# The role of human ventral visual cortex in motion perception

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#### SUMMARY

The perception of visual motion has long been attributed to the selective function of the dorsal visual stream. Here, using fine-grained psychophysical assessment of non-form motion coherence, motion detection and complex structure-from-motion, we examined the contribution of the ventral visual stream to visual motion perception by assessing the performance of five neuropsychological patients with a circumscribed lesion to either right or left ventral cortex. Relative to controls, patients with a right, but not a left, hemisphere ventral lesion evinced widespread impairments in motion perception, and this held true independent of MT/V5 integrity. In contrast with a more traditional view in which human MT/V5 works in coordination with parietal sensory cortices to support motion perception, these novel findings implicate a more distributed circuit in which the integrity of ventral cortex is necessary for the perception of non-form as well as form-based visual motion.

Running title Ventral visual cortex and motion perception

## <u>Highlights</u>

- A lesion to right ventral visual cortex impairs motion perception
- The impairment affects perception of both non-form and form-based motion
- A lesion to the left ventral visual cortex does not impair the perception of motion
- The hemispheric differences occur independent of MT/V5 integrity

#### **INTRODUCTION**

The ability to perceive motion is fundamental to many aspects of visual behavior, and yet, surprisingly, the neural mechanisms necessary to subserve this ability remain elusive. While visual motion supports dorsal stream activities such as directing attention in the visual environment, it also supports ventral stream functions such as segmenting a moving object from its surrounding background. Despite the diverse and multifaceted roles played by visual motion perception, investigation of the neural mechanism of motion perception has largely been restricted to investigations of dorsal stream function especially in the context of attention and eye movement mechanisms.

In humans, the presence of visual motion in a scene activates motion-sensitive cortical regions including MT/V5 and MST, which are homologues of the macaque's motion sensitive regions (e.g. (Heeger et al., 1999; Kolster et al., 2010; Rees et al., 2000; Vanduffel et al., 2002)) and these regions are sensitive to motion anywhere in the visual field,(e.g. (Albright et al., 1984; Huk et al., 2002; Kolster et al., 2010; Maunsell and Van Essen, 1983b; Newsome and Pare, 1988; Tootell et al., 1995; Weiner and Grill-Spector, 2011; Zeki, 1980; Zeki et al., 1991; Zeki, 1974, 1978)).

Given the ubiquitous engagement of these MT/MST regions in representing visual motion, it is not surprising that their functional integrity is essential for normal motion perception, and evidence gleaned from monkey lesion studies (Newsome and Pare, 1988) and other physiology studies (Bradley et al., 1998; Celebrini and Newsome, 1994; Grunewald et al., 2002; Heeger et al., 1999; Newsome et al., 1989; Salzman et al., 1992; Zohary et al., 1994) offers confirmatory evidence. Support is also garnered from studies of patients with akinetopsia (also termed "motion blindness", see review (Zeki, 1991)) who, following bilateral lesions to MT/V5, perceive the visual world around them as a sequence of static snapshots rather than a

smooth sequence of moving images (Marcar et al., 1997; McLeod et al., 1996; Zihl et al., 1983; Zihl et al., 1991). Unilateral lesions to these motion-sensitive regions also adversely affect motion perception, albeit just in the contralateral visual field (Schenk and Zihl, 1997; Vaina et al., 2001). While these findings clearly indicate the necessary engagement of motion-sensitive areas in mediating motion perception, these motion sensitive areas, in and of themselves, may not suffice for normal motion perception (Hedges et al., 2011; Majaj et al., 2007).

Although motion perception is commonly considered a property of dorsal, rather than ventral, stream function (Newsome, 1997; Treue and Maunsell, 1996; Ungerleider and Desimone, 1986), motion-sensitive regions (e.g. MT/MST in humans) are anatomically located in lateral temporal cortex, at the intersection of the dorsal and ventral streams (Goodale and Milner, 1992; Ungerleider and Mishkin, 1982). Indeed, ventral visual cortex is also responsive to visual motion (Desimone and Schein, 1987; Ferrera et al., 1994; Grill-Spector et al., 1998; Kriegeskorte et al., 2003; Mountcastle et al., 1987; Murray et al., 2003; Peuskens et al., 2004; Sary et al., 1993; Vanduffel et al., 2002) although this responsivity is commonly attributed to the form defined by the motion rather than to the motion for itself. Detailed scrutiny of the data, however, indicate that ventral visual cortex does actively participate even in basic (non-form) motion processing and, as such, may play a functional role in motion perception, more generally. For example, electrophysiological studies reveal that a non-negligible portion of V4 neurons (between 13-33%) are sensitive to motion direction (Desimone and Schein, 1987; Ferrera and Maunsell, 2005; Ferrera et al., 1994; Mountcastle et al., 1987; Tolias et al., 2005) and optical imaging studies indicate that V2 and V4 contain a motion direction map ((Lu et al., 2010); Li, Chen, Han, Zhu, Xu, Roe, Lu. Soc Neurosci (2011)). Of note, there is also direct

connectivity between motion sensitive MT/V5 and ventral cortex revealed by fluorescent dye tracings in non-human primates (Maunsell and Van Essen, 1983a; Ungerleider and Desimone, 1986), attesting to the interactivity in these regions.

Thus, whereas ventral visual cortex actively participates in motion processing, whether its functional contribution to motion perception is restricted to form processing or is more generally associated with motion perception remains unknown. While functional imaging studies have been helpful in shedding light on the mechanism subserving motion perception, they do not necessarily inform us of the necessity or sufficiency of ventral visual cortex for this ability. As such, the relationship between cortical activation and behavior remains correlational rather than causal.

To complement the electrophysiological and imaging studies, here, we elucidate the contribution of ventral visual cortex to motion perception by examining the visual motion abilities of five individuals with focal damage affecting the ventral visual cortex on either the right or the left side. These patients, some of whom have preserved MT/V5, were tested several years after lesion onset, and their performance was compared to that of age- and gender-matched control groups. To explore brain-behavior correlations and determine whether motion perception deficits, if present, might be solely attributable to lesioned MT/V5, we delineated the lesion site based on structural imaging data.

Because motion perception is involved in a range of perceptual tasks (e.g. (Gilaie-Dotan et al., 2011; McLeod et al., 1996; Murray et al., 2003; Newsome and Pare, 1988)), some of which engage form representations (for example, perceiving structure-from-motion) but some of which do not (merely detecting the presence of motion in a display), we examined whether different types of motion perception were

equally affected by the neural insult (Hedges et al., 2011). In sum, we assessed performance in basic motion tasks (motion coherence or detection (Albright and Stoner, 1995; Newsome and Pare, 1988; Rees et al., 2000; Stoner and Albright, 1992; Thiele and Stoner, 2003)) and in more complex motion tasks (three dimensional structure-from-motion (SFM) (Gilaie-Dotan et al., 2011; McLeod et al., 1996; Murray et al., 2003; Vaina et al., 1990)).

Furthermore, given that four of the five patients had a focal unilateral lesion, we had the unique opportunity to contrast the relative contribution of the hemispheres to motion perception and to elucidate whether the right and left hemispheres mediate motion perception equivalently.

We hypothesized a priori that, if the ventral visual cortex plays a necessary role in motion perception, then a lesion to this region would impair motion perception across all tasks. Moreover, this should hold true even if MT/V5 regions were intact. Alternatively, if ventral visual cortex is only engaged when motion contributes to form knowledge, then a ventral visual lesion would only impair motion perception when form representations are invoked (e.g. by structure from motion stimuli), but not when basic motion perception is tapped (e.g. motion coherence or motion detection). Finally, if the hemispheres contribute asymmetrically to motion perception, then a right hemisphere lesion might impair motion perception to a greater degree than a left hemisphere lesion because of the critical role of the right hemisphere in form perception (Konen et al., 2011).

----- Insert Table 1 about here -----

#### RESULTS

#### Motion perception behavioural performance

To assess the contribution of the ventral stream to motion perception, we compared the performance of five patients to specifically selected controls (see Table 1 for biographical and lesion details) on a series of motion perception tasks that have proven effective in uncovering impairments in motion perception (Gilaie-Dotan et al., 2011; McLeod et al., 1996; Milne et al., 2002; Spencer et al., 2000; Vaina et al., 1990). These tasks ranged from basic motion perception tasks such as perceiving motion coherence (e.g. (Newsome and Pare, 1988; Rees et al., 2000; Stoner and Albright, 1992; Thiele and Stoner, 2003) and detecting motion (Albright and Stoner, 1995), both of which are usually attributed to basic motion perception mechanisms. We also assessed the patients' perception of more complex structure-from-motion (SFM) (Gilaie-Dotan et al., 2011; McLeod et al., 1996; Murray et al., 2003; Singer and Sheinberg, 2008; Vaina et al., 1990), a process attributed to both motion perception mechanisms and to structure perception mechanisms (Grill-Spector et al., 1998; Peuskens et al., 2004; Sary et al., 1993).

#### **Motion coherence**

To characterize sensitivity to motion coherence, we calculated the proportion of coherently moving dots required by the participant to detect the direction of coherent motion embedded in dots moving in random directions (Green, 1961; Levinson and Sekuler, 1976). Further details of the methods and of additional motion coherence control paradigms and results are provided in Supplementary Material.

The motion coherence thresholds for the two left ventral visual patients were normal (see details in Table 2 and Figure 1A). In contrast, the motion coherence thresholds for the patients with right ventral visual lesions were significantly impaired, with thresholds 3-4 times higher than those of the matched controls (see Table 2, Figure 1A). We were not able to obtain motion coherence thresholds from EC as she was no longer available for testing.

 Insert Figure 1 about here
 Insert Table 2 about here

#### **Motion detection**

Further exploration of the patients' basic motion perception skills was done by having participants detect the motion of a coherently moving cluster of dots embedded in the centre of a random dot flickering field (Gilaie-Dotan et al., 2011). The task was conducted under two conditions: in a fast motion condition (Fst) in which a few fast-moving dots defined the motion, and in a slow motion condition (vSlw), when many slow-moving dots defined the motion (see Experimental Procedures for full details). In the easy, fast-motion condition, when the motion cues are robust, all patients and their controls were at or close to ceiling (see Table 3 for details). In the more difficult, slow-motion task, whereas the left ventral patients were unimpaired (see vSlw in Table 3), the patients with a right ventral visual lesion were all significantly impaired (see Figure 1B and Table 3).

----- Insert Table 3 about here -----

#### **3D** structure-from-motion

The evidence above indicates that the right-lesioned individuals were impaired at basic non-object motion perception (except under very simple conditions as in the fast moving dots), but that the patients with left lesions performed normally. In this experiment, we assessed the patients' recognition of three-dimensional structures (sphere or cylinder) based on motion cues alone (termed structure-from-motion, SFM (Gilaie-Dotan et al., 2011)). Figure 1C depicts the 3D structure-from-motion recognition results (see Table 4 for full details). Recognition was at ceiling for all the patients and controls when the motion was fast and many dots defined the form (1600pnt condition). The left ventral patients' recognition was unimpaired (and actually at ceiling) even for the most difficult condition (100pnt). The right ventral patients' recognition impairment, however, was revealed as less structural information was available in the display (i.e. fewer points defining the rotating structure, 500pnt or 100pnt). For the most difficult condition (100pnt), all the right ventral patients were significantly impaired in recognizing the rotating form (Fig. 1C), despite almost ceiling performance in detecting the local motion in such a display (accuracy between 95%-100% for motion detection, see Fst condition in Table 3 with the same local motion and dot density as the 100pnt condition). That the right ventral patients had normal detection ability of fast motion (despite being impaired in recognizing the structure defined by that motion) might be explained by the presence of the robust local motion cues available in the display. These local motion cues could easily be detected, but the recognition of a coherent shape likely requires additional integration of these motion cues (Singer and Sheinberg, 2008). Two of the three right ventral visual patients have previously been shown to be impaired at integrating local information to form more global shape configurations (Behrmann and Kimchi, 2003).

In sum, the left ventral patients performed within normal limits across the board. In contrast, the right ventral patients displayed impaired recognition of form from 3D motion even when they were clearly able to detect the local motion cues (see Motion Detection above).

----- Insert Table 4 about here -----

#### **Lesion comparisons**

The behavioral findings implicate the right, but not the left ventral cortex, in normal motion perception, and even in non-object basic motion perception (motion coherence and motion detection). One potential, simple explanation for this result might be that the lesions in right but not left hemisphere patients impinge on the motion sensitive region MT/V5. To rule out this possibility, we delineated each patient's lesion based on previously acquired anatomical images (Figure 2) and situated the lesion relative to the expected location of MT/V5 (Kolster et al., 2010). For SM, EL, and CR, high-resolution MR anatomical images were used and, as such, provided continuous spatial coverage across the brain. The best anatomical images available for GB and EC were from clinical scans, thus lacking full spatial coverage and optimal resolution of the brain, but still permitting sufficient precision for lesion demarcation. Lesion delineation was performed in native space and the images were then transformed into normalized Montreal Neurological Institute (MNI) space for comparison with the anatomical location of the middle temporal motion-sensitive region MT/V5 as reported in the literature ((Kolster et al., 2010; Watson et al., 1993), see Experimental Procedures).

The process of lesion delineation and the subsequent lesion siting for each of the patients are depicted in Figure 2, and Table 5 provides a summary of the lesion volume, and determination of whether or not each lesion overlapped MT/V5.

In the individuals with left ventral lesions (EL and GB), the lesions were extensive, while in the right lateralized patients, the lesions varied both in size (SM small, EC extensive, CR scattered) and location along the ventral stream. Importantly, however, neither the size of the lesion nor its location with respect to MT/V5 were correlated with the impairment in motion perception: as evident from Table 5, despite EL's extensive *left* ventral lesion, probably overlapping left MT/V5, EL's motion perception was normal on all tasks, from basic motion perception to more complex structure-from-motion tasks. GB's motion perception was also normal on all tasks despite an extensive left ventral lesion. In contrast, SM, who has a small lesion in the *right* ventral cortex with sparing of right MT/V5 (and normal activation of MT/V5 as revealed through functional neuroimaging (Konen, Kastner and Behrmann, in prep)), was significantly impaired on all motion perception tasks. CR, who has a right ventral lesion along with some other punctate abnormalities, but spared right and left MT/V5, was also significantly impaired in all motion perception tasks. EC, who has an extensive right ventral lesion, was impaired in all motion perception tasks tested. Note that for both GB (left lesion) and EC (right lesion), we cannot definitively conclude whether MT/V5 is spared due to the low spatial coverage of their clinical structural images and so we urge caution in interpreting these two cases.

#### DISCUSSION

The goal of this study was to explore the functional contribution of the ventral visual cortex to the ability to perceive motion. Five neuropsychological patients, each with a lesion to either the right or left ventral visual cortex (note that one patient had additional punctate lesions in the other hemisphere albeit not in ventral cortex), participated in a range of psychophysical tasks examining the perception of non-object motion and the perception of object structure from motion. Two major results were obtained. First, surprisingly, in addition to the impairment in perceiving formbased motion, even basic motion perception (such as detecting the presence of motion in a display or discerning the coherence of random moving dots) was adversely affected by ventral visual lesions. Second, the perturbation in the perception of motion was only observed in those patients with right ventral visual lesions, whereas the motion perception of those with left ventral visual lesions remained in the normal range.

Our novel findings suggest that the perception of motion, even when it is not dependent on form or structure (as in simply detecting the presence of motion and coherence in a visual display) is dependent on the integrity of the right ventral visual cortex. This dramatic finding indicates that motion-sensitive areas of cortex by themselves do not suffice for normal motion perception and that additional cortical regions are required even in support of basic types of motion perception.

#### Ventral visual cortex affects motion perception

The fact that basic, non-object motion perception relies on (right) ventral stream integrity, as revealed here, runs counter to prevalent views in neuroscience. Motion inputs are considered to be processed in specialized, perhaps even dedicated cortical motion-sensitive brain regions (e.g. MT/V5, MST (Heeger et al., 1999; Kolster et al., 2010; Newsome and Pare, 1988; Rees et al., 2000; Tootell et al., 1995; Zeki, 1974)), and the activity of these regions is strongly linked to motion perception (Bradley et al., 1998; Celebrini and Newsome, 1994; Grunewald et al., 2002; Heeger et al., 1999; Newsome et al., 1989; Salzman et al., 1992; Zohary et al., 1994). However, recent studies indicate that MT/V5 responses depend on local rather than global motion, and suggest that motion perception relies on activity in multiple brain regions, not just on MT/V5 (Hedges et al., 2011; Majaj et al., 2007). Specifically, since these motion sensitive regions are more clearly associated with dorsal stream function, dorsal (rather than ventral) regions are natural candidates to be contributing to motion perception. However, this seemingly natural association between motion perception and dorsal stream function might be a consequence of experimental paradigms that tap motion perception in the context of action planning, saccadic movements or attention, all of which engage dorsal function (Newsome, 1997; Newsome et al., 1989; Treue and Maunsell, 1996). These contexts are rather limited, however, since motion perception involves many other aspects of visual perception, as well. For example, the segmentation of visual scenes is greatly aided by motion perception, whether through movements in the scene or caused by the observer's motion. It is possible, therefore, that investigations of the neural systems mediating motion perception might have been limited by the task or context, and thus, the extent to which motion perception genuinely depends on dorsal function remains unknown.

Although the ventral visual cortex is not considered a viable candidate to support motion perception, given that it is "only" a downstream (feedforward) recipient of motion signals and relevant for shape perception (Grill-Spector et al., 1998; Kriegeskorte et al., 2003; Peuskens et al., 2004; Sary et al., 1993), various

sources suggest otherwise. First, ventral visual cortex itself is motion sensitive. Electrophysiology in macaque reveals that ventral cortex consists of a direct motion selective pathway from V1 to V2 that bypasses MT/V5 (Gur and Snodderly, 2007), that 10%-30% of V4 neurons are direction selective (Desimone and Schein, 1987; Ferrera et al., 1994; Mountcastle et al., 1987), and that V4 is sensitive to changes in motion direction (Tolias et al., 2005). Furthermore, optical imaging shows that macaque V2 and also V4 contain a columnar organization of motion directional maps in the foveal aspects of its representation (5° visual angle (Lu et al., 2010); Li, Chen, Han, Zhu, Xu, Roe, Lu Soc Neurosci (2011)). Second, direct bilateral connectivity and inter-dependency exists between ventral stream and motion sensitive regions (e.g. between MT/V5 and V2, V4 (Maunsell and Van Essen, 1983a; Ungerleider and Desimone, 1986)), and a relatively new motion sensitive region, discovered in the ventral aspects of macaque STS, suggests an additional motion-sensitive processing route (Nelissen et al., 2006). Finally, area MT/V5, the pre-eminent motion area, shows sensitivity to *static* object shape in the presence and even in the absence of implied motion cues (Kourtzi et al., 2002; Kourtzi and Kanwisher, 2000). Taken together, these findings implicate direct and probably critical involvement of ventral stream in motion processing. Here, we have demonstrated, for the first time, that ventral stream is necessarily engaged in motion perception, and importantly, that this engagement is not simply in the service of form perception in motion displays, but is required for the perception, and even detection, of basic motion information.

There is, however, an alternative, simpler explanation that needs to be addressed and that is, that the impairment in motion perception following ventral lesions, as documented here, might result from compromised white matter tracts providing inputs to MT/V5 rather than from the ventral lesion itself. This does not

seem to be the case here. First, the locations of the lesions in the three patients with right ventral lesions (and impaired motion perception) differed along the ventral stream, and while SM's lesion was close to the location of MT/V5, CR's lesion was far more anterior and not even in the vicinity of MT/V5. Second, according to our lesion delineation, but also as revealed by fMRI data, SM evinces a normal pattern of motion-selectivity in the standard MT/V5 regions (Konen, Kastner and Behrmann, in prep). Taken together, these results indicate that ventral stream is not a recipient of motion inputs from motion sensitive regions, but is likely an active and critical player in the motion processing network supporting motion perception.

How can we then reconcile the critical role of the ventral visual stream in motion perception, as we have shown here, with the previously reported critical role of MT/V5 (Newsome and Pare, 1988)? Both sides of this equation require further explication, especially when EL, whose lesion implicated left MT/V5, was not impaired in motion perception, whereas SM, with undamaged bilateral MT/V5 was impaired in motion perception. We suggest two possible resolutions to this paradox. The first is based on the idea that intact motion perception depends on both normal central and peripheral motion perception. We speculate that while motion-sensitive visual regions such as MT/V5, MST, and dorsal stream are critical for peripheral normal motion perception covering the whole visual field, ventral visual cortex is critical for *central* motion perception. According to this division of labor, lesions to right ventral cortex would give rise to central field motion perception impairments, while lesions to MT/V5 would cause contralateral visual field motion perception impairments, perhaps sparing central motion perception. SM, with uncompromised right and left MT/V5, is impaired in *central* motion perception following his right ventral visual lesion. Despite her lesioned left MT/V5, EL's central visual motion

perception is normal, presumably due to her undamaged right ventral visual cortex. Half field akinetopsia due to contra-lateral MT/V5 lesion can be attributed to impaired *peripheral* motion perception as impairments are more evident with off-center stimuli (Newsome and Pare, 1988) and, indeed, LM's bilateral motion blindness is more apparent in the periphery (Marcar et al., 1997; McLeod et al., 1996; Zihl et al., 1983; Zihl et al., 1991). A hypothetical scenario to be tested in the future for the division of labor between the streams would be that the dorsal stream detects a moving object across the visual field, and frontal eye fields swiftly direct eyes on the moving object. Ventral stream motion processing becomes critical then once the moving object is in foveal view. Interestingly, none of our patients report suffering from motion perception deficits. Along with our hypothesis, a motion perception deficit in central visual field following a ventral lesion could subjectively be attributed to the structure perception deficits, whereas a motion perceptual deficit across the visual field (in the absence of a form deficit) is subjectively recognized as a motion perception deficit. A second possible resolution suggests that while MT/V5's role is processing the basic building blocks of motion stimuli, right ventral cortex spatially integrates basic motion cues into holistic moving percepts such as surfaces or objects. Further evidence and testing will be necessary to accept or refute these theoretical alternatives.

#### Differential hemispheric contributions to motion perception

Not only do our findings, for the first time, implicate ventral cortex in motion perception, but they suggest a hemispheric asymmetry in this process as the perceptual impairment was evident only following a right hemisphere lesion. We speculate that bilateral human motion–sensitive regions such as MT/V5 and MST (Heeger et al., 1999; Kolster et al., 2010; Rees et al., 2000; Tootell et al., 1995) may

well process motion equivalently, and that the laterality effect we have observed is related to right hemisphere dominance in form and object perception. Although form and object representations appear to activate both hemispheres (Gilaie-Dotan et al., 2010; Gilaie-Dotan et al., 2008), neuropsychological studies suggest that the right ventral cortex plays a more critical role in object recognition (Barton, 2011; Davidoff and Warrington, 1999; Humphreys and Riddoch, 1984; Konen et al., 2011). Left ventral lesions do not typically give rise to profound agnosia and tend to result in deficits in visual word recognition (Behrmann et al., 1998; McKeeff and Behrmann, 2004). This asymmetry is supported in our patient sample, with the right ventral patients more profoundly impaired in object recognition than the left ventral patients (Behrmann et al., 2006). Thus, we speculate that motion perception is tightly linked to structure and object perception and therefore, the right ventral visual dominance for object and structure perception might serve as the basis of the motion perception laterality effect we have observed in this study.

#### CONCLUSIONS

Our study reveals that a wide range of motion perception skills is dependent on ventral visual cortex integrity, and, specifically, on the integrity of the right ventral visual cortex. This finding suggests a tight interplay between ventral visual cortex and motion perception, that has not been observed to date, and, hence, challenges the received view that dorsal and motion sensitive regions suffice for normal motion perception. The results also license an account in which normal motion perception is subserved by distributed functional circuits rather than by circumscribed regions.

#### **EXPERIMENTAL PROCEDURES**

#### **Participants**

All participants gave written informed consent to participate in the study and the protocol was approved by the Institutional Review Board, Carnegie Mellon University and by the UCL local ethics committee. All five patients were tested in Pittsburgh and control participants were tested in the same setting or in London. Patients were tested either at the university or in their home.

#### Patients

Five premorbidly normal individuals, all of whom were right handed, participated in these experiments. Following a lesion sustained in adulthood (except for CR who was aged 16 years at lesion onset), all individuals reported visual perceptual problems. Table 1 summarizes basic aspects of the patients' case description; more detailed information is available in Supplementary Material and in previous publications (SM (Behrmann and Kimchi, 2003; Behrmann and Williams, 2007; Gauthier et al., 1999; Konen et al., 2011; Marotta et al., 2001; Nishimura et al., 2010); CR (Behrmann and Williams, 2007; Gauthier et al., 1999; Marotta et al., 2001); EL (Behrmann et al., 1998; McKeeff and Behrmann, 2004; Montant and Behrmann, 2001; Mycroft et al., 2009); GB (Behrmann & Plaut, submitted manuscript)). Aside from EL, who has an upper right visual field quadrantanopsia, the other patients all have full visual fields. In spite of her field defect, EL performed normally on all motion perception tasks.

#### **Control participants**

A group of 22 control participants completed this study: 11 male control participants served as age-matched controls for CR (mean age 31.36 years  $\pm$  3.2 (S.D.)), nine males served as controls for SM (mean age 35.89 years  $\pm$  3.86, six of whom were matched for both CR and SM), eight females and one male (matched for SM as well) served as controls for EC (mean age 48.22 years  $\pm$  3.53). The control group for GB and EL was composed of four women (aged 58-66, mean 63  $\pm$  3.56). All control participants were right-handed, had normal or corrected-to-normal vision and no history of neurological disorders.

#### **Motion Perception Experiments**

#### Motion coherence

#### Stimuli

Non-object-based motion processing was assessed using motion coherence thresholds. The stimulus consisted of a circular random dot pattern (Green, 1961; Levinson and Sekuler, 1976) displayed in the center of the screen (half in each hemifield). Each stimulus consisted of 500 grey dots (luminance =  $2.50 \text{ Cd/m}^2$ , width = 2.77 minArc) against a black background (luminance =  $0.37 \text{ Cd/m}^2$ ) covering a circular area (width = 9.13 degrees). A two-interval forced choice design was employed. In each trial, two stimuli were presented (inter stimulus interval = 1000ms). One randomly chosen interval consisted of coherent motion plus noise and the other interval only consisted of noise. For the signal plus noise stimulus, a randomly chosen subset of the dots was vertically displaced upwards or rightwards by  $0.45 \text{ degree steps in twenty consecutive frames (total motion time= 333ms; speed =$ <math>27.27 degree/second). See Supplementary Material for additional motion coherence paradigms and results for specific directions (up, down, right and left). The remaining

dots were repositioned randomly from one frame to the next. Coherently moving dots reaching either end of the display area were repositioned on the other side for the next frame. A central fixation square (width = 0.55 deg) was displayed throughout the experiments. Viewing distance was approximately 52cm. Stimuli were generated using the Cogent toolbox (http://www.vislab.ucl.ac.uk/Cogent/) for MATLAB (Mathworks, Natick, MA, USA). Stimuli were presented at 60Hz using a TFT-LCD display (resolution = 800×600, 15.4" screen).

#### Thresholding procedure

Using the accelerated stochastic approximation method (Kesten, 1958; Treutwein, 1995), for each observer we determined the threshold coherence level that enabled 75% correct identification of target interval that contained upward motion. Each run of the staircase consisted of 48 trials.

#### **Testing Procedure**

The task was a 2AFC and participants had to press one of two buttons to indicate whether the first or second viewing interval contained more coherent motion. No time limit was imposed on responses. Participants started with a practice session with initial coherence level of 70%-90% (see Results for details) and a verbal explanation. Thereafter, participants performed additional 1-2 runs, with first run starting with an initial coherence threshold of 50%. For the second run, the input threshold was taken as the output of the previous run. The output threshold of the last session each participant performed was taken as the individual's coherence threshold. We compared the patient's coherence threshold to those of his/her corresponding control participants and determined whether performance was significantly different from that of the controls' (Crawford et al., 2009), as presented in Table 2.

#### 3D structure-from-motion

In this experiment, we assessed the patients' abilities to recognize three-dimensional objects defined by motion. We used three-dimensional objects (spheres and cylinders) defined by motion cues where the motion conveyed surface and three-dimensional structure (Figure 1 C, (Gilaie-Dotan et al., 2011)). The paradigm we used allowed the presentation of an object (sphere or cylinder) based only on the local motion vectors across the object (Singer and Sheinberg, 2008). An animated three-dimensional scene composed of a rotating object and a static background was rendered in real-time as a pattern of points. A global percept of the moving object emerged from the integration of the local motion vectors into a coherent moving shape. Since each point followed the trajectory of the underlying motion in the scene, only points located on the rotating object surface actually had local motion, while the points located "in the background" did not. Each static frame of the animation appeared to be a uniform random field of points (see Fig. 1 C, static snapshot). By varying the number of points in the display (and thus those defining the object), we were able to modulate task difficulty (see below).

#### Stimuli

The experimental display was composed of flickering white points (diameter 0.16 ° visual angle) that randomly appeared on a black screen ("formless dot field"). Each point had a short lifetime (1.33 s, 80 frames at 60 frames/sec) and the appearances of the points on the screen were not synchronized. When a trial began, the motion of the rotating object was embedded into the flickering point display, so flickering points that appeared in the object's location followed the local motion of the object's surface for the full extent of their lifetime (1.33s). When a trial ended, all the points in the display were stationary. The rotating object was a three dimensional

sphere or cylinder (Figure 1C). In each run, half of the trials were of a rotating sphere and half of a cylinder, and the order was determined randomly. The spinning object rotated around its north-south axis, which was tilted 27° away from the screen's yaxis plane (north end farther away, south end closer), similar to the Earth's tilt. The object rotation direction was determined randomly (clockwise or anticlockwise, 50% trials to each direction). The sphere and cylinder, viewed from 55cm distance, subtended a visual angle of 12°x 9.9° (width x height) and 8.2°x9.4° (width x height), respectively. Three conditions included a parametric change to the number of points composing the formless point field (1600pnt, 500pnt, and 100pnt) while the rotation speed remained constant (0.5 rotations/s, see Fig 1 C). Screen resolution was 1024x768, refresh rate 60Hz. Stimuli were presented using MATLAB (Mathworks, Natick, MA, USA) and Psychophysics Toolbox 3 (Brainard, 1997). The experimental stimuli were based on the FDFDemo and mogIFDF functions provided with the Psychophysics Toolbox 3, which provides an OpenGL (Silicon Graphics Inc.) interface for MATLAB.

#### Procedure

Participants were first familiarized with the stimuli by viewing example trials with 1600 points and rotation speed of 0.5 rotations/sec, parameters that provided easy recognition of the objects even for all the patients (see Fig. 1C, 1600pnt condition), and reported verbally what object they saw on the screen. Following this, each condition included a session of 20 trials with fixed parameters throughout the session (number of points, rotating speed). The participants' task was to press a key once the object was recognized and then to tell the experimenter, who recorded the response, which object was present. The object rotated until the response was given without time restriction. Once the participant was ready, the next trial was initiated.

Recognition accuracy was assessed for each participant for each condition. All patients and controls followed the same experimental order to rule out order-based learning effects. For each experimental condition, we compared the patient's accuracy to those of his/her corresponding control participants and determined whether performance was significantly different from that of the controls' (Crawford et al., 2009), as presented in Table 4.

#### Motion detection

Motion detection was assessed after the 3D structure-from-motion experiment described above. The stimuli in the motion detection sessions were identical to those in the 3D structure-from-motion object recognition task in all respects, except that a moving rotating object was present in only half of the trials (10 out of 20, randomly ordered within the session). In the remaining trials, there was no local motion. Therefore, the participants were all familiarized with the stimuli and the instructions were given to them verbally.

The participants had to verbally indicate by "yes" or "no" whether or not there was an object moving in the centre of the screen. The experimenter terminated the trial immediately after the verbal response and then initiated the next trial. There was no time restriction for providing responses. There were two conditions: Fst of fast motion (0.5 rotations/s, average horizontal dot motion speed of 8.2-12 deg/s) of a sparsely spaced points, and vSlw depicting very slow motion (0.0033 rotations/s, average horizontal dot motion speed of 0.055-0.08 deg/s) of densely spaced points. Motion detection accuracy was assessed for each participant for each condition, and we compared the patient's accuracy to those of his/her corresponding control participants as described above ((Crawford et al., 2009), see Table 3).

#### Patients' structural image acquisition

EL

EL's anatomical MR scans were acquired at the Brain Imaging Research Center (BIRC) Pittsburgh on a Siemens Allegra MRI 3T scanner using a head coil, when she was 60 y.o., approximately one year prior to her participation in this study and 14 years after her injury. The scan acquired 192 MPRAGE sagittal slices (1mm thickness, inplane resolution of  $1x1 \text{ mm}^2$ , matrix = 256x256, repetition time 1740ms, echo time 3.04ms, inversion time 1000ms, flip angle =8°).

#### GB

GB's MR clinical structural scans were acquired on a 1.5 T GE Genesis Signa MR scanner equipped with a head coil, approximately 3 years prior to her participation in this study. These included 23 axial T2 images (slice thickness = 5.5mm, 7mm gap, image size 512x512, pixel spacing 0.42968 x 0.42968 mm<sup>2</sup>, echo time = 96.512 ms, no. of averages = 2, flip angle = $90^{\circ}$ ).

#### SM

SM's MRI structural scans were acquired with identical parameters to those of EL's (see above) at the Brain Imaging Research Center (BIRC) Pittsburgh when he was 35 y.o. This was 17 years after his injury and approximately 2 years prior to his participation in this study (for details, see (Konen et al., 2011)).

CR

CR's MRI structural scans were acquired at the Magnetic Resonance Research Center, University of Pittsburgh Medical Center on a 1.5 T Signa whole body scanner (General Electric Medical Systems, Milwaukee, WI), approximately 3 years after he had metabolic encephalopathy and approximately 12 years prior to his participation in this study. This included 124 slices of 1.5mm thickness with an inplane resolution of 0.9375x0.9375 mm<sup>2</sup>, matrix of 256x256.

#### EC

EC's CT clinical structural scans were acquired on a GE Medial Systems LightSpeed QX/i CT scanner when she was 40 y.o., and approximately 8 years prior to her participation in this study. These included 34 axial images without contrast with slice thickness of 2.5mm (through the posterior fossa) and 7.5mm (from the posterior fossa to the vertex), 512x512 image size, and pixel spacing of 0.449219x0.449219 mm<sup>2</sup>.

#### Lesion delineation procedure

For patients with high resolution anatomical images, the images were coregistered onto a T1 MNI canonical SPM image using SPM (http://www.fil.ion.ucl.ac.uk/spm), after which their lesions were traced manually in MRIcroN (http://www.cabiatl.com/mricro/mricro, see below for tracing criteria) and saved as a binary image (see Figure 2 A,B). For each patient, the coregistered anatomical images and the demarcated lesion were normalized into MNI space using the unified normalization segmentation of SPM (http://www.fil.ion.ucl.ac.uk/spm) as shown in Figure 2 C. For GB and EC, who had low resolution anatomical images from the clinical scan, the lesion was traced manually onto the corresponding anatomical location in an MNI canonical SPM image (see Figure 2 E,F, and details below).

The locations of motion-sensitive region Left MT/V5 (L-MT/V5) and Right MT/V5 (R-MT/V5) were based on previously reported coordinates (Kolster et al., 2010). Spheres with a 10mm radius were created around these locations (Figure 2 C, F). The normalized brain and lesion, and the location of R- and L-MT/V5 were then loaded onto MRIcroN for display purposes for each patient (see Figure 2D, G)

#### Lesion tracing criteria

For EL, since the average intensity values varied across each structural image regardless of the lesion (e.g. between anterior and posterior regions, or right and left hemispheres), the definition of the lesioned tissue was not based only on absolute intensity values. Instead, the definition of the lesioned tissue also took into account abrupt local changes in intensities between lesioned and adjacent healthy tissue, and continuity of abnormal lesioned tissue. Locally, lesioned tissue always had substantially lower values of intensity than healthy tissue. In most parts of EL's brain and around the lesion, healthy tissue intensity values ranged from 200 to above 350 (a.u.), while lesioned tissue intensity values ranged from 54 to 170s. However, in specific locations 170 was considered healthy tissue (adjacent to value of 117 of a lesioned tissue). We provide lesion size estimates based on local intensity variations and continuity assessment, and also a more conservative estimation based on intensity values <150 in the predefined lesion zone (see Table 5).

With SM's structural images, we also faced the issue of local variations in intensity values; thus, as with EL, we delineated the lesion based on intensity values, continuity of the lesioned tissue, and abrupt changes in local intensity values between the lesion and adjacent healthy tissue. Common values for healthy tissue were above 200 to even above 350, however, locally healthy tissue could have value of 171. Lesioned tissue typically had values ranging from as low as 60's to values around 150, however, locally, values of 160 or 174 could be attributed to lesioned tissue. We provide the lesion size estimate according to the criteria laid out above, along a conservative estimate for lesion when lesioned intensities < 160.

Since CR's lesion is of a different etiology than that of EL and SM, the lesion delineation criteria were different. On top of a medial right temporal lobe hemorrhage, there are foci of petechial hemorrhage seen along the grey/white junction at multiple areas that appear as a very dark centre (intensity values of below 35) bordered by very bright intensity tissue (intensity above 120). Healthy tissue in CR's structural images had intensity values of 55-95.

Lesioned tissue in GB's and EC's original clinical structural images were used to guide delineation in normalized MNI. GB's original DICOM images were loaded into MATLAB (Mathworks, Natick, MA, USA) where intensity values of lesioned tissue were above 0.5, while healthy tissue intensity values were below 0.4. Due to technical issues with MATLAB reading EC's DICOM images, EC's structural images were loaded into MicroDicom (<u>http://www.microdicom.com/</u>) and then exported to bitmap images. Typically lesioned tissue intensity values were below 105, while healthy tissue values were above 110. In the lesion delineation process we also took into account (as with the other patients) continuity of the lesioned tissue, and abrupt intensity changes between the lesion and adjacent healthy tissue. After the lesion

tracing in the original images detailed above, an approximated corresponding delineation was carries out onto a single subject T1 SPM MNI-normalized template (as illustrated in Figure 2 E, F).

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## TABLES

## Table 1

Case histories summary. Further detailed description is provided in Supplemental

Material.

	Left ventral lesion		Right (+ left ?)	al lesion	
	EL	GB	CR	SM	EC
Age(gender)	61(F)	70(F)	31(M)	37(M)	48(F)
Controls' ages (number, gender)	63 ± 3.6 (4F)	63 ± 3.6 (4F)	31.4 ± 3.2 (11M)	35.9 ± 3.9 (9M)	48.2 ± 3.5 (8F, 1M)
Time from injury (motion perception testing)	15 years	3 years	15 years	19 years	8 years
Visual impairments	Pure alexia, mild impairment in object recognition	Pure alexia, mild impairment in object perception	Object agnosia and prosopagnosia	Object agnosia and prosopagnosia	Face and object recognition difficulties (screening)

Motion coherence thresholds: Detailed coherence thresholds results and statistical analysis. Perceptual thresholds indicate the percentage of points that need to move coherently so that a participant can detect correctly the coherent direction of motion (that is embedded in dots moving in random directions). Lower thresholds indicate better performance. Results in bold indicate that a patient's coherence threshold was significantly higher than their matching controls (t test results are reported with t(10) for CR, t(8) for SM, and t(3) for GB and EL). Right ventral lesioned patients' data is shaded in gray. Note that the motion coherence thresholds of the right ventral lesioned patients (CR and SM) were significantly impaired while the left hemisphere patients were normal even when compared to younger controls (see also Figure 1A and Supplementary Material for more details).

	Coheren	nce threshol	T <sub>Crawford</sub>	P <sub>Crawford</sub>	
Patient initials	Controls (mean)	Controls (S.D.)	Patient		
EL	27.6	19.6	17.92	-0.404	0.755
GB	27.6	19.6	19.97	-0.31	0.803
CR	16.24	5.31	53.58	5.73	0.00036
SM	16.80	4.97	70.57	8.84	0.00045

Motion detection: Detailed detection accuracy results and statistical analysis. Results in bold indicate conditions that a patient performed significantly below their matching controls (t test results are reported with t(10) for CR, t(8) for SM and EC, and t(3) for GB and EL). Fst condition depicts very rapid motion of sparsely spaced points (same presentation parameters as 100pnt condition in the 3D SFM experiment). vSlw condition depicts very slow motion of densely spaced points. Right ventral lesioned patients' data is shaded in gray. EL's performance for vSlw is missing but since she performed at 95% accuracy for recognition in this task (controls 68.75±14.93) we are confident she is not impaired in detecting the motion at very slow speed (vSlw). As evident in the easy Fst condition, all patients (but CR) were at ceiling (100%) accuracy, corresponding to 0% lapse rates (for CR 5% lapse rate). Note that for the very slow motion (vSlw) all right ventral lesioned patients were significantly impaired in detecting the motion (SM and EC at chance level) while the left hemisphere patients were normal (see also Figure 1B). Furthermore, left hemisphere patients were also normal in very slow motion detection even when compared to younger controls, while right hemisphere patients were still impaired when compared to older controls (see Supp. Mat.).

Rotation/s	No. of	Condition		Accur	acy (% cor	Т	P <sub>Crawford</sub>	
	points	name					Crawford	
			Patient	Controls	Controls	Patient		
			initials	(mean)	(S.D.)			
0.5	100	Fst	EL	100	0	100	0	1
			GB	100	0	100	0	1
			CR	99.55	1.51	95	-2.89	0.016
			SM	99.44	1.67	100	0.32	0.76
			EC	98.33	3.54	100	0.45	0.67
0.0033	1600	vSlw	EL	100	0			
			GB	100	0	100	0	1
			CR	100	0	80	-95742	4*10 <sup>-46</sup>
			SM	99.44	1.67	50	-28.14	3*10 <sup>-9</sup>
			EC	100	0	50	-94898	2*10 <sup>-37</sup>

3D structure-from-motion recognition: Detailed recognition accuracy results and statistical analysis. Results in bold indicate conditions that a patient performed significantly below their matching controls (t test results are reported with t(10) for CR, t(8) for SM and EC, and t(3) for GB and EL). Right ventral lesioned patients' data is shaded in gray. As evident in the easy 1600pnt condition, all patients and all controls are at ceiling (100%) accuracy (corresponding to 0% lapse rates). Note that EL's recognition at 500pnt is probably due to a verbal mistake since in 100pnt which is much more difficult she is at ceiling. Note that when less form information is provided (100pnt) all right ventral lesioned patients are significantly impaired while the left ventral lesioned patients are at ceiling performance (see also Figure 1C). Moreover, left hemisphere patients were also normal even when compared to younger controls, while right hemisphere patients were still impaired when compared to older controls (see Supp. Mat.).

Rotation/s	No. of	Condition		Accuracy (% correct)				P <sub>Crawford</sub>
	points	name					Crawford	
			Patient	Controls	Controls	Patient		
			initials	(mean)	(S.D.)			
0.5	1600	1600pnt	EL	100	0	100	0	1
			GB	100	0	100	0	1
			CR	100	0	100	0	1
			SM	100	0	100	0	1
			EC	100	0	100	0	1
	500	500pnt	EL	100	0	95	-5000	<b>2*10<sup>-11</sup></b>
			GB	100	0	100	0	1
			CR	100	0	100	0	1
			SM	100	0	90	-9486	2*10 <sup>-29</sup>
			EC	100	0	100	0	1
	100 100pnt	EL	92.50	15	100	0.5	0.651	
			GB	92.50	15	100	0.5	0.651
			CR	98.18	3.37	60	-10.84	<b>8*10</b> <sup>-7</sup>
			SM	97.22	4.41	70	-5.857	4*10 <sup>-4</sup>
			EC	95.00	9.01	70	-2.631	0.03

Summary of patients' lesions and performance on motion perception tasks. The lesion approximation size is based on the outcome of the lesion delineating procedure, and an additional conservative size estimate is also provided (see Experimental Procedures). Assessment of lesion overlapping MT/V5 is based on MT/V5 reported location ((Kolster et al., 2010), see Experimental Procedures). Due to the low spatial coverage of GB and EC's clinical scans, we were not able to estimate the lesion size or MT/V5 lesion overlap. Motion perception classifications (normal, low range and impaired, shaded accordingly) for each patient is with respect to their control group.

	Left ventral lesion		Right (+ left ?)	Right ventral lesion	
	EL	GB	CR	SM	EC
Lesion extent	Extensive	Extensive	Intermediate	Small	Extensive
Lesion approximate size iso space (mm <sup>3</sup> )	43028	?	1510	952	?
Lesion conservative size iso space (mm <sup>3</sup> )	29616	?		516	?
MT/V5 overlap	Yes	?	No	No	?
Motion perception tasks					
Motion coherence	Normal	Normal	Impaired	Impaired	UNKNOWN
Motion detection (very slow)	Normal	Normal	Impaired	Impaired	Impaired
SFM (3D)	Normal	Normal	Impaired	Impaired	Impaired

#### **FIGURE LEGENDS**

#### Figure 1

Performance on basic motion perceptual tasks and 3D structure-from-motion. Each patient's data are plotted to the right of the matched control group; EL and GB have the same group of controls. A. Motion coherence perceptual thresholds (percentage of the points moving in a coherent direction) needed to detect direction of coherent motion. Lower thresholds correspond to better performance (see also Table 2). B. Motion detection (very slow motion) accuracy levels (see also Table 3). C. 3D structure-from-motion for many (left) and few points (right) defining the structure (see also Table 4). Below each histogram a static snapshot from the motion display, and an illustration of the percept caused by the motion are provided. Asterisks denote significant impairment of patient vs. matched controls (Crawford et al., 2009) with p<0.05. Error bars denote S.D. of control group. Bars of left ventral patients (EL and GB) indicated by patterns, right ventral patients (CR, SM, and EC) colored in gray shades (legend in top right). Note that right ventral patients were impaired in all motion perception tasks here, while left ventral patients performed at ceiling.

#### Figure 2

Lesion delineation procedure. Delineation for patients with high resolution anatomical images (EL, SM, and CR) followed the procedure depicted in panels A-D, while for patients with low spatial resolution images (GB and EC), the procedure is depicted in panels E-F. The original high resolution structural images (of patient EL) displayed (A) in neurological conventions (right on right) and the delineated lesion marked in red (B), are normalized into MNI space (C). The approximate location of left/right

MT/V5 (L/R-MT/V5) is depicted in blue/pink (Kolster et al., 2010). These are presented in a three-dimensional overlay seen from a posterior left lateralized view (D). GB's original low resolution structural images (E) are used to approximate the lesion extent on a normalized MNI brain (F) as depicted in red on axial (top), coronal and sagittal views (bottom), along with the approximate location of L/R-MT/V5 as in panel C. G. Three-dimensional lesion overlay of four of the patients (EL's brain appears in D), viewed from posterior lateral views (CR viewed from ventral view). Note that while EL's extensive lesion overlaps the expected location of left MT/V5 (D), SM's small lesion probably does not overlap his right MT/V5.





## **Inventory of Supplemental Information**

# The role of human ventral visual cortex in motion perception

Gilaie-Dotan S, Saygin AP, Lorenzi L, Rees G & Behrmann M

Gilaie-DotanEtAl\_SuppMat\_revised.doc includes:

Case descriptions: Additional details Motion Coherence: Additional details Motion Coherence: Additional control paradigms Motion Coherence: Lapse rates analysis Motion Detection: Additional details 3D structure-from-motion recognition: Additional details Lesion delineations: Additional details

## **Supplementary Material**

# The role of human ventral visual cortex in motion perception

Gilaie-Dotan S, Saygin AP, Lorenzi L, Rees G & Behrmann M

**Case descriptions: Additional details** 

Left hemisphere lesions

#### EL case description

EL (see Figure 2 A-D for lesion delineation and Tables 1 and 5 for case description details) has participated in many previous studies, which provide detailed description about her abilities and impairments (Behrmann et al., 1998; McKeeff and Behrmann, 2004; Montant and Behrmann, 2001; Mycroft et al., 2009). Briefly, previous testing revealed that she has pure alexia as well as some difficulty in object recognition.

#### GB case description

GB (see Figure 2 E-G for lesion delineation and Tables 1 and 5 for basic case description details) too suffers from pure alexia and, although she has not undergone extensive previous testing, all indications are that she has some mild impairment in object recognition, too.

#### Right hemisphere lesions

We provide more neuropsychological description for these cases who evince the impairment in motion perception.

#### SM case description

SM was 37 years old at the time of testing. He sustained a closed head injury in a motor vehicle accident at the age of 18 and recovered well after rehabilitation, aside from a persisting visual agnosia and prosopagnosia. Recent imaging (Konen et al., 2011) has located the lesion in a circumscribed region in the posterior portion of the right lateral fusiform gyrus, and the lesion is comprised of a volume of 990 mm<sup>3</sup>. Further details of his medical and neuropsychological history can be found in previous studies (Behrmann and Kimchi, 2003; Gauthier et al., 1999; Marotta et al., 2001; Nishimura et al.) and his lesion is shown in Figure 2G. SM performs within the normal range on tests of low-level visual processing and shows normal color vision. SM's object agnosia is evidenced by his object-naming performance in the Boston Naming Test (66% correct; normal: 96.4%) and his mean reaction time per correct item (2.14 s; normal: 0.88 s). When he fails to recognize an object, he does not appear to possess any semantic or action information about this object. His auditory identification of objects is unaffected and he can provide detailed definitions in response to the auditory label of an item that he missed when it was presented visually. SM's prosopagnosia is indicated by his impaired performance in the Benton Facial Recognition Test (36/54; normal 41-54). He is unable to recognize pictures of any famous people, despite being able to provide a good verbal identification when presented with their names auditorily. SM works in a photography studio.

#### CR case description

CR was 31 years old at the time of testing. He suffered from a right temporal lobe abscess with a complicated medical course including a history of Group A toxic shock syndrome, pneumonia, cardiac arrest, candida bacteremia, and metabolic encephalopathy in May 1996, approximately 15 years prior to his participation in this study. MR scans reveal a right temporal lobe lesion consistent with acute microabscesses of the right temporal lobe and medial occipital lobe (see Figure 2G). CR has no visual field deficits and performs within the normal range on all tests of low-level visual processing (judging size, length, and orientation of stimuli) as well as on the BORB subtests that require matching of objects from different viewpoints or along a foreshortened axis. CR is impaired at recognising some common familiar objects, as was made evident by his poor recognition scores of 46/60 (76.6% correct) on the Boston Naming Test and 149/185 on the Snodgrass and Vanderwart black-and-white line drawings. His ability to recognize static living things (64%) was worse than for static nonliving things (89%). CR's performance on tests of face recognition is in the "severely impaired" range on the Benton Facial Recognition tests (scores of 36/54 and 37/54 on two separate administrations of the test). CR has participated in several previous studies (Behrmann and Williams, 2007; Gauthier et al., 1999; Marotta et al., 2001) that highlight his use of part-based recognition mechanisms. CR completed community college and now runs a restaurant.

#### EC case description

EC, a 48-year old female, was tested four years after suffering an infarction. The radiology report states that there is low attenuation at the right temporal lobe and right occipital lobe posteriorly, consistent with a right PCA infarct (see Figure 2G).

She showed difficulties in both face and object recognition on screening tests conducted prior to these experiments. She was unavailable for further testing and did not complete the motion coherence experiment.

#### **Motion Coherence: Additional details**

We note further that the right hemisphere patients started their practice with an easier coherence threshold than did the left hemisphere patients (SM at 90% and CR at 80%, vs. GB and EL at 70%), and, in spite of this, they still failed to perceive the motion coherence direction normally. Since SM finished his practice run with a final coherence of 44.78%, his starting coherence threshold was 50%, similar to controls and to GB and EL. He did not however manage to improve during the motion coherence thresholds. CR finished the first run at >62% and the second at 70.57% coherence thresholds. CR finished his practice at >85% and that is why his first run started with that same threshold, his second run with 77.16% coherence. The lack of threshold improvement is *not* due to the initial starting threshold, as three controls who also started their practice runs with an easier opening threshold of 90% (like SM) finished their practice with thresholds of 18.4%, 20.8%, and 47.4%.

We also performed comparisons between patients and different aged controls to rule out possibilities of age affecting the results. To further examine the performance of the left hemisphere patients, we compared the performances of 61 y.o. EL and 70 y.o. GB with the performance of 8 young controls (aged  $31 \pm 3.1$ ), revealing normal performance ( $t_{crawford}(7) < 0.25$ , p > 0.8). For CR (31 y.o.) and SM (37 y.o.), we compared their performances to older controls (aged  $48.3 \pm 3.2$ ) and their thresholds were at the lower end of the controls (CR:  $t_{crawford}(2) = 2.18$ , p = 0.16, SM:  $t_{crawford}(2) = 3.31$ , p = 0.08). These comparisons provide further support that the profile of normal performance revealed by the left ventral visual patients, and that of abnormal performance revealed by the right ventral visual patients is not a function on the age of the comparison group.

#### Motion Coherence: Additional control paradigms

Our motion coherence paradigm consisted of target motion, directed upward or rightward, that was embedded in noise dots moving in random directions. To confirm that the rightward target motion component in our paradigm did not confound the hemispheric asymmetry results we found, we conducted detailed separate unidirectional motion coherence threshold measurements for upwards, downwards, rightwards, and leftwards motion, following precisely the same thresholding procedure as in our original paradigm.

#### Stimuli

The stimuli were as in our original paradigm, but the target motion in each run was consistently moving in one (of four) directions, and the participants (patients and controls) were notified about that direction in advance of each block of trials.

#### **Testing Procedure**

The testing procedures for each motion direction followed those of our original paradigm. For all participants, motion coherence thresholds were measured for upwards motion first, then rightwards, leftwards, and downwards motion. Each motion direction began with a practice block (starting threshold of 0.9), which was followed by one or two full experimental blocks of 48 trials each. The practice of the upwards motion was a full experimental block of 48 trials, as it was meant to familiarize the participants with the paradigm at the outset. Several practice trials were provided at the beginning of each block to familiarize participants with the new direction of motion to be tested.

#### **Participants**

To examine whether the rightward motion component in our original paradigm affected the motion perceptual threshold measurements, we needed to determine whether (a) a lesion to right ventral cortex impairs motion coherence thresholds for *leftwards* motion as it does for rightwards motion (as observed in our original paradigm) and (b) motion coherence thresholds are unaffected following left ventral lesion for *leftwards* motion as they were unaffected for rightwards motion. To explore these two complementary findings, we tested patient SM (age 38 at the time of testing) and four age and gender matched controls (men aged  $39 \pm 2.9$  s.d.), and patient EL (age 62 at the time of testing) and two age and gender matched controls (women aged  $65 \pm 1.5$  s.d.) in these additional, motion coherence control experiments.





SM's motion coherence thresholds were significantly impaired in all directions (rightwards: 25.29% vs. 7.26%  $\pm$  0.6% for controls, t<sub>crawford</sub>(3) = 24.7, p < 0.0009, leftward: 26.95% vs. 8.7%  $\pm$  1.3% for controls, t<sub>crawford</sub>(3) = 12.15, p < 0.004,

upwards: 15.72% vs. 10.1%  $\pm$  1.4% for controls,  $t_{crawford}(3) = 3.55$ , p < 0.04,

downwards: 23.48% vs. 11.59%  $\pm$  1.7% for controls, t<sub>crawford</sub>(3) = 6.02, p < 0.02). EL's motion coherence thresholds were unaffected by her left ventral visual lesion, and her performance was superior to that of her controls in all directions (rightwards: 9% vs. 63%  $\pm$  38% for controls, t<sub>crawford</sub>(3) = -1.15, p = 0.23, leftwards: 6.3% vs. 66%  $\pm$  38% for controls, t<sub>crawford</sub>(3) = -1.29, p = 0.21, upwards: 15. 9% vs. 68.9%  $\pm$  28.3% for controls, t<sub>crawford</sub>(3) = -1.53, p = 0.184, downwards: 11.4% vs. 52 %  $\pm$  49.2% for controls, t<sub>crawford</sub>(3) = -0.67, p = 0.31).

These additional motion coherence results confirm our original findings that right ventral visual lesion causes impairments in motion coherence thresholds, and rules out the possibility that the original results were biased by the rightward motion component in our original paradigm. Furthermore, these results confirm our original findings that a lesion to right, but not left ventral, cortex impairs motion coherence thresholds. Specifically, SM's motion coherence thresholds were impaired in all directions whereas EL's thresholds were unimpaired in all directions.

#### Motion Coherence: Lapse rates analysis

Lapse rates measure the rate at which observers make stimulus-independent errors, and, thus, can be measured as the error rate at the tail end of the psychometric function, where ceiling performance is expected. Since the motion coherence was a threshold estimation experiment, it is not as simple to measure lapse rates as the stimulus intensity constantly changes. Nevertheless, when we examined the error rates at high coherence levels, for GB and EL, accuracy was at 100% corresponding to lapse rates of 0. For SM, in the control paradigm when all coherent stimuli were at the same direction throughout the run, his lapse rate was 0 (no errors at high intensities of coherence). For CR, when he felt confident with the task his lapse rate (at high coherence levels) was 0 (no errors, except for the 1st trial in that session, which could be due to the beginning of the session) for thresholds above 55% coherence (9 trials). Thus, it is highly unlikely that the motion coherence thresholds of SM and CR are due to lapse rates. Instead, they seem to genuinely reflect their motion coherence impaired thresholds.

#### **Motion Detection: Additional details**

To verify that the observed patterns of normality and impairment were not dependent on age effects, we carried out further comparisons, pitting the patient performance against that of control groups of various ages. We conducted a stringent test of the normality of motion detection in patients with left hemisphere lesions by comparing the performances of 61 y.o. EL and 70 y.o. GB with that of 11 young controls (controls selected for CR; aged  $31.4 \pm 3.2$ ). Given that EL and GB scored 100% accuracy as did all the young controls, the older patients and young controls have equivalent scores (for EL and GB:  $t_{crawford}(10) \sim = 0$ ,  $p \sim = 1$ ). To assess the extent of the impairment (relative to age-matched controls) for CR (31 y.o.), SM (37 y.o.), and EC (48 y.o.), we compared their very slow motion detection performance to that of older controls (aged  $63 \pm 3.6$ ). All three patients' thresholds were significantly impaired even under these conditions (for CR, SM, and EC:  $t_{crawford}(3) < -10^5$ ,  $p < 10^{-10}$ <sup>13</sup>). These comparisons provide further support that the results we obtained were not merely a function of the age of the control group enlisted and that the intact performance of the left ventral visual patients, and the impairment in motion coherence thresholds in the right ventral visual patients hold even under a wide range of statistical comparisons.

#### **3D** structure-from-motion recognition: Additional details

In the same manner detailed above for motion coherence and motion detection, we also performed statistical comparisons for the patients against different aged controls. CR and SM were compared to older controls (aged  $48.2 \pm 3.5$ ), and here too they were significantly impaired even when compared to these older controls (CR:  $t_{crawford}(8) = -3.68$ , p = 0.0062, SM:  $t_{crawford}(8) = -2.63$ , p = 0.03). On the other hand, the older patients, EL and GB, scored within the normal range even when compared to younger (aged  $31.4 \pm 3.2$ ) controls (EL and GB:  $t_{crawford}(10) = 0.52$ , p = 0.62).

#### Lesion delineations: Additional details

CR has a definitive lesion in the right temporal lobe (see above) that is evident and confirmed by an expert neuroradiologist in the past and during the current study. There are, however, some suspicious hyperintensities in the left hemisphere (perhaps resolved abcesses) and so we adopt a conservative approach here and leave open the possibility of additional left hemisphere insult. Critical for our case, however, the lesion impinged on the ventral aspects of the right hemisphere (see Figure 2G), but did not overlap the expected locations of R/L-MT/V5. Of relevance too is that CR did not have a visual field defect.