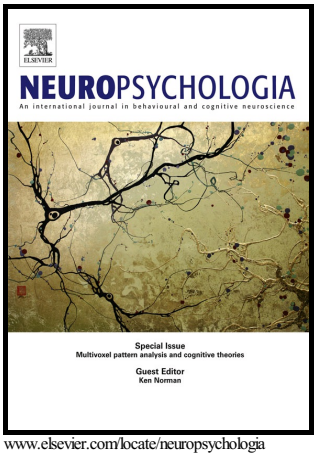

www.elsevier.com/locate/neuropsychologia

Can you guess the colour of this moving object? A dissociation between colour and motion in blindsight

E. Chabanat, S. Jacquin-Courtois, L. Havé, C. Kihoulou, C. Tilikete, F. Mauguière, S. Rheims, Y. Rossetti

PII: S0028-3932(18)30470-6
DOI: https://doi.org/10.1016/j.neuropsychologia.2018.08.006
Reference: NSY6879

To appear in: *Neuropsychologia*

Received date: 2 November 2017
Revised date: 1 June 2018
Accepted date: 8 August 2018

Cite this article as: E. Chabanat, S. Jacquin-Courtois, L. Havé, C. Kihoulou, C. Tilikete, F. Mauguière, S. Rheims and Y. Rossetti, Can you guess the colour of this moving object? A dissociation between colour and motion in blindsight, *Neuropsychologia,* https://doi.org/10.1016/j.neuropsychologia.2018.08.006

Can you guess the colour of this moving object? A dissociation between colour and motion in blindsight

Chabanat, E.[1,2], Jacquin-Courtois, S.[1,2,3,4], Havé L. [1,2], Kihoulou, C.[1], Tilikete, C.[1,5], Mauguière, F.[2,6,7], Rheims, S.[2,6,8] & Rossetti, Y.[1,2,3,4]

1. Inserm UMR-S 1028, CNRS UMR 5292, ImpAct, Centre de Recherche en Neurosciences de Lyon, France
2. Université de Lyon, Université Claude Bernard Lyon 1, France
3. Service de rééducation neurologique, Pavillon Bourret, Hôpital Henry-Gabrielle, Hospices Civils de Lyon, 20, route de Vourles, Saint-Genis-Laval, France
4. Plate-forme 'Mouvement et Handicap', Hôpital Henry-Gabrielle et Hôpital Neurologique Neurologique Pierre Wertheimer, Hospices Civils de Lyon, 20, route de Vourles, Saint-Genis-Laval, France
5. Service de Neuro-Cognition et Neuro-Ophtalmologie, Hôpital Neurologique Pierre Wertheimer, Hospices Civils de Lyon, 59 boulevard Pinel, 69677 Bron Cedex
6. Département de Neurologie Fonctionnelle et Epileptologie, Hôpital Neurologique Pierre Wertheimer, Hospices Civils de Lyon, France
7. Inserm UMR-S 1028, CNRS UMR 5292, NeuroPain, Centre de Recherche en Neurosciences de Lyon, France
8. Inserm UMR-S 1028, CNRS UMR 5292, TIGER, Centre de Recherche en Neurosciences de Lyon, France

Abstract

Blindsight has been primarily and extensively studied by Lawrence Weiskrantz. Residual visual abilities following a hemispheric lesion leading to homonymous hemianopia encompass a variety of visual-perceptual and visuo-motor functions. Attention blindsight produces the more salient subjective experiences, especially for motion (Riddoch phenomenon). Action blindsight illustrates visuo-motor abilities despite the patients' feeling that they produce random movements. Perception blindsight seems to be the weakest residual function observed in blindsight, e.g. for wavelength sensitivity. Discriminating motion produced by isoluminant colours does not give rise to blindsight for motion but the outcome of the reciprocal test is not known. Here we tested whether moving stimuli could give rise to colour discrimination in a patient with homonymous hemianopia. It was found that even though the patient exhibited nearly perfect performances for motion direction discrimination his colour discrimination for the same moving stimulus remained at chance level. It is concluded that easily discriminated moving stimuli do not give rise to colour discrimination and implications for the 3 levels of blindsight taxonomy are discussed.

Key words: perception-blindsight, attention-blindsight, coulour, motion, vision, cortical blindness, dorsal stream

1. Introduction

Blindsight (Weiskrantz, 1986) has been initially described in humans in the early 70' (Pöppel et al, 1973; Weiskrantz et al, 1974; Perenin & Jeannerod, 1975) as a neurological condition whereby patients with hemianopia exhibit residual visuo-motor processing. The oxymoron "blindsight" emphasizes that patients experience a lack of visual information in at least one part of their visual field and yet show a peculiar ability to direct their eyes or their hand toward the unseen stimuli presented within this scotoma (Weiskrantz 1986, 1996, 2009; Danckert & Rossetti 2005). Typically, patients with cortical blindness exhibit homolateral hemianopia, with no visual experience coming from one hemifield. In order to present visual stimuli within this blind portion of their visual field, visual fixation has to be controlled. Accuracy of saccades and hand reaches is poorer than in their healthy visual field and in healthy subjects, but it is significantly better than chance, i.e. the saccade or reach endpoints are correlated with target positions. It has been proposed that subcortical substrates such as the superior colliculus may be responsible for this residual visuo-motor ability (Weiskrantz et al., 1974; Zihl & Werth, 1984), which is directly supported by the observation of blindsight in patients with hemispherectomy (Perenin & Jeannerod, 1978), by functional brain imaging (Leh, Ptito, Schönwiesner, Chakravarty & Mullen, 2010) and correlation between blindsight occurrence and pupillary reactions (Sahraie, Trevethan, MacLeod, Urquhart & Weiskrantz, 2013). However the accuracy of pointing movements appears to be better than that of saccades performed by some blindsight patients (Danckert & Rossetti, 2005). Furthermore, this ability is not restricted to processing of the stimulus location, which is required for saccades and reaches, but may also concern stimulus orientation or size (Perenin & Rossetti 1996). This result implies that other brain structures are involved in blindsight. It has therefore been proposed that subcortical structures project visual information onto cortical areas involved in motor control, i.e. the parietal cortex (Rossetti, 1998; Danckert & Rossetti 2005). As the matter of anatomical facts, the reversible inhibition of monkey primary visual cortex (V1) is accompanied by the suppression of electrophysiological activity in the occipito-temporal pathway while some residual activity can be recorded in the occipito-parietal pathways (Girard, Salin & Bullier; 1991-1992).

Blindsight has also been described to occur for other qualities of visual information than just localization. Some patients exhibit more or less vivid reactions to motion stimuli, for which they experience no visual phenomenology but a clear sense of motion, i.e. the Riddoch phenomenon (Morland, Jones, Finlay, Deyzac, Le & Kemp, 1999 ; Zeki & ffytche, 1998). Some of these patients may discriminate motion direction. In some cases, psychophysical experiments were able to report that patient exhibit some discrimination capacity for shape (Trevethan, Sahraie & Weiskrantz, 2007), wavelength (Stoerig & Cowey, 1989) or even emotions (Weiskrantz, 2000 ; Pegna, Khateb, Lazeyras, & Seghier, 2005). Altogether Danckert & Rossetti (2005) proposed to distinguish between three main categories of blindsight. Attention-blindsight is characterized by a vivid experience of the subjects, as if it corresponded to some sense of alertness. Action-blindsight corresponds to the initial observation of visuo-motor residual abilities. Agnosopia or Perception-blindsight correspond to cases where static visual qualia can be statistically discriminated over numerous trials.

Functional connectivity from the colliculus onto cortical structures has been described in attention-blindsight (Leh, Ptito, Schönwiesner, Chakravarty & Mullen, 2010). Building up on Schmid et al. (2010), Ajina et al. (2015) recently demonstrated that perception of grating in blindsight patients was correlated with the quality of visual projections from the lateral geniculate nucleus (LGN) to human MT complex (hMT+) confirming that at least some of the blindsight phenomenon can be associated

to inputs to the dorsal stream that are distinct from the main V1 origin. If one considers MT as the main cortical input of visual signals responsible for blindsight, it is interesting to know that inhibiting this brain region with TMS leads to an impairment of both chromatic and achromatic motion perception (Kaderali et al. 2015). Therefore visual inputs to MT may contribute to colour vision, especially when V1 is damaged. One may therefore hypothesize that using moving stimuli may increase the quantity or the quality of visual information that is projected to colour processing areas.
It has been shown that blindsight patients cannot discriminate motion produced in isoluminant conditions (Alexander & Cowey, 2013), but reciprocally it is not known whether the colour of moving stimuli would be better perceived than static hue. One may hypothesize that because moving stimuli are most readily processed by patients with blindsight their colour discrimination might be boosted.

S.A. was reported to us following clinical examination because he exhibited a preservation of the opticofacial winking reflex within his hemianopic field. He could also discriminate the presence of motion or reach fairly accurately to objects presented within his hemianopic field, while being assertive about his lack of visual experience. We explored whether his visual discrimination was specific to motion or could expand to other qualia of moving stimuli, e.g. colour.

2. Materials and methods

2.1. Case History

Following a stroke, SA was admitted to the neurology unit. He exhibited a complete somatosensory deficit on his left side including the face, a motor deficit of the left arm (3/5), a left hemianopia (automated perimetry) and a mild corticonuclear cataract. The proprioceptive deficit was responsible for a severe hand and foot ataxia precluding investigations for action-blindsight. His medical record included thyroidectomy and mild angor. His brain scan and MRI revealed an earlier ischemic lesion in the superficial sylvian territory on the left side and a recent right posterior lesion affecting the medial occipital lobe, the right thalamus and the right hippocampus. Figure 1 shows brain lesions on MRI and visual fields highlighting a left homonymous hemianopia.

2.2. Experimental design

Six healthy volunteers, (aged 22.5 $\pm$ 4.0), without any visual deficit has been recruited to form a control group with respect to colour and movement discrimination. One age-matched control individual (77) without neurological history has also been included in this study. All participants have given consent in accordance to Helsinki's ethic rules.

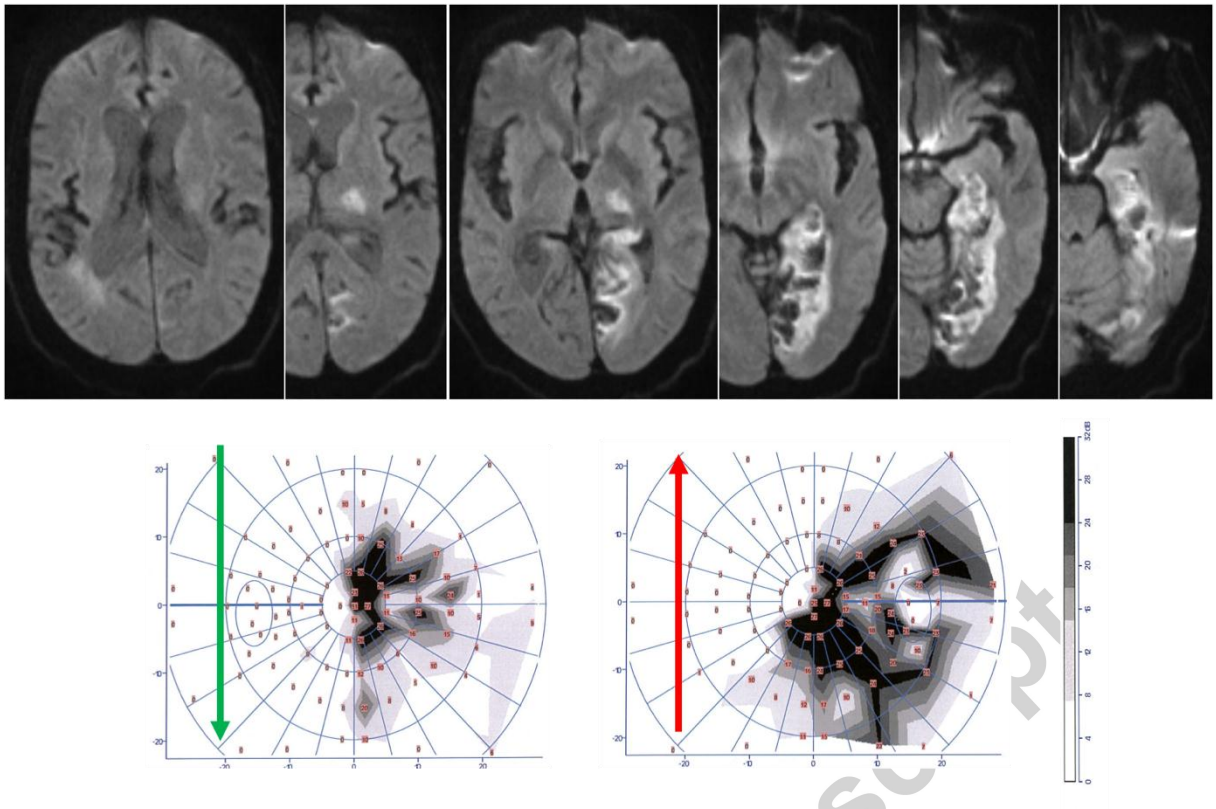

Figure 1 : patient lesions and visual field.

Upper row shows SA's MRI scans revealing a right posterior lesion affecting the medial occipital lobe, right thalamus and right hippocampus. Lower row shows SA's 30° central visual fields (automated static system, Metrovision ®, Pérenchies, France) where white parts refer to blind parts of the visual fields. The homonymous hemianopia was more severe for the left eye and some sparing inferior quadrant of the right eye was observed. Green and Red arrows exhibit the 20° angular position of stimuli presentation in the left hemianopic fied, i.e. beyond the relatively preserved area of the left inferior quadrant.

Healthy subjects and SA were seated in front of a uniform white wall, with their eyes 57 cm away from the wall surface. They were tested for their ability to discriminate between upward and downward motion using different colour hand-held laser pointers. During each trial they were asked to fixate a dot presented in front of them at their eye level without any head deviation while one experimenter visually controlled that fixation was maintained during each trial. When eye movement occurred occasionally, the trial was cancelled. A total of 13 such trials was cancelled in healthy subjects while none had to be cancelled with SA. Stimuli were presented between 20° and 25° in the left visual-hemifield as shown by green and red arrows in Figure 1; all subjects provided an oral response at the end of each trial. Preliminary testing showed that SA performed this double task easily in his healthy (right) visual field.

Two stimulus dimensions have then been taken into account: direction of movement (upward vs downward) and spot colour (green vs red). All combinations of a total of 48 experimental conditions (12 red-up, 12 red-down, 12 green-up and 12 green-down) were pseudo-randomly (latin square design) presented to subjects. Following Perenin and Rossetti (1996) preliminary trials were performed within the healthy visual field of SA in order to facilitate comprehension of the task. All subjects in this study had been made aware of all 4 possible stimuli. Red and green colours have been selected considering known wavelength sensibility in blindsight (Marzi, Mancini, Metitieri &

Savazzi, 2009). Laser spot were used in order to maximize stimulus contrast (Alexander & Cowey, 2012). Movement speed was chosen to be 40 cm.s^{-1} because residual visual sensibility is best shown with rather fast motion (Zeki & ffytche, 1998; Azzopardi & Cowey, 2001). Preliminary testing showed that SA performed this double task easily in his healthy (right) visual field.

2.3. Statistical analysis

This study consisted of pure Bernouilli experiments where subjects obtained success or defeat for each trial with unknown probability *p*. A total of 48 trials have been presented to subjects and results were analysed using a binomial test measuring the probability that *p* Bernouilli parameter well reproduced experimental data. For example, if a subject obtain 24 successes on 48 trials, the binomial test states that the probability of *p*=.5 is 1 whereas the probability of *p*=.8 is only 3.3x10^{-6}.

The response pattern of our patient concerning colour responses was unexpected because it varied among more than the two alternative responses expected from our forced-choice paradigm. Therefore, this specific issue was assessed by Pearson's χ^2 tests to determine the proportions' profile of his answers.

3. Results

Average number of successful answers for healthy control group are given in Table 1, together with SA's score and the age-matched control subject.

Table 1 : Average values of successful answers for 48 presentations of colours and directions of motion

	control group		patient SA	age-matched
	mean	st. dev		
Spot colour	47.0	0.89	12	48
Motion direction	47.8	0.41	47	48

In healthy subjects, the task was very easy and individuals reached nearly 100% correct. The binomial test on healthy subjects group exhibited more than 99.99% chances that all control subjects's responses were correct (*p* parameter equals 1). The age-matched healthy control performed with 100% in each task and the binomial test shows the same subject's success level confirming the easiness of the task. SA performed in the same range for motion discrimination with 47/48 correct, even though his responses were produced very spontaneously and immediately around the end of the stimulation. In only one trial he initially answered 'down' and then changed his answer into the correct one 'up'. This response was therefore considered as correct. With only one true error, the binomial test stated more than 99.99% chances of p=1 successful answers and confirmed that he had a clear sense of motion even without proper visual experience. By contrast, for the colour discrimination task SA exhibited obvious difficulties. First, he assumed that he could not guess about the colour because of the lack of any phenomenological experience of it. Second, we tried to compensate for this difficulty by asking him to answer first about colour and second about motion, as during preliminary trials he would spontaneously reply about motion and then only about colour if at all. Despite reiterated instructions he overall answered about motion first in 45/48 trials. Out of the other three colour responses only one was correct. The other colour responses were delayed by several seconds due to hesitations. Even if the instruction to provide a response by chance was repeated, he could not utter a colour name in 8/48 trials (blank response). Altogether the correct

colour answer was provided in 12/48 trials. In 18 trials, colours other than red and green were called (mostly white and blue) and regularly SA was reminded that only red and green were used. A response bias toward green colour was observed and the total number of red or green responses was 22, rendering the 12 correct responses close to the 50% random level of performance. In order to clarify this response pattern, patient's 5 answers (green, red, white, blue, and no answer at all) were compared between the 2 experimental conditions (green and red stimuli). We then found that 1) answers produced for each stimulus colour were compatible with equally distributed proportions between 5 possibilities ($\chi^2(4)=7.6$, p=.11) and 2) patterns of answers were not different between green and red stimuli ($\chi^2(4)=4.14$, p=.38). This result corroborated the random response pattern of SA concerning the colour determination.

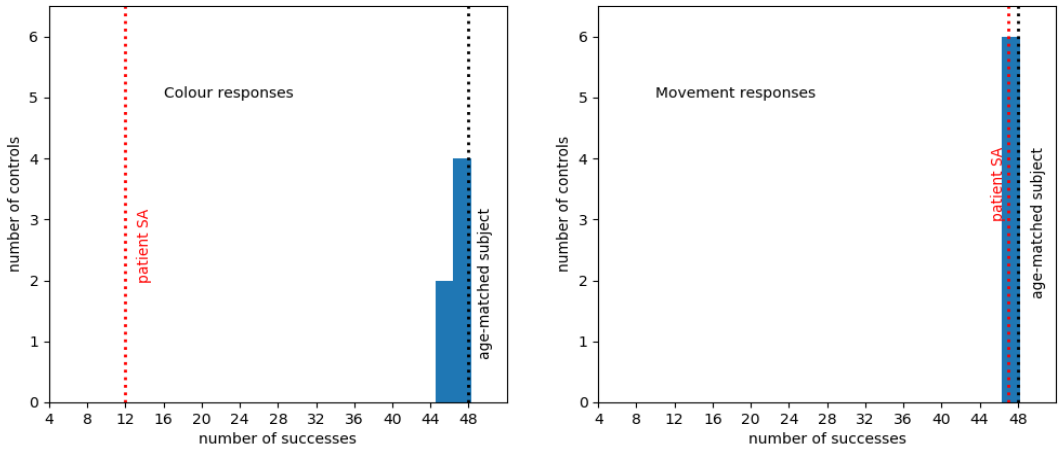

Figure 2: participants' responses.
This figure shows the distribution of participants as a function of probability of success for colour stimuli (left panel) and motion stimuli (right panel). Nearly all healthy subjects achieved 100% correct, showing a total of two errors for colour and only one for motion. The age-matched control subject was 100% correct for the two visual qualia whereas SA was in the control range for motion and random for colour.

Figure 2 presents the distance in number of successes between the individual cases and the control-group distribution. Whereas movement responses of SA were at the same level as those of the control group, his colour responses did not differ from chance. The age-matched subject was following the same pattern of response than the control group; this allows to state that the huge difference observed in answers concerning colours should not be attributed to aging. Note that because of non-normally distributed data, no significant effect size may be given, only relative risk (or chance) is relevant: SA was 4 time less likely to discriminate colours than motion direction with respect to control group and age-matched subject.

4. Discussion

The present pattern of result indicates that SA was clearly able to sense and discriminate motion direction while being unable to sense and guess about the colour of this moving stimulus. First, this suggests that distinct processes or pathways may be at work in motion-blindsight and in colour-blindsight (Danckert & Rossetti, 2005) and confirms that colour discrimination may result from a

more complex and thus slower processing (Pisella, Arzi & Rossetti, 1998). Second, it expands the finding that motion could not be discriminated from hue in isoluminant conditions: reciprocally we find that hue cannot be discriminated for moving stimuli. As emphasized by Smits, Heywood, Seijdel, Kentridge & de Haan (2018), to validate the 3-level taxonomy of blindsight further explorations of correlation and interaction between each level is required. Our result support the idea that attention-blindsight and perception-blindsight correspond to different entities. Lesion of the LGN (lateral geniculate nucleus) abolishes visual responses found in the extra-striate cortex of chronic V1-lesioned monkeys (Schmid et al. 2010). Transient lesions of V1 supress neuronal activity toward the ventral stream, where colour-coding neurons are found, while residual activity can still be recorded in the inputs of the dorsal stream, where neurons coding motion can be found (Girard et al. 1991, 1992). Therefore, projections from the LGN to the extra-striate cortex that bypass V1 must account for the existence of perception-blindsight as confirmed by Schmid et al. (2010) in monkeys and Ajina et al. (2015) in humans. Even if monkey projections from LGN to extrastriate areas do not seem to be decisively different for areas such as V4 and V5 (Schmid et al. 2010), the current results confirm that in humans the strength of the residual activity responsible for blindsight may be different for motion and colour. This confirmation is also in agreement with Kaderali, Kim, Reynaud & Mullen (2015) who showed that TMS inhibition of the motion hMT+ area impaired significantly global motion detection of healthy subjects irrespective of the chromatic dimension of the stimuli. Thus, the wavelength sensitivity described in perception-blindsight by Marzi, Mancini, Metitieri & Savazzi (2009) remains difficult to account for in terms of neuronal pathways. One potential candidate is heralded by Leh, Ptito, Schönwiesner, Chakravarty & Mullen (2010) who have shown that S-cone projections seem to be absent in subcortical-to-cortical pathways involved in attention-blindsight. Further investigations are needed to improve our understanding of colour perception-blindsight. As far as action blindsight is concerned, the existence of LGN projections to MT (Ajina et al, 2015) appears to provide a plausible input to the dorsal stream involved in motor responses (Danckert et al, 2003). Other anatomical pathways may also play a role: several studies (Celeghin et al, 2017 ; Silvanto et al, 2007 ; Bridge et al, 2008 ; Celeghin et al, 2015) support a possible compensation by MT from the healthy hemisphere via information transfer through the corpus callosum. In the case of SA, such a transfer is indeed possible, but the current experiment is not informative on this point. In addition, recent work (Mikiellidou et al, 2017) has highlighted the possible intervention of the prostriata area in motion detection appearing in peripheral vision. However, the speed range involved in this motion detection (500°/s) is much higher than the speed involved in our experiment (around 40°/s) and it is likely that this cortical area plays a rather minor role in the case of SA.

Additionally, our case study suggests that colour processing may not be boosted by moving stimuli, as if residual blindsight activity for motion would be much stronger than for colour even when these features are processed at the same time and belong to the same stimulus. Blindsight for motion has been repeatedly associated with type 1 blindsight, i.e. leading to a conscious experience, whereas blindsight for colour has not (Kentridge, 2015). On another line of thought, this result is also compatible with the view that blindsight may be viewed as the complementary condition to optic ataxia (Rossetti, Pisella, McIntosh, 2017). As a matter of facts it seems that several features of blindsight may be viewed as mirror images of optic ataxia: in blindsight preserved abilities are prominently representative of the dorsal stream functions, visuomotor performance preserved in blindsight seem to match those which are defective in optic ataxia (Pisella et al. 2000; Danckert & Rossetti 2003; Rossetti, Pisella & Vighetto, 2003), and delayed responses may impair blindsight

motor responses (Rossetti, 1998; but see Carey, Trevethan, Weiskrantz & Sahraie, 2012) whereas they paradoxically improve optic ataxia patient's performance (Milner et al. 2001; Revol et al. 2003; Rossetti et al. 2005). These dissociations suggest that optic ataxia is more likely to be dissociated from blindsight that from visual agnosia (Danckert & Rossetti 2005; Pisella, Binkofski, Lasek, Toni & Rossetti, 2006; Rossetti, Pisella, McIntosh, 2017). However, a crucial argument needs to be further investigated to support this proposal. The visual qualia that is most saliently processed by patients with blindsight is motion and there is by now only limited information available in the literature about motion processing deficits in optic ataxia. Low level motion perception (e.g. optic flow) was preserved but more complex motion processing (apparent motion (phi phenomenon), multiple dots tracking) were significantly altered in a patient with a bilateral optic ataxia patient (Michel & Henaff, 2004) as well as in a group of patients with various parietal lesions (Battelli et al., 2001). Specific explorations of motion processing in unilateral optic ataxia should clarify whether it can be considered as a mirror image of blindsight.

References

Ajina, S., Pestilli, F., Rokem, A., Kennard, C., & Bridge, H. (2015). Human blindsight is mediated by an intact geniculo-extrastriate pathway. *Elife, 4*, e08935.

Alexander, I., & Cowey, A. (2013). Isoluminant coloured stimuli are undetectable in blindsight even when they move. *Experimental brain research, 225*(1), 147-152.

Azzopardi, P., & Cowey, A. (2001). Motion discrimination in cortically blind patients. *Brain*, 124(1), 30-46.

Battelli, L. Cavanagh P., Intriligator J., Tramo, M.J. Hénaff, M.A. Michel F., Barton J.J. (2001) Unilateral right parietal damage leads to bilateral deficit for high-level motion, *Neuron* 32, 985–995.

Carey, D. P., Trevethan, C. T., Weiskrantz, L., & Sahraie, A. (2012). Does delay impair localisation in blindsight? *Neuropsychologia*, 50(14), 3673-3680.

Celeghin, A., Barabas, M., Mnacini, F., Bendini, M., Pedrotti, E., Prior, M., Cantagallo, A., Savazzi, S., & Marzi, C.A. (2015) Speeded manual responses to unseen visual stimuli in hemianopic patients: What kind of blindsight? *Consciousness and Cognition* 32, 6–14.

Celeghin, A., Diano, M., de Gelder, B, Weiskrantz, L., Marzi, C.A., Tamietto M. (2017). The intact hemisphere and corpus callosum compensate for visuo-motor functions after early visual cortex damage. *Proceedings of the National Academy of Sciences, U.S.A.*, 114(48), E10475-83

Danckert, J. & Rossetti, Y. (2005). Blindsight in action: what can the different sub-types of blindsight tell us about the control of visually guided actions ? *Neuroscience and Biobehavioral Reviews*, 29, 1035-1046.

Danckert J, Revol P, Pisella L, Krolak-Salmon P, Vighetto A, Goodale MA, Rossetti Y (2003). Measuring unconscious actions in action-blindsight: exploring the kinematics of pointing movements to targets in the blind field of two patients with cortical hemianopia. *Neuropsychologia 41*(8), 1068-81.

Girard, P., Salin, P. A., & Bullier, J. (1991). Visual activity in areas V3a and V3 during reversible inactivation of area V1 in the macaque monkey. *Journal of Neurophysiology, 66*(5), 1493-1503.

Girard, P., Salin, P. A., & Bullier, J. (1992). Response selectivity of neurons in area MT of the macaque monkey during reversible inactivation of area V1. *Journal of Neurophysiology, 67*(6), 1437-1446.

Kaderali, S., Kim, Y. J., Reynaud, A., & Mullen, K. T. (2015). The role of human brain area hMT+ in the perception of global motion investigated with repetitive transcranial magnetic stimulation (rTMS). *Brain stimulation, 8*(2), 200-207.

Kentridge, R. W. (2015). What is it like to have type-2 blindsight? Drawing inferences from residual function in type-1 blindsight. *Consciousness and cognition, 32*, 41-44.

Leh, S. E., Ptito, A., Schönwiesner, M., Chakravarty, M. M., & Mullen, K. T. (2010). Blindsight mediated by an S-cone-independent collicular pathway: an fMRI study in hemispherectomized subjects. *Journal of Cognitive Neuroscience, 22*(4), 670-682.

Marzi, C. A., Mancini, F., Metitieri, T., & Savazzi, S. (2009). Blindsight following visual cortex deafferentation disappears with purple and red stimuli: a case study. *Neuropsychologia, 47*(5), 1382-1385.

Mikellidou, K., Kurzawski, J.W., Frijia, F., Montanaro, D., Greco, V., Burr, D.C., & Morrone, M.C. (2017). Area prostriata in the human brain. *Current Biology*, 27, 3056–3060.

Milner, A. D., Dijkerman, H. C., Pisella, L., McIntosh, R. D., Tilikete, C., Vighetto, A., & Rossetti, Y. (2001). Grasping the past: Delay can improve visuomotor performance. *Current Biology, 11*(23), 1896-1901.

Morland, A. B., Jones, S. R., Finlay, A. L., Deyzac, E., Lê, S., & Kemp, S. (1999). Visual perception of motion, luminance and colour in a human hemianope. *Brain, 122*(6), 1183-1198.

Pöppel, E., Held, R. & Frost, D. (1973). Residual visual function after brain wounds involving the central visual pathways in man. *Nature, 243*(5405), 295-296.

Pegna, A. J., Khateb, A., Lazeyras, F., & Seghier, M. L. (2005). Discriminating emotional faces without primary visual cortices involves the right amygdala. *Nature neuroscience, 8*(1).

Perenin, M.T. & Jeannerod, M. (1975). Residual vision in cortically blind hemifields. *Neuropsychologia, 13*, 1-7.

Perenin, M.T. & Jeannerod, M. (1978). Visual function within the hemianopic field following early hemidecortication in man. I Spatial localization. *Neuropsychologia, 16*, 1-13.

Perenin, M.T. & Rossetti, Y. (1996). Grasping without form discrimination in a hemianopic field. *Neuroreport, 7*, 793-797.

Pisella, L., Arzi, M., & Rossetti, Y. (1998). The timing of color and location processing in the motor context. *Experimental Brain Research, 121*(3), 270-276.

Pisella, L., Grea, H., Tilikete, C., Vighetto, A., Desmurget, M., Rode, G., Boisson, D. & Rossetti, Y. (2000). An 'automatic pilot'for the hand in human posterior parietal cortex: toward reinterpreting optic ataxia. *Nature neuroscience, 3*(7), 729-736.

Pisella, L., Binkofski, F., Lasek, K., Toni, I., & Rossetti, Y. (2006). No double-dissociation between optic ataxia and visual agnosia: Multiple sub-streams for multiple visuo-manual integrations. *Neuropsychologia, 44*(13), 2734-2748.

Revol P, Rossetti Y, Vighetto A, Rode G, Boisson D, Pisella L. (2003) Pointing errors in immediate and delayed conditions in unilateral optic ataxia. *Spat Vis.* 16(3-4):347-64.

Rossetti, Y. (1998). Implicit short-lived motor representations of space in brain damaged and healthy subjects. *Consciousness and cognition,* 7(3), 520-558.

Rossetti, Y., Pisella, L., & Vighetto, A. (2003). Optic ataxia revisited. *Experimental Brain Research, 153*(2), 171-179.

Rossetti, Y., Revol, P., McIntosh, R., Pisella, L., Rode, G., Danckert, J., Tilikete, C., Dijkerman, H.C., Boisson, D., Vighetto, A., Michel, F. & Milner, A.D. (2005). Visually guided reaching: bilateral posterior parietal lesions cause a switch from fast visuomotor to slow cognitive control. Neuropsychologia, 43(2), 162-177.

Rossetti, Y., Pisella, L., & McIntosh, R. D. (2017). Rise and fall of the two visual systems theory. *Annals of physical and rehabilitation medicine, 60*(3), 130-140.

Sahraie, A., Trevethan, C. T., MacLeod, M. J., Urquhart, J., & Weiskrantz, L. (2013). Pupil response as a predictor of blindsight in hemianopia. *Proceedings of the National Academy of Sciences, 110*(45), 18333-18338.

Smits, A.R., Heywood, C.A., Seijdel, N., Kentridge, R.W. & de Haan, E.H.F. (2018). Action Blindsight and Antipointing in a Hemianopic Patient., submitted.

Schmid, M. C., Mrowka, S. W., Turchi, J., Saunders, R. C., Wilke, M., Peters, A. J., ... & Leopold, D. A. (2010). Blindsight depends on the lateral geniculate nucleus. *Nature, 466*(7304), 373-377.

Silvanto, J., Cowey, A., Lavie, N., Walsh, V. (2007) Making the blindsighted see. *Neuropsychologia* 4, 3346–3350.

Stoerig, P., & Cowey, A. (1989). Wavelength sensitivity in blindsight. *Nature, 342*(6252), 916-918.

Trevethan, C. T., Sahraie, A., & Weiskrantz, L. (2007). Form discrimination in a case of blindsight. *Neuropsychologia, 45*(9), 2092-2103.

Weiskrantz, L., Warrington, E.K., Sanders, M.D., Marshall, J. (1974). Visual capacity in the hemianopic field following a restricted occipital ablation. *Brain, 97*, 709-728.

Weiskrantz, L. (1986). Blindsight: A Case Study and Implications. Oxford University Press, Oxford.

Weiskrantz, L. (1996). Blindsight revisited. *Current opinion in neurobiology, 6*(2), 215-220.

Weiskrantz, L. (2000). Blindsight: implications for the conscious experience of emotion. *Cognitive neuroscience of emotion*, 277-295.

Weiskrantz, L. (2009). Blindsight: a case study spanning 35 years and new developments, 3rd edn. Oxford University Press, Oxford.

Weiskrantz, L. (2009). Is blindsight just degraded normal vision?. *Experimental brain research, 192*(3), 413-416.

Zeki, S., & Ffytche, D. H. (1998). The Riddoch syndrome: insights into the neurobiology of conscious vision. *Brain: a journal of neurology, 121*(1), 25-45.

Zihl, J. & Werth, R. (1984a). Contributions to the study of 'blindsight' I. Can stray light account for saccadic localization in patients with postgeniculate visual field defects ? *Neuropsychologia, 22*, 1-11.

Zihl, J. & Werth, R. (1984b). Contributions to the study of 'blindsight' II. The role of specific practice for saccadic localization in patients with postgeniculate visual field defects. *Neuropsychologia, 22*, 13-22.

Highlights:

- A neurological patient with visual cortex lesion showed exhibited preserved ability to process motion direction (up vs down) but not the colour (red vs green) of a moving dot.
- This case study shows that neural processing of the motion and the colour of a visual stimuli can be dissociated.