in cerebellar motion perception deficits remains to be determined.

Midbrain lesions

Perhaps illustrating a relation to the cerebellar lesion effects on motion perception, Heide et al [64] report two patients with a vertical gaze palsy resulting from bilateral tegmental lesions that also showed a specific deficit in judging vertical motion. Horizontal motion and vernier acuity for vertical and horizontal lines was normal. Neuroimaging showed no damage to lateral geniculate nucleus, cortex, or superior colliculus. Explanations relying on eye movements or oscillopsia were ruled out by stationary eye- and head-testing conditions. Heide et al [64] suggest the motion perception deficit was the result of an adaptive mechanism to overcome oscillopsia. That is, by making the visual system less sensitive to motion, fewer ineffective eye movements are elicited, which reduces the oscillopsia. Dieterich and Brandt [65] suggest a similar mechanism to explain a deficit in motion perception resulting from an ocular motor palsy.

Vestibular dysfunction

The same conditions of oscillopsia occur with vestibular dysfunction. If the eyes fail to move in compensation for head movement because of an ineffective vestibular ocular reflex (VOR), the point of fixation moves on the retina and the entire visual scene appears to move and bounce with each head movement. If the perception of motion is reduced, the subjective oscillopsia similarly is reduced. Exploring this possibility, Grünbauer et al [66] found that patients with bilateral vestibular failure show impaired motion perception even with stationary heads and eyes. This result is consistent with a suppressed central motion-processing mechanism

sensitivity, which reduces perceived movement for moving and stationary observers and reduces oscillopsia in moving observers.

Together the deficits from cerebellar, midbrain, and vestibular damage indicate a much broader network for the perception of visual movement and integration with head, body, and eye movements. Understanding the neural mechanisms underlying the corollary discharge/efferent copy system is an important part of understanding how the brain processes motion.

Amblyopia

Several studies have suggested that the visual deficit in amblyopia may be accompanied by a motion-perception defect [67–69]. For instance, using a motion perimetry paradigm, Donahue et al [70] found that patients with anisometric amblyopia required that a moving patch be more than 50% larger to be identified in their affected eye. An abnormal motion aftereffect in the affected eye [71] and poor interocular transfer of the motion aftereffect [72] further demonstrate abnormal motion perception. Giaschi et al [73], moreover, found abnormal perception of form from motion in normal and affected eyes of patients with amblyopia. Although the perception of depth from binocular stereopsis is compromised in these observers, Thompson and Nawrot [74] also found deficits in the perception of depth from motion parallax in patients with amblyopia. Using their normal eye for the task, patients with amblyopia had difficulty perceiving motion parallax. One possible explanation is that together, abnormal motion perception and abnormal stereopsis interfered with the normal function of neurons, showing responses to motion and binocular disparity.

Other motion perception deficits—degenerative diseases

Several neurologic diseases have produced deficits in motion perception. These perceptual deficits suggest a disconnection of the neural networks required for motion perception. For instance, whereas dyslexia is known as a reading disorder, recent investigations suggest abnormal magnocellular visual pathway processing [75]. This magnocellular deficit provides input to the cortical motion-processing area and is believed to be the cause of the motion perception deficit found in dyslexia [76] and the reduced activity measured in inferior temporal sulcus by fMRI in response to a motion-perception task [77]. Whereas dyslexia is believed primarily to affect subcortical sensory pathways involved in temporal processing and motion perception, a few other diseases may help our understanding of cortical mechanisms of motion perception.

Alzheimer's disease

Motion perception is one of many visual functions to be explored in patients with Alzheimer's disease (AD) [78–81]. As a result of the variability

of patients in progression of the disease and associated cognitive and behavioral deficits, a clear picture of the effect of AD on motion perception is still emerging. Several studies [82–84] have found that patients with AD performed worse than age matched controls in direction coherence tasks of motion perception. Mendola et al [78], however, found normal speed discrimination in patients with AD and Silverman et al [83] reported that patients with AD had preserved optokinetic nystagmus. Whereas it is unclear what pattern of brain damage generates such results [85,86], the most influential idea comes from Hof and colleagues [87–89]. Although AD produced few tangles in area 17, it did produce significant cell loss and a disconnection between early visual processing areas and area MT. That is, information is being degraded or lost as it as it passes along the processing hierarchy.

More recently, it has been proposed that a visual variant of AD exists that has more prominent posterior cortical atrophy, affecting occipito-parieto-temporal regions involved in motion perception and other visual spatial processing [90]. Studies suggest that more complex forms of motion processing are affected. For instance, Rizzo and Nawrot [91] found mildly elevated thresholds in a motion coherence task, but the leptokurtic distribution of patients with AD was not significantly different from controls. Patients with AD were significantly worse in the perception of SFM in random-dot displays, however. Using a signal/noise procedure, patients with AD required twice as much signal as did control patients to recover 3-D shape from motion. Similarly, Duffy et al [92] and O'Brien et al [93] found only a small difference between controls and patients with AD in a simple linear motion perception paradigm. Patients with AD, however, were significantly worse at using motion information in broad optic flow fields. This optic flow task requires integration of motion information over a wide visual field. It is reasonable to believe that a loss of posterior cortex neurons, with its broader receptive fields, could interfere with performance of this task.

Schizophrenia

A link between impaired motion perception and the well-known low smooth pursuit gain is emerging in schizophrenia. Stuve at al [94] measured slow eye movement gain in schizophrenia patients. Highly correlated with low gain values were high motion perception thresholds for detecting coherence direction of flow in a signal/noise paradigm. Chen at al [95] found similar results studying aspects of motion perception and eye movements. Using a velocity discrimination paradigm and measuring the accuracy of the open-loop slow eye movement acceleration, Chen et al [95] found the same (-0.60) correlation as did Stuve et al [94]. Although the identical correlation value can be coincidence, these studies suggest that area MT, subserving motion perception and slow eye movement gain, may be compromised in schizophrenia.

Williams syndrome

Williams syndrome is a genetic disorder causing low IQ and impaired visual spatial ability [96,97], leaving verbal, musical, and social abilities intact. Children with Williams syndrome tend to have abnormal binocular vision and reduced stereo acuity [98,99]. Atkinson et al [100] found that children with Williams syndrome show a deficit in motion coherence tasks, but perform normally on a static form coherence task. These visual deficits may be linked to abnormal dorsal forebrain and cerebellar vermis development [101]—the regions of the brain involved in motion perception. These abnormalities include increased gyrification in right occipital and right parietal regions [102] and right occipital volume loss [103] resulting from increased cell size, but reduced neuron density [104]. Even the cerebellar vermis, damage to which results in motion-perception abnormalities [55,56], is abnormal as it is enlarged compared with normal brains [105].

The relationship between structural and functional abnormalities, however, is variable. Children with Williams syndrome have normal perception of biologic motion [106]. Functional imaging shows that biologic motion is processed more commonly in the lateral right hemisphere region just anterior to MT and in the medial cerebellum [107]. Down syndrome, moreover, another genetically determined form of mental retardation that does not specifically affect the same brain regions as Williams syndrome, produces motion-perception abnormalities. For instance, Fox and Oross [108] found that young adults with Down syndrome performed much more poorly than control subjects in the detection and localization of motion-defined forms in random-dot cinematograms. Perhaps both forms of mental retardation affect the neural connectivity required for normal motion perception similar to the neural disconnection and perceptual deficit seen in AD.

Nefazodone toxicity

Horton and Trobe [109] reported two cases of transient akinetopsia resulting from nefazodone (Serzone) toxicity. Nefazodone is a newer selective serotonin reuptake inhibitor (SSRI) antidepressant that also blocks 5-HT_2 receptors. Although motion perception was not investigated with standard psychophysical techniques, these patients reported a subjective loss of visual motion perception. These reports also include visual trails behind moving objects and polyopia or palinopsia: seeing multiple "freeze-frame" images of moving objects even after the object has moved from view. Further investigation using standard psychophysical techniques is required to document an objective measurable change in patients taking 5-HT_2 blocking drugs and to clarify if these drugs have the potential for disrupting motion perception.

The investigators suggest that cortical neurons involved in the perception of motion, perhaps neurons in human MT, may rely on serotonin as a neurotransmitter or as a neuromodulator. Given the role of midbrain and cerebellum in motion perception and the known neuromodulatory effect of

serotonin there [110], action at other sites in the motion-processing network cannot be ruled out.

Depth from motion

Motion is an important source of information about depth. Often termed structure from motion (SFM), a moving pattern of two-dimensional dots can give rise to a percept of a 3-D rotating figure. This moving pattern of dots usually is constructed by a computer model of a rotating figure, and the dots are drawn on the flat face of a computer monitor. Although vivid, the perceived depth is ambiguous, meaning the figure may appear to rotate in either direction, often fluctuating between the two distinct percepts. The perception of depth from motion alone, however, underscores the importance of depth from motion. Recent results from the monkey suggest that area MT has a role in SFM [111]. Even more, MT has a role in the perception of depth from stereopsis [112], providing an integrated neural processing site for the perception of depth from motion and stereopsis [113].

The perception of 3-D SFM seems to be related to motion perception deficits measured with random-dot stimuli depicting a flat moving surface. For instance, patients LM [26], AF [114], and SF [30], having elevated motion-perception thresholds, could perform a 3-D SFM task. As with control observers, LM reported spontaneous reversals in the direction that the figure appeared to rotate, suggesting her experience was similar to that of control patients. As with motion perception, however, their performance declined when a large proportion of randomly moving noise dots was added to the SFM stimulus. Similarly, patients with AD [91] had significantly increased thresholds for the perception of SFM, even though their motion perception thresholds were no different from those of controls. As with an explanation for the AD decline in perception of optic flow, it is possible that SFM requires integration of motion information over greater regions than translational motion tasks and that any disruption of processing within this broad region adversely affects performance on the SFM task.

Motion parallax, another type of depth from motion, is produced by observer movement through the environment and produces a vivid, unambiguous depth percept. As the observer moves, objects at different distances from the observer move in relation to each other and in relation to the observer's point of fixation. The visual system uses this relative movement of objects to recover the relative depth of objects in the scene.

There have been few studies of deficits in the perception of depth from motion parallax. Thompson and Nawrot [74] found that a group of patients with amblyopia had much higher thresholds on a motion parallax task than did controls. These investigators concluded that a compensatory hypothesis is incorrect: patients with amblyopia do not have good depth from motion to compensate for their lack of depth from stereopsis. Instead, these results suggest that stereopsis and motion parallax are processed by the same neural

mechanisms, which, as a result of amblyopia, fail to develop properly for the perception of depth from stereopsis or motion parallax.

Depth from stereopsis

Binocular stereopsis is an important source of depth information. Random-dot stereograms are useful for studying stereopsis as depth information, providing that object shape (for example) (Fig. 4) is not visible to either eye alone. For stereopsis, information from the two eyes travels separately, but in close parallel, to adjacent layers of the lateral geniculate nucleus and to adjacent ocular dominance columns in layer IV in primary visual cortex. In neighboring layers within primary visual cortex, the information from the two eyes comes together, producing binocular neurons

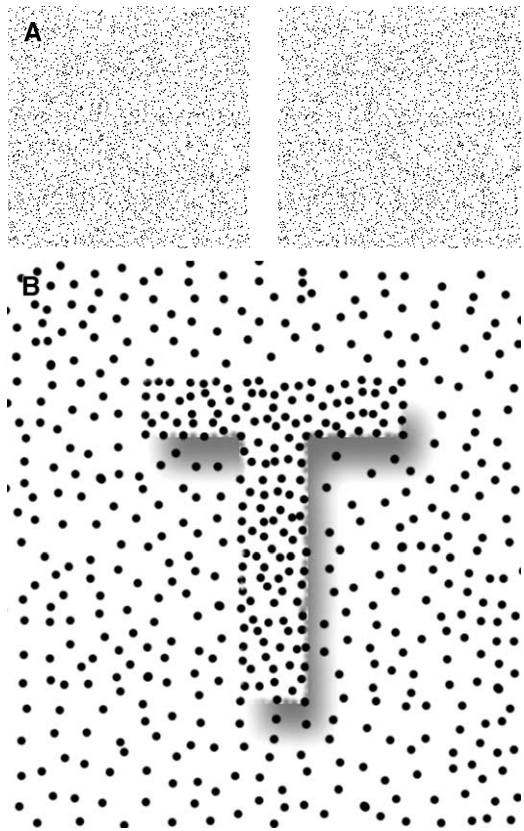

Fig. 4. Random-dot stereograms have separate images for the left and right eyes (*A*). No shape is visible in either image alone. When the images are viewed with a stereoscope, which aligns the two images with the two eyes, and the observer has normal binocular stereopsis, however, then a shape becomes visible to the observer. This visibility is depicted in (*B*) using shading and texture cues.

with disparity selectivities, a preference for objects to be in slightly different positions as viewed by each eye. These disparity selectivity neurons also are found in extrastriate visual cortex, including area MT [115,116]. Schiller [117], however, found that MT lesions in the monkey did not produce significant deficits in binocular stereopsis.

In humans, peripheral defects such as strabismus and amblyopia disrupt the perception of depth from binocular stereopsis [118,119]. These peripheral defects have an ultimate cortical effect by disrupting the development of normal binocular cortical neurons. Even if the peripheral deficit is corrected, as with aligning the two eyes, the deficit in binocular vision remains because the cortical neurons have not developed so that they integrate information from the two eyes.

A loss of stereopsis and depth perception, however, as a result of cortical damage is much less common [119], and there is little agreement on the specific cortical regions involved. Several studies suggest a dominant role for the right hemisphere in the perception of depth from stereopsis [120–122]. Ptito et al [123] found right-hemisphere anterior temporal lobe lesions can produce a deficit in global or coarse stereopsis, leaving local or fine stereopsis intact. This pattern is similar to the stereo blindness that Richards [124] described in a small proportion of control patients. In contrast, Rizzo and Damasio [125] found that the largest stereo deficits were produced by bilateral lesions of more superior, occipitoparietal cortex, rather than in more inferior occipitotemporal cortex. This result suggests that stereopsis is the result of neural processing throughout the striate cortex, with a bias towards parietal lobe and its visuospatial function [126].

Summary

Damage to the human homologue of area MT produces a motion perception deficit similar to that found in the monkey with MT lesions. Even temporary disruption of MT processing with transcranial magnetic stimulation can produce a temporary akinetopsia [127]. Motion perception deficits, however, also are found with a variety of subcortical lesions and other neurologic disorders that can best be described as causing a disconnection within the motion processing stream. The precise role of these subcortical structures, such as the cerebellum, remains to be determined.

Simple motion perception, moreover, is only a part of MT function. It undoubtedly has an important role in the perception of depth from motion and stereopsis [112]. Psychophysical studies using aftereffects in normal observers suggest a link between stereo mechanisms and the perception of depth from motion [9–11]. There is even a simple correlation between stereo acuity and the perception of depth from motion [128]. Future studies of patients with cortical lesions will take a closer look at depth perception in association with motion perception and should provide a better understanding of how motion and depth are processed together.

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