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The effect of target speed on perception of visual motion direction in a patient with akinetopsia

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ABSTRACT

Although much research has been devoted to the neural correlates of motion perception, the processing of speed of motion is still a topic of discussion. Apart from patient LM, no in-depth clinical research has been done in the past 20 years on this topic. In the present study, we investigated patient TD, who suffered from the rare disorder akinetopsia due to bilateral lesions of V5 after stroke. By means of a Random-Dot-Kinematogram (RDK) in which speed was varied systematically, it was found that TD was impaired in perceiving the direction of movement at speeds exceeding 9 deg/s. Our study suggests that V5 plays an important role in processing high-speed visual motion and further implies that V5 does not play a crucial role in processing low-speed visual motion. A remarkable finding, which has not been shown before, was that TD always reported the opposite direction of the actual movement at a speed of 24 deg/s. This suggests a form of the continuous wagon wheel illusion, which might have been caused by intact brain areas operating at different sampling rates than area V5.

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1. Introduction

Visual motion is defined as the perception of changes in optical information over space and time (Schiffman, 2001). Although the processing of visual motion in the brain has been studied quite extensively in the past, there are still

controversies about the neural correlates of specific components of visual motion. Especially the processing of speed is still a topic of discussion.

Earlier research with rhesus monkeys showed that the middle temporal area (MT) is related to the perception of visual motion (Albright, 1984; Zeki, 1974). The human analogue of the MT area is the V5 area, at the junction of the parietal, temporal

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and occipital cortices (Zeki et al., 1991). Bilateral damage to this area may lead to a condition called akinetopsia, or motion blindness. Akinetopsia is an extremely rare disorder and most clinical knowledge is based on experiments with only one patient, LM (Zihl & Heywood, 2015). After acquiring a lesion involving area V5 bilaterally, LM reported an inability to perceive motion, whereas other visual and visual-perceptual abilities remained largely intact. Apart from LM, difficulties with several aspects of motion perception have been described in patients with unilateral lesions, although their functional impairments are not described in as much detail as in LM. Cooper et al. (2012) described two patients with transient akinetopsia as a result of unilateral lesions. Patient 1 suffered from infarcts in the right inferior parietal lobe and parietal-occipital junction (sparing area V5) whereas patient 2 showed a lesion in his left hemisphere encompassing area V5. Although the akinetopsia resolved in both patients, impaired saccadic eye movement to moving stimuli persisted. In a different study, another patient was described with persisting akinetopsia after two expanded lesions in the right temporoparietal region (Otsuka-Hirota, Yamamoto, Miyashita, & Nagatsuka, 2014).

LM participated in multiple experiments showing that her visual perception of stimuli with high speed was impaired, while her visual perception of stimuli with slow speed was intact (Campbell, Zihl, Massaro, Munhall, & Cohen, 1997; Hess, Baker, & Zihl, 1989; Schenk, Mai, Ditterich, & Zihl, 2000; Zihl, Von Cramon, & Mai, 1983). This dissociation is in line with the so-called dynamic parallelism theory, which proposes different cortical pathways for slow and fast-moving stimuli (ffytche, Guy, & Zeki, 1995). The indirect pathway extends from the parvocellular layers of the lateral geniculate nucleus in the thalamus, via V1 to the prestriate cortex including V5 and seems to be dominant for the processing of slow motion. The other pathway is a direct pathway from the magnocellular layers of the lateral geniculate nucleus in the thalamus to V5 and possibly V3 in parallel. This direct pathway is thought to be dominant for the processing of fast motion.

Most evidence for the dynamic parallelism theory is provided by psychophysiological studies in healthy participants. In an EEG/MEG study, fftyche et al. (1995) demonstrated that V5 is activated before V1 when a stimulus was moving faster than 22 deg/s. When the same stimulus was moving slower than 6 deg/s, V1 was activated before V5. Another study employing transcranial magnetic stimulation (TMS) showed that magnetic stimulation of V5 may disrupt perception of direction of moving dots on a video monitor at a speed of 11 deg/s, while TMS on V1 does not cause such disruption (Beckers & Zeki, 1995). Further evidence for a direct pathway between the thalamus and V5 bypassing V1 is also found in an experiment by Gaglianese, Costagli, Bernardi, Ricciardi, and Pietrini (2012) using functional magnetic resonance imaging (fMRI). In their study, ten healthy participants performed a motion detection task while brain scans were made using fMRI. It was found that neural activity in the lateral geniculate nucleus (in the thalamus) directly influences V5, independently from V1. A more prominent pathway was the indirect pathway from the thalamus to V5 via V1. The direct pathway from the thalamus to V5 may play a role in the fast detection of motion, and it may even play a role in the preconscious detection of motion (Gaglianese et al., 2012).

Apart from LM, patient GY provided clinical support for the double dissociation postulated by the dynamic parallelism theory. GY suffered from a unilateral lesion of V1 in his left hemisphere, resulting in a homonymous right visual field defect with macular sparing. When a moving stimulus was presented in his blind hemifield, he was able to report the direction of this movement when speeds were higher than 6 deg/s (Barbur, Watson, Frackowiak, & Zeki, 1993). An EEG study further showed that movement at a speed of 5 deg/s did not generate a cortical response in GY (ffytche, Guy, & Zeki, 1996), whereas the early brain response to faster moving stimuli was preserved.

However, a number of studies have provided evidence against the dynamic parallelism theory. Clinical studies have shown that the double dissociation of fast and slow motion processing does not always hold for patients with lesions in V1 (e.g., not all patients studied were able to perceive fast movement whilst being impaired with processing slow movement after V1 damage; Azzopardi & Cowey, 2001; fftyche & Zeki, 2011). For example, fftyche and Zeki (2011) tested three patients with unilateral lesions in the primary visual cortex and consequently hemianopic field defects using psychophysical experiments in which speed and direction of moving stimuli had to be determined. Two of these patients (FB and GN) were able to detect the direction of moving stimuli in their blind hemifield only when the stimulus was moving fast (>18 deg/s). The other patient (CG), on the other hand, was not aware of the moving stimulus in blind hemifield and reported that he could only see a flash when the moving stimulus appeared. However, the extent of the lesion in patient CG was unknown. Other evidence against the dynamic parallelism theory comes from a Diffusion Tensor Imaging (DTI) study that showed that fiber tracts that are supposed to represent the direct pathway between thalamus and V5, were only found in four of the ten participants (Lanyon et al., 2009). In addition, Van Boxtel, van Ee, and Erkelens (2006) used mathematical modelling based on neurophysiological principles to show that only a single system is responsible for speed processing. In the literature, the dynamic parallelism theory is thus still controversial.

In the present study, patient TD is presented, who suffered from bilateral lesions of V5 after stroke. Similar to LM, this patient also reported problems with the visual perception of motion. Since cerebral akinetopsia is extremely rare, TD's clinical case offered the unusual opportunity to investigate motion perception from a clinical perspective approximately 20 years after research with LM has been executed. Specifically, the aim of this study was to investigate the effect of target speed on visual motion perception. The processing of motion speed is still a topic of controversy and apart from LM, there are no other cases with bilateral akinetopsia that are extensively described in the literature. Findings will be compared with LM's case and with theories about motion processing in the brain.

2. Materials and method

2.1. Case description

TD, a 37-year-old right-handed female, was referred to Royal Dutch Visio, Centre of Expertise for Blind and Partially Sighted

People, with several visual complaints following a stroke. She experienced problems with perceiving visual motion and reported that looking at bright colours, bright light, sharp contrasts and certain patterns made her feel nauseous. She further complained that objects at a distance exceeding approximately five meters were difficult to see. Eight months before referral, TD had experienced a sudden dizziness followed by a short period of loss of consciousness. Three months after the incident, an MRI scan showed an ischaemic infarction of the occipito-temporal region in the right hemisphere and a smaller infarction in the left occipital hemisphere, which was confirmed by an MRI scan another three months later. Figs. 1 and 2 (see also [supplementary figure](#) for animated GIF) show that the damaged areas contained area V5 in both hemispheres with the right hemisphere being more affected compared to the left. More specifically, part of the radiatio optica was damaged as well in the right hemisphere. V1 was still intact in both hemispheres. No other neurological impairments or disorders of cognitive functioning were found.

2.1.1. Assessment of lower visual functions

Assessment of visual functions took place 20 months post-stroke. Smooth pursuit, saccades, optokinetic nystagmus and vestibulo-ocular reflex were shown to be normal in both the horizontal and the vertical directions. Ocular alignment was normal and TD was able to move her eyes in every direction. Measures of visual field and visual acuity were inconsistent over time and measurements (Goldmann, Humphrey, Octopus) not reliable. There were some indications that the left visual field was impaired, although TD's response to stimuli in the left visual field varied. Likewise, visual acuity measurements were inconsistent and differed between .32

and .7 (Snellen decimals). Contrast sensitivity, measured 23 months post-stroke, was found to be normal and matched a visual acuity of approximately .9, therefore suggesting normal visual acuity. Colour vision was not disturbed. Binocular vision was largely intact, although TD's score on a stereopsis test was not optimal. To summarize, even though TD showed some impairment when testing the lower visual functions, these findings could not explain TD's difficulties with motion perception.

2.1.2. Assessment of higher visual functioning

Visual perceptual functioning was tested 10 and 20 months post-stroke to examine if TD's impaired motion perception could be caused by Bálint's Syndrome and to exclude other perceptual disorders. For this purpose, a number of standardised and neurobehavioural tests were used (see [Tables 1 and 2](#)). There were minor indications of impaired spatial cognition, but there was no evidence of Bálint's Syndrome, unilateral neglect or visual extinction. Furthermore, there was no convincing evidence for impaired object perception or prosopagnosia. Although minor indications for an impaired spatial cognition were demonstrated, these were not sufficient to explain TD's visual complaints with regards to motion perception.

2.1.3. Assessment of visual motion perception

To gain more insights into TD's symptoms of impaired motion detection, a number of tasks were carried out assessing TD's motion perception. In these tasks TD's performance on moving stimuli was compared to her performance on static stimuli whilst controlling for factors such as contrast, colour, viewing distance and target size, letter recognition, and object recognition. On all tasks, TD made errors in the dynamic conditions

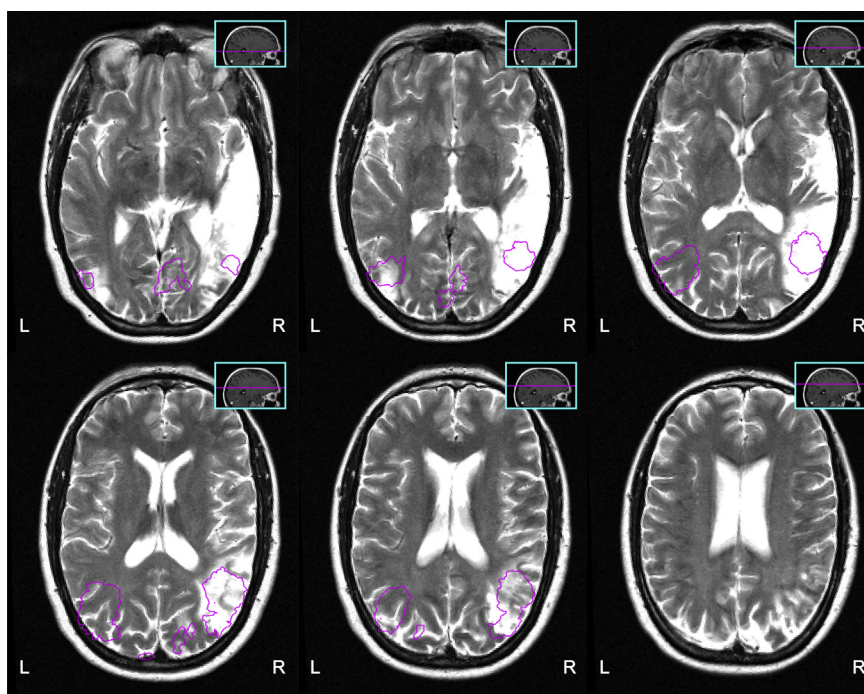


Fig. 1 – Selected axial slices (top row: $z = 10, 15, 20$; bottom row: $z = 25, 30, 35$) from the T2 volume, together with a functional ROI of V5 which has been overlaid as a contour map. Basis for the ROI is a map generated for the keyword ‘V5’ at neurosynth.org.

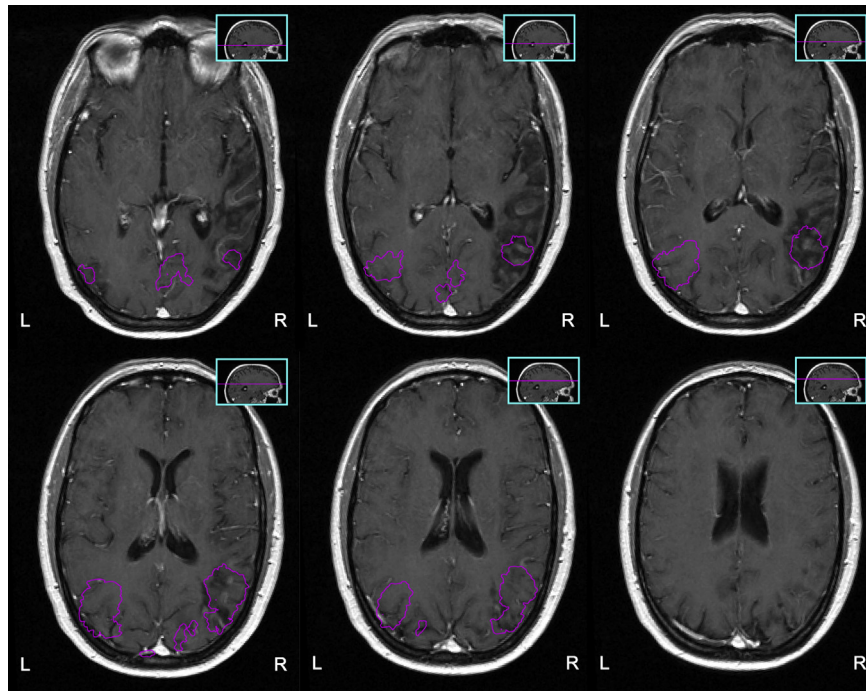


Fig. 2 – Selected axial slices (top row: $z = 10, 15, 20$; bottom row: $z = 25, 30, 35$) from the T1 volume, together with a functional ROI of V5 which has been overlaid as a contour map. Basis for the ROI is the map generated for the keyword ‘V5’ at neurosynth.org.

only and her performance deteriorated with faster movement of the stimuli.

In conclusion, TD’s impaired motion perception is both specific and selective. TD’s overall performance appears to be comparable to the performance of LM (Hess et al., 1989;

Schenk et al., 2000; Zihl et al., 1983; Zihl & Heywood, 2015), although self-reported perception of fast movement differs between LM and TD. LM reported seeing a fast moving object at successive stationary positions, while TD reported perceiving a smear or cloud around a fast moving object.

2.2. Control participants

The six healthy control participants were all female and were of similar age as TD. Participants were aged between 36 and 47 years old ($M = 41$), had no history of neurological or psychiatric disorders, and all had normal or corrected to normal vision. The experiment was approved by the Ethical Committee Psychology of the University of Groningen, the Netherlands, according to the Declaration of Helsinki. All participants provided written informed consent.

2.3. Random-Dot-Kinematogram (RDK)

The RDK was presented on a Macintosh (MacBook) laptop with a screen size of 13 inch. RDKs are often-used stimuli in studies concerning motion perception. An advantage of this approach is that motion is presented continuously, thereby preventing participants to guess the direction of the motion based on the location of the moving stimuli. The random dot stimuli were created and presented in MATLAB using the Psychophysics Toolbox extensions (Brainard, 1997; Pelli, 1997). The task consisted of trials in which small blocks all moved in the same direction with the same speed against a black background. The blocks moved within an imaginary circle with a diameter of 20° . When a block reached the edge of the circle it reappeared on the other side of the circle to continue its

Table 1 – TD’s performance on the visual perceptual assessment.

	Test	Score	Compared to norm data
VOSP	Screening	19	+
	Incomplete Letters	20	+
	Silhouettes	17	+
	Object Decision	15	+/-
	Dot Counting	10	+
	Position Discrimination	15	-
	Number Location	5	-
	Cube Analysis	8	+
BT	A: left	10	
	A: right	10	
	A: total	20	
	B: left	8	
	B: right	8	
	B: total	16	-
BFRT	Corrected Long Form Score	34	severe impairment

+ above cut-off; - below cut-off; +/- on cut-off.

VOSP = Visual Object and Space Perception Battery (Warrington & James, 1991).

BT = Balloons Test (Edgeworth, Robertson, & McMillan, 1998).

BFRT = Benton Face Recognition Test (Benton, Sivan, Hamsher, Varney, & Spreen, 1994).

Table 2 – TD's performance on neurobehavioural assessment.

Assessment	Observation
Famous persons test	TD was able to indicate whether people that were presented on photos were a famous person, a look-a-like of a famous person or an unknown person
Overlapping figures	TD was able to identify all the figures except one. When the examiner went with a pen along the line of the unrecognized figure she was able to identify it.
Giuseppe Arcimboldo paintings	TD was able to identify both the details (vegetables, fruits, etc.) as well as the whole composition (face)
Optic ataxia	TD was able to touch a designated finger of the test leader in the air.
Oculomotor apraxia	TD was able to follow the movement of the test leader's finger with her eyes.

movement in the same direction. This way, a continuous movement was shown. In the middle of the screen a fixation cross was present during the trials and the participants were asked to keep looking at this cross. Participants were asked to report both verbally and by pointing their finger in which direction the blocks were moving.

Since TD reported feeling nauseous when she was presented with a high number of blocks (150) or blocks with a high contrast, 30 blocks with a lower contrast were used in the present study. Block size was .2 by .2°. Coherence, which is defined as the chance that a given block will move in the global direction on a following frame, was set at 100 percent as we were primarily interested in the effect of speed and direction of motion and therefore we also decreased the influence of other variables, such as non-optimal coherence. Speed was varied randomly, with values of 2, 4.5, 9, 15 and 24 deg/s. Direction was also randomly varied, with the restriction that the same direction would not appear more than two times in a row. Four directions were used: leftward, rightward, upward and downward.

One block of trials consisted of 20 trials to include every combination of speed ($n = 5$) and direction ($n = 4$) once. The order of the trials was randomly set by the Matlab program. Each trial had a maximum duration of 10 sec. If a response was given within these 10 sec, the trial was terminated by the examiner.

2.4. Procedure

TD was tested at a table in her own house on two different occasions. Testing took place at 10 A.M. The room was darkened by closing the curtains and turning down the lights. Viewing distance was 50 cm. On both occasions six blocks of trials were presented. Because TD fatigued with increasing exposure to the task and because the moving blocks caused discomfort when presented for a long time, frequent rest intervals were applied. The same test conditions were applied for the six control participants.

2.5. Data analysis

The answers on the RDK can be classified as either correct or incorrect. Fisher's exact test was used to test whether the proportion of TD's correct and incorrect responses was the same in the different speed conditions. The performance of TD on the RDK was also compared with the performance of the six control participants.

3. Results

Speed of movement had a significant effect on TD's performance (Fig. 3). At 2, 4.5 and 9 deg/s, TD did not make any errors on the RDK. However, her performance deteriorated at 15 deg/s (25 out of 48 correct) compared to trials with a speed of 2, 4.5 or 9 deg/s ($p < .001$, Fisher's Exact test, 2-tailed). She made even more errors when speed was 24 deg/s (1 out of 48 correct) compared to 15 deg/s ($p < .001$, Fisher's Exact test, 2-tailed). This means that her performance was far below chance level as she reported the exact opposite of the actual direction in 47 of the 48 trials. Direction of movement had no influence on TD's performance. The amount of errors was respectively 17, 18, 18 and 17 for the directions left, right, upwards and downwards. TD made no errors in the axis of the movement, which means that all her errors were in the direction opposite to the actual presented motion.

The six control participants did not make any errors at all on the RDK (48 out of 48 trials correct at each speed). Both TD and some of the control participants reported that it took more effort to maintain the fixation in the faster conditions. Nevertheless, no significant eye movements were observed.

4. Discussion

In the present study, we investigated the effect of target speed on visual motion perception in a patient with akinetopsia after bilateral lesions of V5. Compared to a healthy control group, who was able to perceive the direction of the stimuli at all speeds correctly, TD's performance dropped dramatically

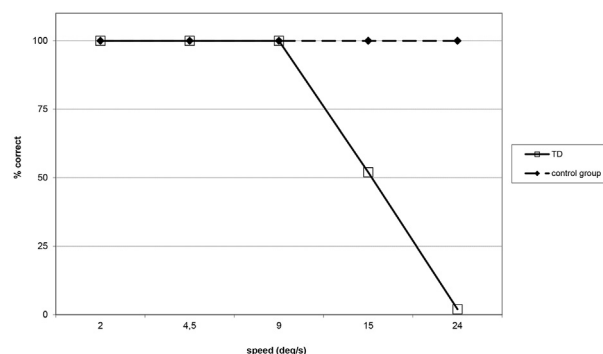


Fig. 3 – Percentage correct responses for TD and control group on the RDK. TD's performance deteriorates after a speed of 9 deg/s, whereas the control group showed 100% correct performance in all conditions resulting in a standard deviation of zero.

when speed exceeded 9 deg/s. This result suggests that V5 plays an important role in perception of movement above a speed of 9 deg/s. The present findings are partially similar to a study with LM, using a comparable RDK, in which LM's performance deteriorated at a threshold ranging from 8 to 16 deg/s (Hess et al., 1989). However, LM's score was never 100% correct when speed of motion was between 1 and 8 deg/s. Some involvement of V5 in processing slow movement could therefore not be fully excluded in LM. The present experiment, with TD reporting 100% correctly on all trials with a speed of 9 deg/s or lower, therefore gives stronger evidence that V5 might not be crucially involved in processing slow movement. Both this flawless perception of motion direction at speed 9 deg/s and lower and the rapid deterioration of detection at 15 deg/s and faster are in support for the dynamic parallelism theory, which proposes different cortical pathways for slow and fast-moving stimuli (ffytche et al., 1995).

However, the size, extent, and asymmetry of the brain damage prevent a definitive conclusion in favour of the dynamic parallelism theory. For instance, spared V5 tissue or other brain areas not affected by the lesion might have contributed to TD's preserved ability to perceive the direction of slowly moving stimuli. Alternative studies have suggested that motion is processed by different pathways based on other characteristics than the speed of the stimulus, such as systems selective for luminance and colour (Gorea, Papathomas, & Kovacs, 1993), systems for low-level and high-level motion processing (Cavanagh & Mather, 1989), or pathways for binocular disparity and motion direction (Ponce, Lomber, & Born, 2008). In addition, Van Boxtel et al. (2006) proposed a single system for visual motion perception in which speed points (slow and fast motion) are described as points along a continuum of motion perception.

With regard to direction discrimination, TD reported the axis of the movement correctly on all trials, which means that TD's errors were always opposite to the presented direction. This is comparable to the performance of LM on an RDK study by Shipp, Jong, Zihl, Frackowiak, and Zeki (1994). Surprisingly, TD's performance at a target speed of 24 deg/s dropped to 1 out of 48 correctly (with 12 correct being at chance level), yet always reporting in the exact opposite direction of the actual movement of the targets. This finding might suggest at least two things: First, that V5 might not be crucial for the perception of the axis of movement and second, that processing of fast movement might not be exclusively linked to V5, but to other brain areas, such as V1 and V3, as well. In terms of the dynamic parallelism theory, this could mean that although the direct route from the thalamus to V5 may be dominant for processing fast-moving stimuli, the indirect route, involving the thalamus, V1 and pre-striate areas, is not disengaged when fast-moving stimuli are presented. Again, these explanations need to be viewed with caution, as other reasons, such as partly spared motion detection, need to be considered as well. Although V5 has shown to be important for direction discrimination, direction-selective neurons might not be exclusive to this area (Newsome, Britten, Salzman, & Movshon, 1990).

The finding that TD always reported the wrong direction for high speed motion, yet across the right axis, may suggest a form of illusory motion reversal comparable to the wagon wheel illusion, in which the movement of a rotating wheel

can be perceived as moving in opposite direction rather than its actual direction when presented stroboscopically (Finlay & Dodwell, 1987). Several studies have shown that the wagon wheel illusion can also occur in continuous light (Andrews & Purves, 2005; Purves, Paydarfar, & Andrews, 1996; Van Rullen, Reddy, & Koch, 2005; Van Rullen, Zoefel, & Ilhan, 2014). Kline and Eagleman (2008) argue that this phenomenon can be explained with the occurrence of motion aftereffects which move in the opposite direction and are superimposed on the actual moving stimuli. Accordingly, the continuous wagon wheel illusion arises when motion detectors for the opposite direction get activated. Other authors, however, argue in favour of the fact that visual information is processed as a sequence of discrete snapshots rather than continuously (Andrews & Purves, 2005; Purves et al., 1996; Van Rullen, Reddy, & Koch, 2006; Van Rullen, Pascual-Leone, & Battelli, 2008; Van Rullen et al., 2003; Van Rullen et al., 2016).

In the light of these explanations, TD is an exceptional case, since she appeared to see a form of the continuous wagon wheel illusion at fast speeds even though her perception for fast moving stimuli was impaired. In line with theory of discrete sampling, one could argue that this might be a consequence of spared brain areas which might operate at a different sampling rate compared to the affected areas (in this case V5).

5. Conclusion

Our study provides valuable information on motion perception by a patient with akinetopsia. The performance of this patient is in favour of the dynamic parallelism theory and suggests that V5 may be more important for high-speed than for low-speed visual motion. In addition, our patient perceived motion in the opposite direction at high speeds. This might suggest that other brain areas – involved in processing slow motion – may still be involved in processing fast motion, but operating at different sample rates than area V5. However, due to the size, extent, and asymmetry of the lesion, interpretations need to be viewed with caution. The present study may, however, give insights into and stimulate further research on motion processing and theories on discrete processing of visual information at different frequencies across different brain areas.

Conflicts of interest

None.

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Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cortex.2018.12.002>.

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