CORTEX 132 (2020) 113–134

Available online at www.sciencedirect.com

ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex

Research Report

What cortical areas are responsible for blindsight in hemianopic patients?

Javier Sanchez-Lopez [a,*], Nicolò Cardobi [a], Caterina A. Pedersini [a], Silvia Savazzi [a,b,c] and Carlo A. Marzi [a,c]

[a] Physiology and Psychology Section, Department of Neuroscience, Biomedicine and Movement Sciences, University of Verona, Verona, Italy
[b] Perception and Awareness (PandA) Laboratory, University of Verona, Verona, Italy
[c] National Institute of Neuroscience, Verona, Italy

ARTICLE INFO

Article history:
Received 24 February 2020
Reviewed 9 April 2020
Revised 7 June 2020
Accepted 7 August 2020
Action editor Yves Rossetti
Published online 26 August 2020

Keywords:
Blindsight
Perceptual awareness
Visual cortex
Precuneus
Posterior cingulate gyrus

ABSTRACT

The presence of above-chance unconscious behavioral responses following stimulus presentation to the blind hemifield of hemianopic patients (blindsight) is a well-known phenomenon. What is still lacking is a systematic study of the neuroanatomical bases of two distinct aspects of blindsight: the unconscious above chance performance and the phenomenological aspects that may be associated. Here, we tested 17 hemianopic patients in two tasks i.e. movement and orientation discrimination of a visual grating presented to the sighted or blind hemifield. We classified patients in four groups on the basis of the presence of above chance unconscious discrimination without or with perceptual awareness reports for stimulus presentation to the blind hemifield. A fifth group was represented by patients with interruption of the Optic Radiation. In the various groups we carried out analyses of lesion extent of various cortical areas, probabilistic tractography as well as assessment of the cortical thickness of the intact hemisphere.

All patients had lesions mainly, but not only, in the occipital lobe and the statistical comparison of their extent provided clues as to the critical anatomical substrate of unconscious above-chance performance and of perceptual awareness reports, respectively. In fact, the two areas that turned out to be critical for above-chance performance in the discrimination of moving versus non-moving visual stimuli were the Precuneus and the Posterior Cingulate Gyrus while for perceptual awareness reports the crucial areas were Intracalcarine, Supracalcarine, Cuneus, and the Posterior Cingulate Gyrus. Interestingly, the proportion of perceptual awareness reports was higher in patients with a spared right hemisphere. As to probabilistic tractography, all pathways examined yielded higher positive values for patients with perceptual awareness reports. Finally, the cortical thickness of the intact hemisphere was greater in patients showing above-chance performance than in those at chance. This effect is likely to be a result of neuroplastic compensatory mechanisms.

* *Corresponding author*. Strada Le Grazie 8, 37134, Verona, Italy.
E-mail addresses: javier.sanchezlopez@univr.it (J. Sanchez-Lopez), nicolo.cardobi@gmail.com (N. Cardobi), caterinaannalaura.pedersini@univr.it (C.A. Pedersini), silvia.savazzi@univr.it (S. Savazzi), carloalberto.marzi@univr.it (C.A. Marzi).
https://doi.org/10.1016/j.cortex.2020.08.007

1. Introduction

A unilateral lesion of the post-chiasmatic visual pathways and/or the visual cortex often results in a visual loss in the contralateral hemifield of both eyes, known as homonymous hemianopia (HH; see Bouwmeester, Heutink, & Lucas, 2007; Goodwin, 2014). In 1973 Poppel, Held, & Frost and, subsequently, Weiskrantz, Warrington, Sanders, and Marshall (1974) described the existence in some hemianopic patients of unconscious visually triggered responses to stimuli presented to the blind hemifield. This phenomenon was defined "blindsight" by Weiskrantz (see for review Weiskrantz, Barbur, & Sahraie, 1995; Weiskrantz, 1996) and has become an important and controversial tool in the study of the neural mechanisms of visual perceptual awareness (Cowey, 2010; Overgaard, 2011; Overgaard & Mogensen, 2015) as well as for devising visual rehabilitation techniques (Bouwmeester et al., 2007; Dundon, Bertini, Làdavas, Sabel, & Gall, 2015; Zihl, 2010).

The "pure" form of blindsight, where above-chance responses are carried out by sheer guessing (according to participant's report) in absence of any awareness, is named blindsight type 1. However, if blindsight patients report the presence of a feeling that a stimulus has been presented, then this occurrence is defined blindsight type 2 (Sahraie, Hibbard, Trevethan, Ritchie, & Weiskrantz, 2010; Weiskrantz et al., 1995). Whether the nature of this feeling is visual or not is being hotly debated (Brogaard, 2015; Foley, 2015; Kentridge, 2015) and probably both kinds may be present in different patients or even concomitantly in the same patient in different tasks. It is important to mention that another classification has been proposed on the basis of the kind of task yielding blindsight (Danckert, Tamietto, & Rossetti, 2019; Danckert & Rossetti, 2005) thus highlighting the possibility of different kinds of blindsight depending upon the specific task.

After all these years of research, the neural correlates of blindsight in general, and of its different forms, in particular, remain unclear. Some authors have suggested that preserved areas within the primary visual cortex (V1) could be responsible (Campion, Latto, & Smith, 1983; Fendrich, Wessinger, & Gazzaniga, 2001). However, this hypothesis is not universally accepted and has been contradicted by studies where blindsight was demonstrated in total absence of V1 as in hemispherectomy patients (e.g. Georgy, Celeghin, Marzi, Tamietto, & Ptito, 2016; Tomaiuolo, Ptito, Marzi, Paus, & Ptito, 1997). More likely is the possibility that extrastriate visual areas might subserve blindsight via different pathways namely, i) projections from the superior colliculus (SC) and the pulvinar to extrastriate areas including the human visual motion area known as hMT+ (e.g. in humans Tran et al., 2019; in monkeys Kinoshita et al., 2019) ii) projections from the lateral geniculate nucleus (LGN) to extrastriate areas (again including hMT+) also bypassing V1 (the geniculo extrastriate pathway; Ajina & Bridge, 2017, 2018; Ajina, Pestilli, Rokem, Kennard, & Bridge, 2015; Schmid et al., 2010; Smits et al., 2019). The evidence in favor of these two alternative pathways is controversial but of course a likely possibility is that both contribute to different forms of blindsight. It should be noted that most studies on blindsight have been carried out in single (or few) patients although there are exceptions (e.g. Ajina & Bridge, 2018; Celeghin et al., 2015; Garric et al., 2019). Of course, single-case studies represent a classic method that has contributed enormously to advances in many fields of cognitive neuropsychology, see Mazzi and Savazzi (2019). However, as to understanding the neural bases of a given psychological process it is a clear limitation. In this light, trying to relate different aspects of blindsight to distinct or partially overlapping brain areas is actually the main aim of the present study and this is possible only by using a reasonable number of patients. In particular, the novel aspect of our study is to put emphasis on cortical areas in addition to visual pathways as prevalently done until now.

Moreover, an important question that deserves specific investigation concerns the neural bases of the subjective phenomenological reports that may accompany blindsight. In this respect, recently, a study by Mazzi, Tagliabue, Mazzeo, and Savazzi (2019) has systematically described the presence of graded visual sensations in the blind field of hemianopic patients which underlines the important concept that the presence of stimulus related subjective feeling is not an "all or none phenomenon" and can have various levels of perceptual awareness (Mazzi, Bagattini, & Savazzi, 2016; Mazzi, Savazzi, & Silvanto, 2019). Another general question about the phenomenological aspects of blindsight concerns whether they might "simply" represent degraded vision rather than the output of neural structures partially "devoid" of perceptual awareness (Overgaard, Fehl, Mouridsen, Bergholt, & Cleeremans, 2008). Thus, blindsight as well as non-blindsight hemianopic patients with hints of perceptual awareness represent a good opportunity to cast light on these controversies. An example is the recent paper by Garric et al. (2019; see Phillips, 2019 for a critical review) who have coined the term *blindsense* to define patients who did not show above-chance discrimination performance but reliably reported the occurrence of the stimulus. This might represent an example of degraded vision that was not sufficient to enable above chance performance (see below a comparison with one of our patients' group). The anatomical analysis in two patients, one with and the other without *blindsense* revealed lesion of only area 17 and 18 in the former and a larger lesion involving areas 17, 18, 19, 29 and 30 in the latter and this is obviously an important difference.

In the present study we divided hemianopic patients in different groups on the basis of the presence of above-chance discrimination performance accompanied or not by perceptual awareness reports and correlated these characteristics with location and extent of the damaged cortical areas and visual pathways. To analyze cortical areas is a crucial aspect of our study because so far, theories on the neural bases of blindsight have been mainly based on the role of subcortical-cortical pathways but very little effort has been done to specify what cortical areas are responsible, apart from human motion temporal area (hMT+), for moving stimuli.

2. Materials and methods

In the next sections we report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to

data analysis, all manipulations, and all measures in the study.

2.1. Participants

17 patients with post-chiasmatic lesions resulting in homonymous hemianopia were included in the study (Females = 4; Mean age = 59.2, SD = 8.6). Nine patients had right hemianopia, seven left hemianopia and one with bilateral altitudinal hemianopia. Six showed quadrantanopia (three in the upper and three in the lower visual field), 10 had homonymous hemianopia and one bilateral altitudinal hemianopia. In our cohort 82.35% of the cause of hemianopia was stroke (see Table 1 for details), 11.76% traumatic and 5.88% related to brain surgery. This is in broad accord with the data of Zhang, Kedar, Lynn, Newman, and Biousse (2006) in a sample of 904 cases. To be recruited for the study patients must have had a diagnosis of homonymous hemianopia at least three months before testing. Clinical visual campimetry and structural MRI documenting the brain damage were provided by the patients.

A further more detailed MRI acquisition was carried out at the Radiology Unit of Verona Borgo Roma Hospital which is part of our University.

Exclusion criteria were pre-existing neurologic or psychiatric disorders, cognitive impairments, alcohol or drugs addiction and neuropsychological attention disorders such as unilateral spatial hemineglect (see Sanchez-Lopez et al., 2017 for a detailed description of hemineglect testing). All participants were right handed and had normal or corrected to normal visual acuity. See Table 1 and Fig. 1 for detailed clinical information and Table 2 for quantitative description of the proportion of lesions. Given the rather strict requirements for recruiting patients and the broad accord with the clinical-anatomical characteristics of the sample of Zhang et al. (2006) study we consider that the number of patients recruited is suitable for our cross-sectional study, which by the way, is within the usual range of patients' number in functional neuroimaging experimental neuropsychology studies (see review by Roalf & Gur, 2017).

Participants were asked to sign an informed consent form. The study was approved by the Ethics Committee of the European Research Council and of the Verona Azienda Ospedaliera Universitaria Integrata (AOUI), and has been performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments.

2.2. Discrimination tasks

Stimuli: black and white square wave gratings with a width = 4° and height = 4°. Michelson contrast = 1, spatial frequency = .875 c/°, stimulus duration = 250 msec, temporal frequency of moving stimuli = 8.33°/s. Background luminance was the same as the mean luminance of the grating (17.7 cd/m^2). Participants were seated with the head positioned on a chin rest and the eyes at 57 cm from a monitor in a dimly lit room and were asked to keep fixation steady on a central fixation point. Eye movements were monitored by means of a closed-circuit TV.

The behavioral paradigm (see Fig. 2A, B) consisted of two tasks: A **movement discrimination task** with horizontal stationary or moving (downward) grating's bars. Participants were to discriminate between moving and stationary stimuli by pressing one of two keyboard keys. An **orientation discrimination task** with stationary vertical or horizontal oriented gratings. Participants were to discriminate stimulus orientation by pressing one of two different keys. Both tasks were forced-choice and the stimuli were presented either in the blind or the sighted visual field in different blocks. Each participant performed a maximum of 480 trials on each task, 240 in the blind and 240 in the sighted hemifield. Trials were organized in blocks of 80 stimulus trials (50% per condition) and 16 catch trials where no stimulus was presented. When participants missed or anticipated the response by pressing the key quicker than 250 msec the trial was repeated until a valid response was obtained. Stimulus was positioned in a location depending on the site of the blind area as determined on the basis of clinical campimetry and of a visual mapping carried out in the lab, see Sanchez-Lopez et al., 2017, and Table 1 in Appendix A for further details. At the end of each blind field trial, patients were to verbally indicate the level of perceptual awareness by using a three-level scale: 1 = "I saw nothing", 2 = "I realized that there was a stimulus but I could not discriminate it", 3 = "I clearly saw the stimulus". In the rare occasion of response 3 the trial was canceled because invariably corresponded to a shift of fixation.

Percentage of correct responses, errors and reaction time (RT) were recorded for both visual fields and tasks. Additionally, for the blind hemifield, we recorded the percentage of subjective perceptual awareness reports in the three-level scale.

2.3. MRI acquisition and preprocessing

For MRI acquisitions, a 1.5 T scanner (Philips Ingenia, Philips Healthcare, Eindhoven, The Netherlands) with a 15-channels head coil was used. To obtain an anatomical image and locate the brain lesion a whole brain high-resolution ($1 \times 1 \times 1$ mm^3) Ultrafast Gradient Echo 3D T1-weighted image was acquired for all patients (except RC). To estimate the volume of brain lesion, masks using the T1-weighted native brain image of each patient were manually drawn by using the software ITK-SNAP (Yushkevich et al., 2006). Masks of the lesions were registered from the native to the standard MNI space with a spatial resolution of 1 mm using linear transformations (FLIRT). The distribution and extension of the volume of the lesion were estimated by quantifying the percentage of overlap between the masks and the ROIs extracted from occipital, temporal and parietal lobes on the basis of the probabilistic atlas of human cortical brain areas Harvard–Oxford (see Table 2) as implemented in FMRIB Software Library (FSL; Behrens et al., 2003; Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012; Woolrich et al., 2009). To extract the masks for each ROI from the Atlas, a threshold value equal to 5 was used for avoiding overlap between areas. For the final analysis 10 occipital ROI (Intracalcarine, Supracalcarine, Lateral Occipital inferior, Lateral Occipital superior, Occipital Fusiform Gyrus, Occipital Pole, Lingual Gyrus, Cuneus, Temporal Occipital Fusiform, Middle Temporal Gyrus temporo-occipital) plus the Precuneus and the Posterior Cingulate Gyrus were used (see below).

Table 1 – Clinical details of patients.

Patient	Age	Months from event	Gender	Neuroradiological Description	Visual Defect
GB	65	4	M	Ischemic lesion involving the right calcarine fissure, lingual and fusiform giri.	Left lateral hemianopia
RF	52	3	M	Ischemic lesion involving the anterior and middle portion of left calcarine fissure, lingual gyrus and posterior part of fusiform gyrus.	Right lateral hemianopia.
AP	47	6	M	Lesion as result of a surgery involving the inferior anterolateral portion of right occipital lobe with extension in the posterior part of temporal lobe, inferior portion of the right optic radiation and the upper part of right cerebellar hemisphere.	Upper left quadrantanopia.
LF	49	29	F	Ischemic lesion involving the cortex of the anterior half of right calcarine fissure to the origin of parieto-occipital fissure.	Upper left quadrantanopia.
DD	56	14	M	Traumatic lesion involving the left infero-lateral part of the occipital lobe with extension in the lingual and fusiform giri. Laterally, the lesion is below the lateral occipital sulcus.	Upper right quadrantanopia
AM	65	34	M	Ischemic/hemorrhagic bilateral median para-sagittal occipital ischemic lesions involving the lingual gyrus, more evident in the right side. On the right side, a thinning of the anterior portion of calcarine cortex is observed.	Bilateral Altitudinal Hemianopia.
GA	61	20	M	Ischemic lesion involving the left parieto-occipital lobe. In the occipital lobe, the lesion laterally involves the superior, middle, inferior and descending occipital gyri. In the medial portion, it involves the cuneus, and the occipital pole. Small portion of the white matter of the posterior part of optic radiation is damage as well.	Lower right quadrantanopia.
HE	60	4	M	Ischemic lesion involving the medial part of right occipital lobe with peri-calcarine distribution. The alterations are predominately in the upper part of calcarine fissure with extension to the cuneus.	Left lateral hemianopia.
ML	57	15	M	Ischemic bilateral lesion of both inferior part of occipital lobes, more evident on the left side where the occipital pole, lingual and fusiform gyri are involved. On the right side, the lesion involves the occipital pole.	Right lateral hemianopia.
SL	48	79	F	Ischemic/hemorrhagic lesion involving the median para-sagittal portion of the left occipital lobe. The lesion involves the lingual gyrus with peri-calcarine fissure distribution.	Right lateral hemianopia.
AN	54	32	M	Lesion due a hemorrhagic event involving the left temporo-parietal lobe with extension to the occipital lobe in the superior and middle occipital gyri. Involvement of the upper part of left optic radiation.	Right lateral hemianopia.
BC	69	6	M	Ischemic lesion involving the medial portion of right occipital lobe and over the parieto-occipital fissure. There is an involvement of the lingual and fusiform gyri up to the occipital pole with alterations of the calcarine fissure.	Lower left quadrantanopia.
FB	49	17	F	Traumatic lesion involving the right temporal and parietal lobe with development of a poro-encephalic cavity in temporal lobe and ex-vacuo dilatation of right lateral ventricle. In the occipital lobe the lesion involves the superior and part of the middle occipital gyri. Right optic radiation was interrupted. The other parts of occipital lobe are preserved.	Left lateral hemianopia.
GS	75	6	M	Ischemic lesion involving the antero-superior part of the right calcarine fissure with a partial involvement of the cuneus.	Left lateral hemianopia.
LB	62	5	F	Ischemic lesion in the vascular territory of left posterior cerebral artery involving all the occipital lobe including the left calcarine fissure.	Lower right quadrantanopia.
RC	71	8	M	Ischemic lesion involving the medial portion of right occipital lobe. There is an involvement of the lingual and fusiform gyri till the occipital pole with alterations in the inferior part of calcarine fissure.	Left lateral hemianopia.
LC	67	19	M	Lesion due a hemorrhagic event over right temporal and parietal lesion with posterior extension to the white matter of occipital lobe involving the lateral part of optic radiation. The right calcarine fissure is normal.	Left lateral hemianopia.

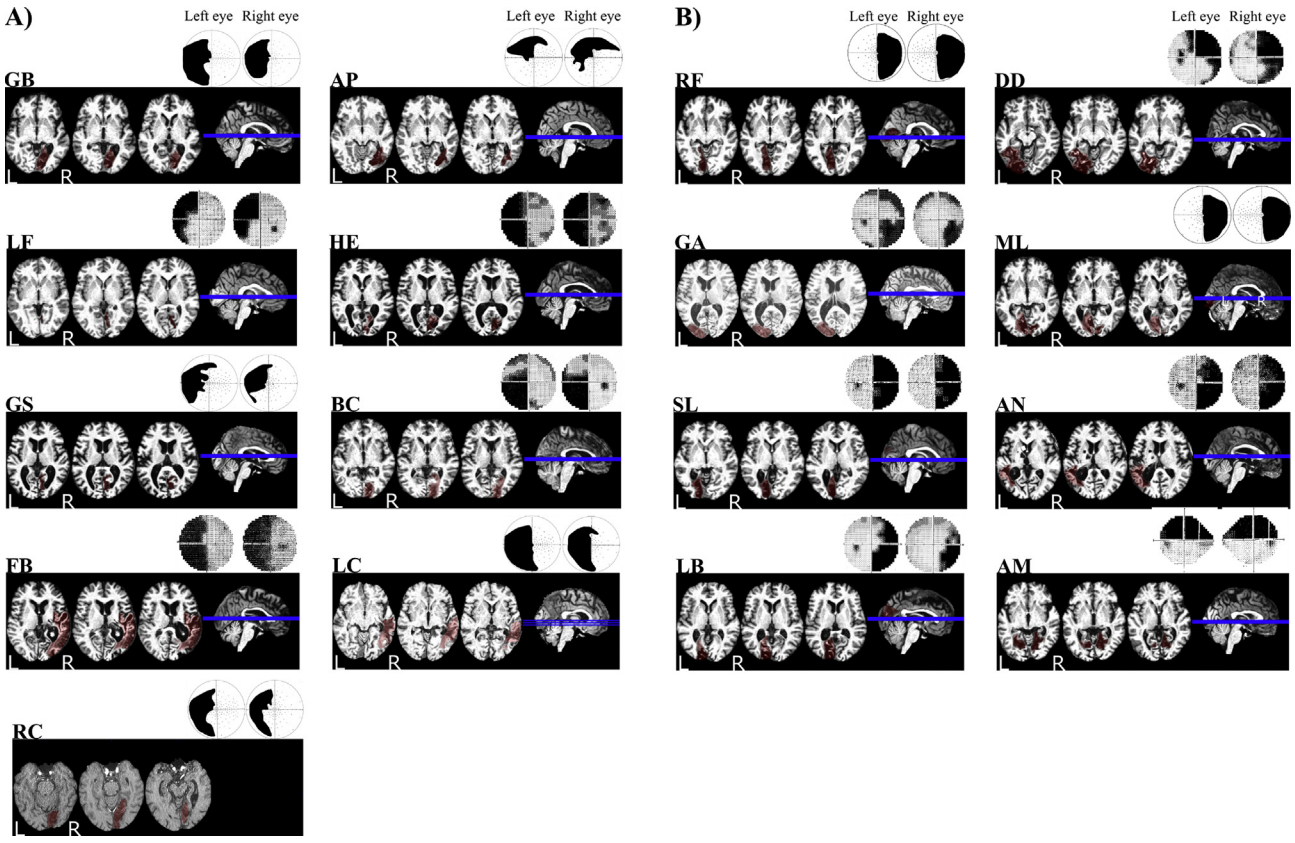


Fig. 1 – Patients' structural MRI and campimetry. A) Patients with right, and (B) with left hemisphere lesion. Site and extension of the lesion are indicated in red on MRI images. Black areas in the campimetry represent blind field. The sagittal brain view of patient RC was unavailable.

Table 2 – Percentage of cortical brain damage in the various groups of patients.

Harvard–Oxford ROI	Group 1	Group 2	Group 3	Group 4	Group 5	Mean
Intracalcarine	61.88	7.74	39.47	64.95	4.78	**35.76**
Supracalcarine	27.14	.24	19.32	76.42	9.21	**26.46**
Lateral Occipital inferior	1.12	21.70	10.87	.89	38.77	**14.67**
Lateral Occipital superior	0	.08	7.94	4.97	38.39	**10.27**
Occipital Fusiform Gyrus	23.63	39.06	28.92	5.75	7.61	**20.99**
Occipital Pole	11.06	7.03	24.26	22.78	5.37	**14.10**
Lingual Gyrus	56.73	16.60	31.55	19.56	1.53	**25.19**
Cuneus	7.15	0	17.06	63.04	6.38	**18.72**
Precuneus	5.29	.34	7.17	23.64	9.33	**9.15**
Temporal Occipital Fusiform	13.86	33.45	7.45	0	12.26	**13.40**
Cingulate Gyrus posterior	6.03	.15	1.98	6.49	4.34	**3.79**
Middle Temporal Gyrus temporo-occipital	0	4.23	0	0	74.68	**15.78**
Mean	**17.82**	**10.88**	**16.335**	**24.04**	**17.72**	**17.36**

Probabilistic Tractography analysis was carried out with FMRIB's Diffusion Toolbox (FDT; Behrens, Berg, Jbabdi, Rushworth, & Woolrich, 2007; Behrens et al., 2003). The regions of interest (ROIs) used as a seed were left/right LGN and left/right SC. The ROIs used as target were left/right V1 and left/right hMT + for LGN and hMT + for SC. Additionally, probabilistic streamlines were also calculated between left/right V1 and left/right hMT+; LGN and V1 were extracted from the probabilistic Juelich Atlas using a 50% threshold.

The hMT + areas were identified individually for each participant by using a functional localizer approach during the MRI session, see Appendix C for further information. For the SC, binary masks were manually drawn and positioned on the anatomical 3D T1 of each patient already registered in MNI 1 mm space. To automatize the process a custom modified AutoPtx script (https://fsl.fmrib.ox.ac.uk/fsl/fslwiki/AutoPtx) was applied (de Groot et al., 2013). This script was customized to calculate and extract the Fractional Anisotropy (FA) and Mean Diffusivity (MD) values of each bundle that represent tissue microstructure in presence of neuronal damage (Jones, Knösche, & Turner, 2013; Werring et al., 2000). The fibre bundles extracted were left/right LGN-V1/hMT+, left/right SC-

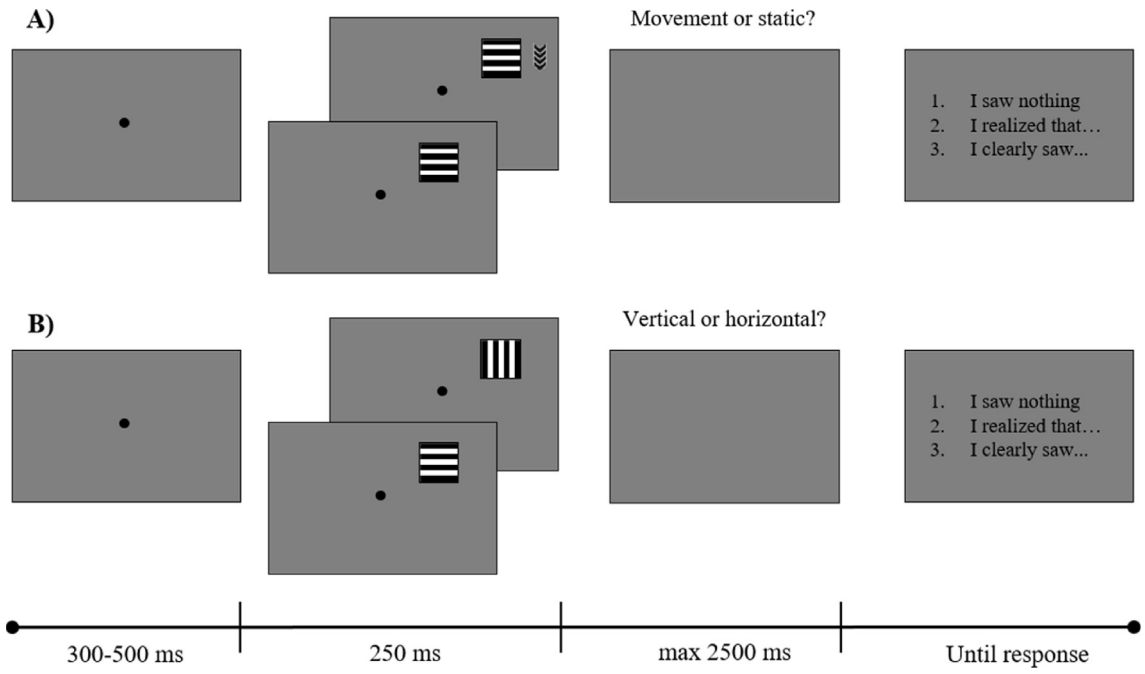


Fig. 2 – Stimuli and timeline of the experimental procedure. A) Movement discrimination and B) Orientation discrimination tasks.

hMT+ and the interconnections between left and right V1 and left and right hMT+.

2.4. *Cortical thickness*

Patients' anatomical T1-weighetd volumetric scan was processed using the default Freesurfer pipeline (Freesurfer v. 6.0.0, http://surfer.nmr.mgh.harvard.edu/) to perform cortical surface extraction, segmentation of subcortical structures (Dale, Fischl, & Sereno, 1999), cortical thickness estimation (Fischl & Dale, 2000), spatial normalization onto the FreeSurfer surface template (Fischl, Sereno, & Dale, 1999) and parcellation of cortical regions based on different atlases (Fischl et al., 2004). Thirty-four cortical brain areas from left and right hemisphere of the whole brain were extracted. For statistical analysis only brain areas involved in visual processing were included: Cuneus, Lingual, Pericalcarine, Lateral Occipital, Isthmus Cingulate, Precuneus, Middle Temporal, Fusiform and Posterior Cingulate Gyrus.

2.5. *Statistical analysis*

2.5.1. *General statistical analysis*

A series of two-way ANOVAs with Task (movement and orientation) and Lesion Side (left and right damaged patients) as factors was carried out on percentage of correct responses, RT and percentage of responses indicating level of perceptual awareness. The analysis was conducted separately for the sighted and blind hemifield. We performed a non-parametric permutation test using 5000 permutations as implemented in EEGLAB function "statcond" (Delorme & Makeig, 2004). Additionally, we carried out a paired *t*-test to assess possible differences in the proportion of perceptual awareness reports between tasks (movement and orientation discrimination) and between conditions, static and moving stimuli in the movement discrimination task.

2.5.2. *Correlation analysis*

We used a Spearman bivariate correlation between a) discrimination accuracy and perceptual awareness reports in the two tasks, separately; b) extension of the blind visual field and discrimination accuracy; and c) extension of the blind visual field and percentage of lesion and Probabilistic Tractography derived values.

2.5.3. *Correlation between lesion extension and behavior: lesion to Symptom Mapping*

The aim of this analysis was to evaluate the relation between damaged brain regions and deficit in performance or absence of perceptual awareness during the discrimination tasks. To do that, the Sparse Canonical Correlation Analysis for Neuroimaging Lesion (SCCAN) was used as implemented in the Lesion to Symptom Mapping package (Pustina, Avants, Faseyitan, Medaglia, & Coslett, 2018). SCCAN consists of a non-statistical multivariate analysis optimization routine that follows machine learning principles in which the method finds a set of voxels that better contribute to explain the behavioral score. With that, a series of voxel weights are obtained indicating that the stronger the weight the more important is a voxel in relation to behavior and perceptual awareness.

SCCAN routine enabled us to have a general view of the association between lesion and performance or visual awareness. Since this analysis is performed at group level it allows to extract a reliable general picture of this association. However, it does not detail specific differences between groups of patients. Therefore, non-parametric ANOVAs were employed to compare the localization and extension of brain

damage in different groups of patients. In summary, the combination of both analyses, which yielded consistent results, gave us a more reliable panoramic of the relation between lesion and behavior-perception in our patients.

To perform this analysis binarized masks of the volume of brain lesion were used to evaluate their correlation with the deficit in performance, i.e. percentage of errors, and absence of perceptual awareness, i.e. PA = 1 (saw nothing) during both movement and orientation discrimination task. Four separated analyses were performed, two for accuracy (movement and orientation) and two for level of perceptual awareness (movement and orientation). Settings included a default sparseness = .045, cluster threshold = 150, smooth sigma = .4 and iterations = 20. In order to enable an overlap of the lesion across patients those with left lesion were flipped left to right.

Patient RC was not included in this analysis because of the low quality of the structural MRI. Patients AN, FB and LC were excluded from this analysis considering that their visual defect was caused by damage to the Optic Radiation. Finally, patient ML was included only in the analysis of movement discrimination (accuracy and perceptual awareness) since he did not perform the orientation discrimination task.

2.5.4. *Group classification*

We divided patients as a function of above chance discrimination performance and presence of perceptual awareness reports for the blind field. For each patient and task, a binomial test was applied to assess whether the proportion of correct responses was reliably different from chance. As to perceptual awareness, only patients with a mean percentage of reports above 10% were considered. All responses below 10% were either 0% or unclear to be classified.

2.5.5. *Group comparisons: extent of cortical and visual pathways lesion*

The aim of this analysis was to test for the existence of differences in the percentage of brain lesion and/or Probabilistic Tractography values between patients performing above or at chance level, and between patients with and without perceptual awareness reports. A series of two-ways ANOVAs was performed for each comparison using Group (above/at chance performance or with/without perceptual awareness) and Anatomical Measures (percentage of brain lesion or Probabilistic Tractography measures). These analyses were performed by means of a non-parametric permutation test using 5000 permutations as implemented in EEGLAB function "statcond" (Delorme & Makeig, 2004). This test was carried out since, given the relatively small sample size, we assumed a non-normal distribution of the data. In this case, non-parametric permutation is appropriate for statistical analysis. It consists of determining the distribution of the statistic tests under the null hypothesis calculating all possible values of the statistics under all rearrangements of the observed data labels (see LaFleur & Greevy, 2009 for a more detailed description of the method and Ludbrook & Dudley, 1998 for its relevance in biomedical research).

Brain areas considered for the statistical analysis involved 10 occipital ROIs plus the Precuneus and the Posterior Cingulate Gyrus (see Discussion). The decision to restrict the analysis to the above areas was made on the basis of the evidence that the vast majority of the damaged cortical areas was in the occipital lobe as shown in Fig. 1 and in Table 2, see also the complete version of this table in Appendix B. Patients with Optic Radiation lesion had the largest proportion of extra-occipital damage and were not included in the cortical but only in the Probabilistic Tractography analyses considering that their visual defect is caused by deafferentation of visual cortical areas (in addition or not to direct anatomical damage). Patient RC was not included because a complete structural MRI for the assessment of the cortical areas was not available.

Same procedure was followed for the analysis of FA and MD. Values obtained from the ipsilesional hemisphere were used to carry out two-ways ANOVAs for each analysis and Probabilistic Tractography values (for patient AM with bilateral damage and visual defect we used the mean values of the two hemispheres). Patients LC, GA and RC were not available for Probabilistic Tractography assessment.

2.5.6. *Group comparisons: cortical thickness*

A third analysis was performed to evaluate a possible involvement of the intact hemisphere in neuroplastic adjustments following damage to its lesioned counterpart. We carried out a statistical analysis of the cortical thickness of nine areas of the intact hemisphere involved in visual processing. For normative values of cortical thickness of visual areas see Alvarez, Parker, & Bridge, 2019. Two-ways ANOVAs were performed to compare the cortical thickness of patients with and without above chance performance for both tasks together as well as for each task separately. Moreover, we performed a comparison between patients with and without presence of perceptual awareness. As described above, a non-parametric permutation test was used. Patients ML and AM were excluded from these analyses since they had bilateral lesions (even though ML shows only a right visual defect). In patients GA, LC, RC and FB was not possible to calculate the values of cortical thickness for the extent of the lesion or for the quality of the T1 image.

No part of the study procedures or analysis were preregistered prior to the research being conducted.

3. Results

3.1. *General analyses*

Separate two-ways ANOVAs (Task by Lesion side) were carried out for the blind and sighted hemifield on discrimination scores and percentage of perceptual awareness reports in the two tasks with all patients. In the blind hemifield the only significant main effect was Lesion Side (p = .01) with perceptual awareness reports higher in patients with left

(43.6; SD = 36.8) than right brain damage (14.2; SD = 23.4). No significant effect of Task or interaction Task by Side was present.

In the sighted hemifield there were no significant effects.

Paired *t*-test, performed to evaluate differences in the proportion of perceptual awareness reports related to Task (movement and orientation discrimination) or Condition, (static and moving stimuli in the movement discrimination task), yielded no significant results.

3.2. *Correlation analyses*

a) No significant correlation was found between discrimination accuracy in both tasks and perceptual awareness reports.
b) No significant correlation was found between extension of the blind visual field and discrimination accuracy or perceptual awareness reports.
c) Blind visual field extension positively correlated with proportion of damage of the Intracalcarine area ($r = .57$, $p = .02$) and Lingual gyrus ($r = .55$, $p = .02$).

3.3. *Correlation between lesion extension and behavior: lesion to Symptom Mapping*

This analysis was performed by using the SCCAN routine to evaluate the sets of voxels that best contributed to explain the behavioral deficits. We found suprathreshold voxels correlating with accuracy in the movement discrimination task and with perceptual awareness in both movement and orientation discrimination. Patients FB, AN, LC with visual pathway damage, and RC with no complete structural MRI for the assessment of the cortical areas were not included in this analysis. For the analysis of the accuracy and perceptual awareness of the orientation discrimination, ML was not included, since he did not perform this task.

Results are shown in Table 3 and displayed in Fig. 3. For the deficit in *accuracy in movement discrimination* a total of 940 overlapped damaged voxels, divided in two clusters, were at suprathreshold to better explain the performance; maximum peaks of the clusters included: Precuneus, Lingual gyrus, Intracalcarine, Supracalcarine, Posterior Cingulate gyrus, Cuneus, Occipital Pole and Lateral occipital cortex superior division. For *orientation discrimination*, no relation between lesion and deficit in accuracy was found. For the percentage of absence of perceptual awareness in either movement or orientation discrimination task, suprathreshold voxels of the damaged brain areas are 262 and 714, respectively. For *perceptual awareness in movement discrimination* voxels were grouped in one cluster which included the Lingual gyrus, Precuneus, Intracalcarine, Supracalcarine, and Posterior Cingulate gyrus. For *perceptual awareness in orientation discrimination* two clusters were found which included Intracalcarine, Lingual gyrus, Supracalcarine, Precuneus, and Posterior Cingulate gyrus.

At first glance one can notice that for discrimination accuracy (movement task) as well as for perceptual awareness (both tasks) most areas reliably involved were in visual cortical areas with the exception of the Precuneus and the Posterior Cingulate Gyrus.

3.4. *Individual discrimination performance and perceptual awareness reports*

Table 4 shows the percentage of correct discrimination responses and of perceptual awareness reports in the two discrimination tasks for stimulus presentation to the blind hemifield subdivided in groups as described below. In Fig. 4 the overlapped brain lesions of the patients subdivided by group are shown.

3.4.1. *Group classification*

We divided the patients in four groups according to scores in accuracy of discrimination and perceptual awareness reports. In addition, a fifth group was separated from Group 4 on the basis of the interruption of the Optic Radiation, that is, lack of input to area 17 and other visual areas. This makes this group anatomically and functionally different from the others.

Group 1 includes patients with above chance discrimination performance without perceptual awareness. Two patients (GB and RF) scored above chance in the orientation discrimination but not in the movement discrimination task.

Group 2 includes patients with above chance discrimination performance and presence of perceptual awareness. Three patients (AP, DD and LF) scored above chance in the movement discrimination but not in the orientation discrimination.

Group 3 includes five patients (AM, SL, GA, ML and HE) with discrimination performance at chance level and presence of perceptual awareness in either task.

Group 4 includes four patients (RC, BC, GS, LB) with discrimination performance at chance level without perceptual awareness in either task.

Group 5 includes three patients (FB, AN, and LC) with discrimination at chance level without perceptual awareness in either task. They have an interruption of the Optic Radiation, see and example in Appendix D, Fig. 1, and therefore were considered separately from Group 4.

3.4.2. *Difference between patients with and without above-chance performance: cortical lesions*

The brain areas, extracted from the Harvard–Oxford Atlas, used for the analyses in subsections 3.4, 3.5 and 3.6, were: Intracalcarine, Supracalcarine, Lateral Occipital inferior, Lateral Occipital superior, Occipital Fusiform Gyrus, Occipital Pole, Lingual Gyrus, Cuneus, Temporal Occipital Fusiform, Middle Temporal Gyrus temporo-occipital, Precuneus and the Posterior Cingulate gyrus.

Statistical analyses in subsection 3.4 and 3.5 were carried out by means of a series of non-parametric two-way ANOVAs with Group (2) and Brain Areas (12) as factors.

3.4.2.1. ORIENTATION DISCRIMINATION. Above-chance performance: two patients (GB, RF); Chance performance: 10 patients (AP, LF, DD, AM, GA, HE, SL LB, BC, GS). The only significant effect was that of Brain area ($p < .01$). No significant

Table 3 – Results of the SCCAN procedure to evaluate the set of voxels that better contribute to explain absence of discrimination accuracy and perceptual awareness. MNI coordinates and Harvard–Oxford Areas of the maximum weight score are indicated.

Cluster	Voxels Number	Xmax MNI	Ymax MNI	Zmax MNI	Harvard–Oxford Area
Accuracy Movement Discrimination Task					
1	270	87	70	81	Precuneus, Lingual gyrus, Intracalcarine, Supracalcarine, Posterior Cingulate gyrus.
2	670	78	44	90	Cuneus, Supracalcarine, Intracalcarine, Occipital Pole, Lateral occipital cortex superior division.
Perceptual Awareness Movement Discrimination Task					
1	262	72	69	76	Lingual gyrus, Precuneus, Intracalcarine, Supracalcarine, Posterior Cingulate gyrus.
Perceptual Awareness Orientation Discrimination Task					
1	473	77	50	80	Intracalcarine, Lingual gyrus, Supracalcarine.
2	241	71	70	76	Lingual gyrus, Precuneus, Intracalcarine, Supracalcarine, Posterior Cingulate gyrus.

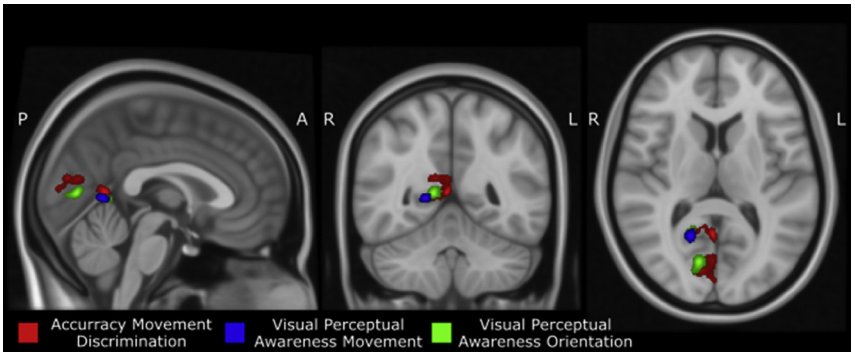


Fig. 3 – Results of the SCCAN procedure showing the location of suprathreshold voxels. In red voxels related to the deficit in accuracy for the movement discrimination task. Blue and green represent voxels related to absence of visual perceptual awareness. Maps are radiologically oriented.

main effect of Group ($p = .74$) and no Interaction of Group by Brain area ($p = .38$) were found. Therefore, no further analysis was performed because the overall difference in the lesions of the various areas examined was not relevant given that they did not interact with Group.

3.4.2.2. Movement discrimination. Above-chance performance: Three patients (AP, LF, DD). At chance performance: 10 patients (GB, RF, AM, GA, HE, ML, SL LB, BC, GS). Significant main effects of Group ($p = .03$), Brain Area ($p = .03$) and of the interaction Group by Brain area ($p = .003$) were found. Post-hoc analysis showed that the above-chance performance group showed a smaller percentage of lesion in Intracalcarine ($p = .01$), Supracalcarine ($p = .01$), Cuneus ($p = .02$), Precuneus ($p = .002$) and Posterior Cingulate gyrus ($p = .009$) with respect to the group with chance performance.

3.5. *Difference between groups with and without perceptual awareness: cortical lesions*

This analysis was done independently of the discrimination task since there were no statistical differences between the two tasks. There were eight patients with perceptual awareness (AP, LF, DD, AM, GA, HE, ML, SL) reports and five without (GB, RF, LB, BC, GS). There was a significant main effect of Group ($p = .01$), Brain Area ($p < .001$) and a significant interaction Group by Brain area ($p < .001$). Post-hoc analysis showed that the group with presence of perceptual awareness reports showed a smaller percentage of lesion in Intracalcarine ($p = .03$), Supracalcarine ($p = .008$), Precuneus ($p = .04$) and Posterior Cingulate Gyrus ($p = .002$) with respect to the group with absence of perceptual awareness.

3.6. *Difference between groups: cortical lesions*

A good way of disentangling the specific contribution of the cortical areas to discrimination or to perceptual reports is to compare all the groups barring Group 5. We analyzed the difference among the four groups of patients in the percentage of cortical lesion in the brain areas considered.

We carried out a non-parametric two-ways ANOVA with Group (4) and Brain Area (12) as factors, see Table 5 (patient RC was not included in this analysis). The main effect of Group ($p = .009$) and Brain area ($p < .001$) as well as the interaction ($p < .001$) were significant. A one-way ANOVA showed that six

Table 4 – Percentage of correct response and of trials with a response = 2 in the perceptual awareness scale for the two discrimination tasks.

Patient	Movement Discrimination					Orientation Discrimination				
	Total trials	% Accuracy Total	% Acc. without perceptual awareness	% Acc. with perceptual awareness	% Perceptual awareness report	Total trials	% Accuracy Total	% Acc. without perceptual awareness	% Acc. with perceptual awareness	% Perceptual awareness report
Group 1. Above chance without perceptual awareness										
GB	160	56.9	56.6	–	0.6	80	61.3*	61.3*	–	0
RF	232	44.8	44.8	44.8	12.5	240	55.8	57.5*	25	5
Group 2. Above chance with perceptual awareness										
AP	157	54.8	38.6	61.1*	70.6	79	57	53.8	63	33.7
LF	240	58.3*	56.1	62*	38.3	240	50	49.6	–	2.5
DD	160	71.3*	–	71.1*	99.4	78	55.1	–	55.1	97.5
Group 3. At chance with perceptual awareness										
AM_R	160	50.6	51.9	45.2	19.4	160	48.8	49	48.4	38.8
AM_L	160	50.6	53.1	49	60	160	50	50.7	49.4	53.1
GA	155	47.7	44.1	56.8	27.5	160	51.9	59.5	49.6	76.9
HE	160	50	48.2	61.9	13.1	160	51.3	50.7	55.6	11.2
ML	159	56.6	56.3	58.1	19.4	0	NA	NA	NA	NA
SL	160	43.1	40	44.2	75	160	50	50	50	62.5
Group 4. At chance without perceptual awareness										
LB	157	54.8	54.4	–	6.3	160	56.3	56.1	–	3.1
BC	157	42	42	–	0	158	48.1	48.1	–	1.3
GS	160	49.4	49.4	–	0	160	50	50	–	0
RC	153	50.3	50.3	–	0	154	51.3	51	–	0.6
Group 5. At chance without perceptual awareness plus optic radiation interruption										
AN	192	52.1	52.1	–	1	0	NA	NA	NA	NA
FB	160	48.1	48.1	–	0	158	47.5	47.5	–	0
LC	151	50.3	50.3	–	0	158	48.7	48.7	–	0

$p < .05$ binomial test (50%). % Acc. = Percentage of correct responses; – = percentage was not calculated because awareness was 0; 0% = less than 10 percent reports; NA = no task was performed. Significant effects are marked with an asterisk.

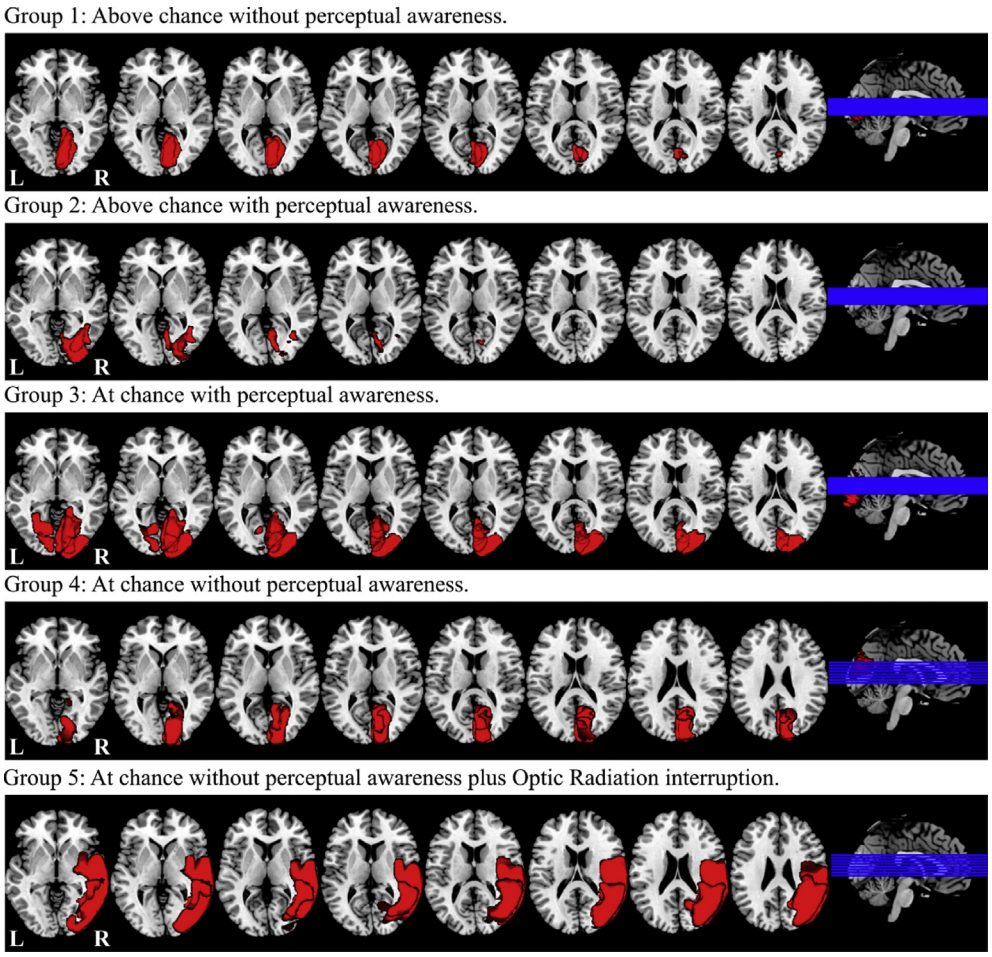


Fig. 4 – 2D representation of the overlapped lesions of the five groups. Clearly, Group 2 has the smallest overall extent of lesion in keeping with showing both above-chance performance in movement discrimination as well as high rate of perceptual awareness reports. Patients with left lesions were flipped left to right. In the figure all lesions are drawn in the right hemisphere.

areas differed significantly across groups. The comparison between Group 1 and 2 yielded a significant difference for four areas: Intracalcarine, Supracalcarine, Cuneus, Precuneus and Posterior Cingulate gyrus with extent of lesion larger in Group 1 than Group 2. The difference between the two groups is clearly related to perceptual awareness and above chance discrimination for the movement task that are both present in Group 2 but not in Group 1. However, there is good reason to believe that the difference in perceptual awareness depends on the four occipital areas while that related to above chance performance depends on the Precuneus and the Posterior Cingulate Gyrus. As shown in Table 5, this hypothesis is confirmed by the comparison between Group 2 and Group 3 which differ in the movement discrimination task performance (above chance in the former) while sharing the presence of perceptual reports. Accordingly, the Precuneus and the Posterior Cingulate Gyrus have a significantly lager lesion in the latter group. Finally, Group 3 and 4 differ for the absence of perceptual reports in the latter group which shows a larger lesion in the Supracalcarine cortex and Posterior Cingulate Gyrus (see Table 5). Given that neither group performed above-chance the difference is likely related to the latter showing no perceptual reports. Thus, it is reasonable to assume that these comparisons indicate that the Precuneus is selectively involved in above chance discrimination in the movement task, the Supracalcarine area is important for perceptual awareness reports, and the Posterior Cingulate gyrus is critically involved in both movement discrimination and visual perceptual awareness, see Discussion.

3.7. *Difference between groups with and without above-chance performance: probabilistic tractography analysis*

A similar analysis as for the cortical areas was carried out for the Probabilistic Tractography data separately for the two discrimination tasks. Fibre bundles included in the analyses

Table 5 – Comparison of cortical areas damage among the four patient groups.

Comparison	Non-parametric ANOVA	Post-hoc pairwise *p*-values	Group mean 1 (SD)	Group mean 2 (SD)
Harvard Oxford Brain Areas				
G1(1)-G2(2)	*Two-way ANOVA Group (4) by Brain area (12):* Group $p = .009$, Brain area $p < .001$, Interaction $p < .001$ *One-way ANOVA Group = 4:* Intracalcarine $p = .02$ Supracalcarine $p = .004$ Cuneus $p = .02$ Precuneus $p = .02$ Cingulate Gyrus posterior < .001	Intracalcarine $p < .001$	61.8 (4.7)	7.7 (7)
		Supracalcarine $p < .001$	27.1 (1.2)	.2 (.4)
		Cuneus $p < .001$	7.1 (1.9)	01 (03)
		Precuneus $p < .001$	5.2 (2.1)	.3 (.5)
		Posterior Cingulate gyrus < .001	6.2 (2.41)	.1 (.2)
G2(1)-G3(2)		Precuneus $p = .03$	.3 (.5)	7.1 (8.4)
		Posterior Cingulate Gyrus = .03	.1 (.2)	1.9 (1.0)
G3(1)-G4(2)		Supracalcarine $p = .03$	19.3 (16.3)	64.9 (21)
		Posterior Cingulate Gyrus = .03	1.9 (1.0)	6.4 (.9)

Note: G1 = above chance performance in orientation discrimination without awareness reports, n = 2; G2 = above chance performance in movement discrimination and awareness reports n = 3; G3 = chance performance and awareness reports, n = 5; G4 = chance performance without awareness reports, n = 3. Group 5 was not included in this analysis. Upper part of the second column from left: Results of a two-way ANOVA. Lower part: One-way ANOVA to individuate the brain areas with significant difference among groups. Third column: *p* values of the post-hoc pairwise comparisons. Only pairwise comparisons where significant results ($p < .05$) were found are reported.

described in subsection 3.7 and 3.8, were left/right LGN-V1/hMT+, left/right SC-hMT+ and the interconnections between left and right V1 and left and right hMT+. Statistical analyses were performed by means of a series of non-parametric two-way ANOVAs with Group (2) and Fiber (5) as factors.

3.7.1. *Orientation discrimination*

Above-chance: two patients (GB, RF); Chance performance: 10 patients (AP, LF, DD, AM, HE, SL, LB, BC, GS, FB). A non-parametric ANOVA Group (2) by Fiber (5) was performed with no significant effects ($p > .05$) for either FA or MD.

3.7.2. *Movement discrimination*

Above chance performance: Three patients (AP, LF, DD); Chance performance: 11 patients (GB, RF, AM, HE, ML, SL, LB, BC, GS, AN, FB). No significant results were found ($p > .05$) for FA and MD.

3.8. *Difference between groups with and without perceptual awareness: probabilistic tractography analysis*

Seven patients with (AP, LF, DD, AM, HE, ML, SL) and seven without (GB, RF, LB, BD, GS, AN, FB) perceptual awareness. There was a main effect of Group for both FA ($p < .05$) and MD ($p < .05$). For FA the group with presence of perceptual awareness reports (Mean = .33, SD = .03) showed higher mean values for all fibers than the group without (Mean = .30, SD = .06). MD had smaller value for all fibers in the group with perceptual awareness reports (Mean = 1.06, SD = .18) than that without (Mean = 1.12, SD = .28). No other significant differences were found.

As for the cortical lesion analysis, with Probabilistic Tractography we compared the patients' groups for the five visual pathways. FA: The results were clear; as expected, all groups showed significantly higher values in comparison with Group 5 (patients with Optic Radiation lesion, see Table 6 in the text and Fig. 1 in Appendix C for a representative example). For MD there was no statistically significant effect. The interactions were not significant.

3.9. *Cortical thickness of the intact hemisphere*

Brain areas included for the analysis of cortical thickness were: Cuneus, Lingual, Pericalcarine, Lateral Occipital, Isthmus Cingulate, Precuneus, Middle Temporal, Fusiform and Posterior Cingulate Gyrus. Statistical analyses were performed by means of a series of non-parametric two-way ANOVAs with Group (2) and Brain Area (9) as factors.

3.9.1. *Difference between groups with and without above-chance performance*

Both tasks together: Above-chance performance: five patients (GB, RF, AP, LF, DD); Chance performance: six patients (HE, SL, LB, BC, GS, AN). A non-parametric ANOVA with Group (2) and Brain areas (9) was carried out. Significant main effects of Group ($p < .001$) and Brain Area ($p < .001$) were found. Patients of the above chance group showed greater cortical thickness (2.16 mm) than those with chance performance (2.09 mm). No significant interaction Group by Brain area ($p = .14$) was found. Importantly, these values are in keeping with normative results recently obtained by Alvarez et al. (2019) in the visual areas of healthy participants ranging between 1.93 and 2.77 mm. Note that the mean of our groups was within the normative values. The two groups did not statistically differ for age (U Mann–Whitney = 8.0, $p = .2$; Median Above Chance Group = 52.2; IQR = 12.41; Median Chance Group = 61.7; IQR = 17.4 [IQR = inter quartile range]). Finally, two separate ANOVAs showed a significantly greater cortical thickness for patients with above-chance performance in either task (Orientation: $p = .03$; Movement: $p = .04$).

3.9.2. *Difference between groups with and without perceptual awareness*

The comparison was done independently of the discrimination task. Five patients with (AP, LF, DD, HE, SL) and six without (GB, RF, LB, BC, GS, AN) perceptual awareness reports. A non-parametric ANOVA with Group (2) and Brain Area (9) as

Table 6 – Comparison of visual pathways' integrity among the five patients' groups.

Comparison	Non-parametric ANOVA	Post-hoc pairwise *p*-values	Group mean 1 (SD)	Group mean 2 (SD)
DTI: FA				
G1 (1)-G5 (2)	*Two-way ANOVA Group (5) by Fibers-FA (5):*	Mean across Fibers (FA) $p < 0.001$	**.33 (.046)**	.23 (.049)
G2 (1)-G5 (2)	**Group $p < .001$, Fiber** $p = .03$, Interaction $p = .62$	Mean across Fibers (FA) $p < .001$	**.32 (09)**	.23 (.049)
G3 (1)-G5 (2)		Mean across Fibers (FA) $p = .03$	**.33 (.028)**	.23 (.049)
G4 (1)-G5 (2)		Mean across Fibers (FA) $p < .001$	**.32 (.031)**	.23 (.049)
DTI: MD				
	Two-way ANOVA Group (5) by Fibers-MD (5):	No significant Post-hoc pairwise results were found for the mean across Fibers (MD).	Mean G1 = 1.05 (SD = .01)	
	Group $p < .001$, Fiber $p = .003$, Interaction $p = .38$		Mean G2 = 1.03 (SD = .07)	
			Mean G3 = 1.09 (SD = .03)	
			Mean G4 = 1.01 (SD = .14)	
			Mean G5 = 1.36 (SD = .25)	

Note: Only pairwise comparisons where significant results ($p < .05$) were found are reported. G1 = above chance performance without awareness reports, n = 2; G2 = above chance performance and awareness reports n = 3; G3 = chance performance and awareness reports, n = 4; G4 = chance performance without awareness reports, n = 3; G5 = chance performance without awareness reports and with Optic Radiation damage, n = 2.

main factors was performed. There was a significant main effect of Brain Area ($p < .001$). No significant effect of Group ($p = .9$) or interaction Group by Brain area ($p = .9$) were found. Therefore, no further post hoc analysis was performed.

4. Discussion

To our knowledge this is one of the few studies on a relatively large cohort of hemianopic patients thoroughly tested for both unconscious above-chance performance and perceptual awareness as well as for damage to several cortical areas and visual pathways. Other studies that meet some of these requirements have been recently carried out (Ajina, Pestilli, et al., 2015; Ajina & Bridge, 2017; Celeghin et al., 2015; Garric et al., 2019). In relation to a widely known subdivision of blindsight (Weiskrantz et al., 1995), our Group 1 that represents 11.76% of the patients tested corresponds to Blindsight type 1 characterized by above-chance discrimination performance and no reported visual (or non-visual) awareness. Group 2 (17.64%) corresponds to Blindsight type 2 characterized by above-chance performance and some form of perceptual awareness (unrelated to the discrimination in our patients). Incidentally, here we are not discussing the important question of whether patients with Blindsight type 2 experience visual or non-visual awareness. By the same token, we are not discussing whether these patients with above chance performance and rudimental form of visual awareness are to be considered with blindsight or degraded vision (see Mazzi, Savazzi et al., 2019; Overgaard et al., 2008). What we can say with our data is that all our patients of this group reported some form of visual perceptual awareness that even though did not bear any structural relationship with the discriminanda, was nonetheless accompanied by an above chance discrimination. Interestingly, Group 3 (29.41%), i.e. patients without above chance performance but with presence of perceptual awareness broadly corresponds to a group of patients recently described by Garric et al. (2019). They studied four hemianopic patients who were not able to discriminate the stimuli (x *vs.* o) but could reliably "sense" stimulus presentation, see below for discussion. Finally, Group 4 (41.17%) with no above-chance performance and no perceptual awareness reports represents the majority of our sample of hemianopic patients. Importantly, from this group we selected out three patients with an interruption of the Optic Radiation (Group 5). We believe that this distinction is crucial because the neural impairment of these patients is fundamentally different from that of those with a cortical lesion without a substantial visual input deafferentation. To our knowledge, in the blindsight literature very rarely patients with an Optic Radiation interruption have been separately analyzed.

By comparing the brain areas more damaged in each of the five groups of patients we have been able to get reasonable insights on the possible anatomical basis of the two characterizing aspects of blindsight, namely, unconscious above chance behavioral performance and presence of perceptual awareness reports of stimuli presented to the blind field.

Given for granted that all our patients are hemianopics, expectedly, in all groups except group 5, the most frequently

and largely affected areas are in the occipital lobe. Of course, patients with lesion of Optic Radiation are visually deafferented but additionally may or may not have anatomically intact visual cortical areas. That the other four groups have important occipital lesions is confirmed by the significant positive correlation of lesion volume of the Intracalcarine (area 17) and Lingual gyrus cortex (area 18 and 19) with the extension of the blind hemifield.

The crucial question that we tackled here was to find out which lesioned areas make a difference between patients with or without unconscious above-chance discrimination performance and with or without perceptual awareness reports. In reference to these comparisons it is important to stress that there was no reliable overall correlation between discrimination accuracy and percentage of perceptual reports. Thus, the two aspects can be separately considered.

One interesting result is that different patients showed above chance discrimination in one (orientation) or in the other (movement) task. For the former there was no correlation between extent or location of lesion and discrimination performance. Moreover, this task difference was accompanied by a differential percentage of perceptual reports which was significantly present only in the group scoring above chance in the movement task. It would be tempting to conclude that perceptual awareness and stimulus movement discrimination are related and this might explain the concomitant presence of both in Group 2. However, as mentioned above, a general correlation analysis between percentage of perceptual awareness reports and discrimination accuracy showed no significant effect for either task. Thus, perceptual awareness did not affect discrimination tasks and vice versa. This is also confirmed by Group 3 in which the presence of perceptual awareness did not enable the patients to reach the above chance threshold in either discrimination task. Nonetheless, it is possible that visual movement discrimination and perceptual awareness are psychophysically related because of the saliency of moving stimuli, as pointed out recently by Phillips (2019) and these two processes may also have partially overlapping anatomical bases. In this context an important issue to discuss is the seemingly inconsistency of our results in respect to those of Zeki and Ffytche (1998) on patient GY where they found a correlation between stimulus awareness and performance. However, it should be mentioned, that Zeki and Ffytche (1998) used a different experimental approach from ours in that they changed the psychophysical properties of the stimuli in order to affect the level of perceptual awareness. In contrast, in our study the two stimulus features (orientation and movement) were constant all along the task. The authors did find a significant correlation between accuracy and perceptual awareness but the correlation was not absolute due to the fluctuating level of GY's visual awareness and discrimination performance. For instance, the level of awareness under particular stimulus conditions varied between sessions without a change in levels of discrimination performance that could vary for the same task without fluctuation in awareness. Interestingly, they proposed a model for the Riddoch syndrome where the tails represent discrimination without awareness and awareness without discrimination, respectively. In sum, in principle our results are not inconsistent with those of Zeki and Ffytche (1998) but the paradigm and the participants population are basically different because our patients' perceptual reports were not related to the discriminanda and no psychophysical testing was done.

To cast light on this and other questions raised by our results, important clues have been provided by direct comparison between groups, as shown in Table 5 and briefly reported in the Results. The logic is straightforward: Group 1 and Group 2 differ from each other in two aspects, namely, above chance performance in different tasks and presence of perceptual awareness reports. Group 1 has a conspicuously larger damage than Group 2 in Intracalcarine, Supracalcarine, Cuneus, Precuneus and Posterior Cingulate Gyrus and shows chance performance in movement discrimination as well as absence of perceptual awareness reports. Which of these areas is responsible for movement discrimination and which perceptual awareness can be clarified by the comparison between Group 2 and Group 3. These two groups differ in above chance movement discrimination (present in Group 2 only) while both show perceptual awareness reports. Group 3 has a larger damage in the Precuneus and Posterior Cingulate Gyrus than Group 2. This suggests that these areas are important for above chance movement discrimination and can explain the difference between Group 1 and Group 2 in which the larger Precuneus and Posterior Cingulate Gyrus lesion in the former group might be related to lack of movement discrimination and the larger lesion in the occipital areas might be related to lack of perceptual awareness. Importantly, this possibility is corroborated by the comparison between Group 3 and Group 4 that differ in perceptual awareness but both show no above chance discrimination performance. Group 4, with no perceptual awareness has larger damage in the Supracalcarine cortex (area 17 and 18) as well as in the Posterior Cingulate Gyrus. On the basis of these comparisons a plausible conclusion is that areas 17, 18 and 19 are important for perceptual awareness and area 7 (Precuneus) and areas 23 and 31 (Posterior Cingulate Gyrus) for unconscious above chance movement discrimination. This does not exclude that the Precuneus (area 7) might be involved in perceptual awareness as well. Unfortunately, nothing can be said about orientation discrimination given the lack of anatomical damage correlates.

Interestingly, the difference between Group 1 and 2 in their ability to discriminate orientation or movement is in broad keeping with the classification by Danckert and Rossetti (2005) distinguishing between agnosopsia (Group 1) and attention-blindsight (Group 2). However, our two groups differed also for the presence of perceptual awareness and in this respect our Group 1 is similar to Blindsight type 1 (above chance but no awareness) and Group 2 to Blindsight type 2 (above chance plus some sort of awareness). A clue to the different neural substrate of the above groups comes from our anatomical findings described below.

Thus, above-chance unconscious performance for movement is likely to have Precuneus area 7 and Posterior Cingulate area 23 and 31 as important anatomical substrate. Interestingly, a PET study by Barbur, Watson, Frackowiak, and Zeki (1993) in blindsight patient GY with moving stimuli presented to the blind hemifield found activation in area MT and 7 as well as in other areas outside area 17. The intriguing aspect of this result is that, at variance with our patients, GY

was perceptually aware of the stimuli despite his massive area 17 destruction. Actually, the activation of area 7 in Barbur et al.'s study fits with our results in that its lesion is likely to be responsible for the absence of perceptual awareness as well as for the lack of above chance discrimination of visual movement direction. That area 7 is involved in visual movement processing either consciously or unconsciously is not surprising given that its neurons receive numerous inputs from cortical visual areas as well as from Pulvinar and SC (see Whitlock, 2017 for review) and they might be sufficient to provide information for unconscious above-chance discrimination. Moreover, further important confirmation of the involvement of the Precuneus in visual movement perception can be found in the fMRI results of Hervais-Adelman et al. (2015) in a patient with bilateral occipital cortex lesion tested with looming contrasted with other forms of visual movement. Interestingly, the Precuneus (as well as the inferior parietal lobule) is included in one the four hierarchical clusters described by Celeghin et al. (2019), in their metaanalysis of the functional neuroanatomy of blindsight. Moreover, mention of a Precuneus activation can be found among the activated sites in a study of the effect of varying motion coherence (Ajina, Kennard, Rees, & Bridge, 2015) in patients with V1 lesion. A further tentative explanation of Precuneus' role in unconscious above-chance discrimination might be put forward on the basis of a recent study by Andersson, Ragni, and Lingnau (2019) in which they found that this area is involved in visual imagery together with early visual areas. An intriguing possibility is that stimuli presented in the blind hemifield (which have been already seen by the patient in trials with presentations to the intact field) might be imaged on the basis of subliminal cues and this might bring to an above chance performance. Some hints about this possibility come from evidence that the Precuneus hosts an anterior region dealing with mental imagery strategies, and a posterior region, involved in episodic memory retrieval (see Cavanna & Trimble, 2006). These regions might subserve the kind of blindsight found in our Group 2 patients representing a phenomenological experience that does not have the qualia necessary for conscious vision but might be sufficient for an implicit above-chance performance.

Strictly anatomically and functionally close to the Precuneus is the other area, namely the Posterior Cingulate Gyrus, that we found to be importantly involved in unconscious above-chance discrimination performance of visual movement, as well as in the presence of hints of perceptual awareness. Both areas are considered to be crucial hubs of the Default Mode Network (DMN) and are structurally and functionally connected to the mesial prefrontal and inferior parietal cortex, see Khalsa, Mayhew, Chechlacz, Bagary, and Bagshaw (2014); Wang, Chang, Chuang, and Liu (2019) and this raises intriguing questions about the role of this network in cognition and in awareness (see Leech & Sharp, 2014). However, a more specific reason for relating the Posterior Cingulate Gyrus to our behavioral and perceptual results is that it contains a visual processing area selective for optic flow that lies in the fundus of the cingulate sulcus (Field, Inman, & Li, 2015) and that is activated by coherent visual motion (Antal, Baudewig, Paulus, & Dechent, 2008). Thus, this area that is practically spared in Group 2 patients might explain the presence of an above-chance discrimination of moving versus stationary gratings as well as contribute to reports of perceptual awareness given that a moving grating is probably more salient than a static one (see Phillips, 2019).

However, for the most part perceptual awareness reports would rely on striate and extrastriate cortex as shown by the comparison between Group 1 and Group 2 and between Group 4 and Group 3. This last group would be able to receive a visual signal that might be enough for reaching perceptual awareness without enabling above chance discrimination performance. The presence of the latter kind of hemianopics has been recently outlined by Garric et al. (2019) in patients who did not show above-chance discrimination performance but reliably reported the occurrence of the stimulus of which they were not perceptually aware (a phenomenon termed "*blindsense*"). Their anatomical analysis in two patients, one with and the other without *blindsense* showed that the former had lesion of only area 17 and 18 while the latter had a larger lesion involving areas 17, 18, 19, 29 and 30. The nature of the phenomenological experience of *blindsense* patients is not clear, that is, whether it is visual or not. As mentioned by Garric et al. (2019) it is not an uncommon occurrence that hemianopic patients report the "feeling" of something occurring in the blind field without any hint about the features of the stimuli presented. However, the perceptual nature of this feeling is difficult to ascertain. They correctly guess that a stimulus has been presented but that does not necessarily imply that had a truly visual experience.

Another important finding in our study is the difference between left and right damaged patients in the frequency of perceptual awareness reports that was higher in the former. This result is in line with the literature reporting the dominance of the right hemisphere in many processes including conscious or unconscious visual spatial attention (see a recent study from our lab Sanchez-Lopez, Savazzi, Pedersini, Cardobi, & Marzi, 2020), and self-awareness disorders such as hemineglect and anosognosia (see for example, Joseph, 1988; Thomas & Barrett, 2019; van den Berg & Ruis, 2017; Vogt & Devinsky, 2000). Moreover, an interesting consideration stemming from our results is that this dominance concerns not only full but also partial perceptual awareness.

The probabilistic tractography analysis of our study which included patients with and without Optic Radiation interruption (see clinical details in Table 1) did not yield significant effects for the two discrimination tasks but there was a reliable advantage for the groups with perceptual awareness for values of FA and MD thus showing that the anatomo-functional status of all the visual pathways examined is important for the presence of reports of perceptual awareness.

As to cortical thickness, which is significantly greater in the intact hemisphere of patients who performed above chance in the discrimination tasks, this result is in keeping with evidence of newly formed pathways to the intact hemisphere that have been described in hemianopic patient GY who suffers from an early lesion of visual cortex (Bridge, Thomas, Jbabdi, & Cowey, 2008). In principle, the cortical thickness difference could be related to a higher rate of cortical degeneration in the group performing at chance. However, this group had values within the normal range described by Alvarez et al. (2019) and therefore we think it is unlikely that it

underwent substantial degeneration. Of course, the differential degeneration hypothesis remains a **reasonable** possibility. Very recently we became aware of an important study by Georgy et al. (2020) on cortical thickness of the intact visual cortex in three blindsight patients: Two with hemispherectomy (one partial and the other complete) and one with a localized left V1 lesion (the frequently studied patient GY). Their data were compared with 188 healthy control subjects. There was a clear thickness increase of the visual cortex of the intact hemisphere in all three patients compared to healthy controls which rules out the degeneration possibility hinted to for our patients. Interestingly, the authors mention that the increase in cortical thickness of V1 in the intact hemisphere may not be functionally driven; obviously, this requires further studies. However, our evidence of a difference in cortical thickness of the intact hemisphere between hemianopic patients performing above versus at chance in visual discrimination does suggest a functional meaning of the anatomical effect. Other evidence of neuroplastic phenomena comes from the discovery of aberrant fibre connections from the lesioned to the intact hemisphere only in hemispherectomy patients with blindsight (Leh, Johansen-Berg, & Ptito, 2006). Further studies have shown an important role of the intact hemisphere in blindsight of hemianopic patients. For example, Celeghin et al. (2017) in a fMRI study with the Poffenberger paradigm, i.e. a behavioral test of interhemispheric visuomotor transmission (see Marzi, 1999), found that in blindsight patient GY there were compensatory changes resulting in increased connections in posterior regions of the corpus callosum between homologous areas of the parietal cortex. An earlier study by Bittar, Ptito, Faubert, Dumoulin, and Ptito (1999) on three hemispherectomized patients found that only in the one patient with blindsight stimulus presentation to the blind hemifield yielded activation in extrastriate areas V3/V3A and MT of the intact hemisphere. They conclude that the crucial contribution of the intact hemisphere was possible via a SC- Pulvinar route. This route has been demonstrated in anatomical studies in non-human primates (see for example, Kaas & Lyon, 2007; Lyon, Nassi, & Callaway, 2010) As to electrophysiological studies, Kavcic, Triplett, Das, Martin, and Huxlin (2015) in cortically blind patients found that visually evoked potentials following stimulus presentation to the intact visual field could be recorded in the lesioned hemisphere via inter-hemispheric transfer. This was not the case for stimulation of the blind field that did not yield any response. This suggests that a lesioned hemisphere can respond to visual stimulation only following contribution of the intact hemisphere. Further functional evidence of neuroplasticity effects involving the spared hemisphere comes from a series of studies by Nelles et al. (2007, 2002) in hemianopic patients. They found that following visual stimulus presentation to the blind hemifield there was a bilateral BOLD activation of extrastriate cortex which was stronger in the intact hemisphere while the striate cortex was not activated in either hemisphere. Finally, a somewhat unexpected finding was that the Middle Temporal Gyrus presumably hosting the hMT+ was not differentially damaged in patients with and without blindsight. In fact, as shown in Table 2 of the manuscript and in Table 2 of Appendix B, the temporal areas were mostly affected in patients with damage of the Optic Radiation but showed little or no damage in the other groups.

5. Conclusions

In sum, we found that:

I) for unconscious above chance performance in a discrimination of static versus moving visual stimuli the Precuneus (BA 7) and the Posterior Cingulate Gyrus (BA 23, 31) were found to play an important role. Obviously, also visual cortical areas are likely to be important for tasks testing different visual attributes, for example unconscious color processing, as shown recently by Hurme, Koivisto, Henriksson, and Railo (2020) in healthy participants following transcranial magnetic stimulation of V1.

II) for the presence of reports indicating some form of perceptual awareness are crucial striate and extrastriate areas in Intracalcarine, Supracalcarine and Cuneus, that is BA areas 17, 18, 19. In addition, again, the Posterior Cingulate Gyrus is importantly involved.

III) a dominant role for these phenomenological aspects of blindsight is played by the right hemisphere; however, more work is warranted to cast further light on this important finding.

IV) all visual pathways examined in our study showed a greater integrity in patients with perceptual reports but there was no difference among pathways. This is in partial contrast with conclusions of an "unique" role of either the LGN or the SC-Pulvinar pathway for blindsight. Our results highlight the importance of analyzing the contribution of cortical areas in addition to pathways in order to provide a more specific response to cast light on the neural substrate of blindsight.

V) finally, the presence of an increased cortical thickness of the intact hemisphere in patients who showed above chance performance in one or the other task strongly suggests the occurrence of neuroplastic changes even in adult chronic patients.

The limitations of the study are essentially represented by the relatively small number of patients, as discussed in the participants' section. This should not represent a major problem for conclusions on the anatomical bases of blindsight which have robust statistical support. In contrast, evidence on laterality effects certainly requires studies with a higher number of patients with unilateral right or left hemispheric damage.

A final general consideration is that we believe that the thrust of our study is double: Firstly, it provides further evidence on the neural correlates of awareness by showing that perceptual awareness is not an all or nothing phenomenon but may consists of various levels subserved by different

brain structures. Secondly, it provides the anatomical background for focusing efforts of devising new rehabilitation techniques that must necessarily be differently tailored for hemianopic patients with different forms of blindsight or without it.

Authors Contributions

Javier Sanchez-Lopez, Silvia Savazzi, Caterina A. Pedersini and Carlo A. Marzi: Conceptualization, Methodology. Javier Sanchez-Lopez, Caterina A. Pedersini and Nicolo Cardobi: Investigation, Data curation. Javier Sanchez-Lopez, Nicolo Cardobi and Silvia Savazzi: Formal Analysis. Javier Sanchez-Lopez and Carlo A. Marzi: Writing- Original draft preparation. Carlo A. Marzi: Supervision, Funding acquisition. Javier Sanchez-Lopez, Nicolo Cardobi, Silvia Savazzi, Caterina A. Pedersini and Carlo A. Marzi: Writing- Reviewing and Editing.

Funding

This work was supported by the European Research Council (ERC), European Union [Grant number 339939 "Perceptual Awareness" (P.I.: CAM)].

Open practices

The study in this article earned Open Materials and Open Data badges for transparent practices. Behavioral and structural MRI data for this study sufficient to replicate all of the analysis that are reported, as well as the code of the discrimination tasks used for this study, are available at: https://doi.org/10.6084/m9.figshare.11771619.

Acknowledgements

We wish to thank Giorgia Parisi and Gina Joue for helping with behavioral and MRI testing, Valentina Varalta, Cristina Fonte, Massimo Prior, and Nicola Smania for patients' recruitment and clinical screening.

Appendix A. Visual mapping

As described in a previous paper (Sanchez-Lopez et al., 2019), to determine the extension of the blind visual field a binocular visual mapping was performed in the lab. Two-degree square-wave gratings with spatial frequency = 1.75 c/°, Michelson contrast = 1 and background mean luminance = 4.15 cd/m^2, were used. Stimuli were presented on LED video monitor (resolution = 1920 pixels width x 1080 pixels height and refresh rate = 100 Hz) during 150 msec in a pseudo-random order at different eccentricities with an inter-stimulus interval = 1200 msec. Patients were positioned at a distance of 57 cm from the monitor and were instructed to press the space bar of a keyboard as quickly as possible following detection of the stimulus. For patients with a full homonymous hemianopia, stimuli were presented three times randomly in 195 positions in the blind field and 20 in the contralateral sighted field. For patients with quadrantanopia they were presented in 91 positions in the blind and 10 in the sighted field. Data were processed in MATLAB (version 8.2.0, The MathWorks, Inc., Natick, MA, 2010) to assess the extension of the blind visual field in degrees (shown in Table 2) and to generate a greyscale map of the visual field defect to be compared with the patients' clinical campimetry.

Appendix B

Appendix Table 1 Blind visual field extension, distance from fixation point to center of the stimulus, and to medial border of visual field.

Patient	Blind Visual Field Extension (degrees)	Stimulus Position (degrees) from the center of the screen	Stimulus Position (degrees) from the border of the blind visual field
GB	624	x = 9, y = 7; lower visual field	x = 5
RF	636	x = 6, y = 6; upper visual field	x = 4
AP	160	x = 6, y = 11; upper visual field	x = 4, y = 4*
DD	304	x = 7, y = 7; upper visual field	x = 5, y = 7*
LF	260	x = 12.5, y = 8.5; upper visual field	x = 10.5
AM	588	x = 12.5, y = 9; both visual fields	y = 7**
GA	56	x = 13, y = 12; lower visual field	x = 7, y = 4*
HE	616	x = 10, y = 8; lower visual field	x = 6
ML	624	x = 9, y = 5; upper visual field	x = 9
SL	480	x = 21.5, y = 6; upper visual field	x = 19.5
AN	284	x = 8.5, y = 8; lower visual field	x = 4.5
BC	460	x = 9, y = 3; lower visual field	x = 7, y = 3*
FB	556	x = 14.5, y = 8.5; lower visual field	x = 10.5, y = 8.5
GS	540	x = 16, y = 7; upper visual field	x = 10
LB	604	x = 8, y = 8; lower visual field	x = 6, y = 7*
LC	592	x = 14, y = 7; lower visual field	x = 12
RC	368	x = 8, y = 7; upper visual field	x = 8

* The asterisk indicates the distance from x and y border of the blind visual field in patients with quadrantanopia while in those with hemianopia only the distance from x border is shown since vision along the y axis is blind.

** In AM only distance from y border is shown considering that he has an altitudinal hemianopia.

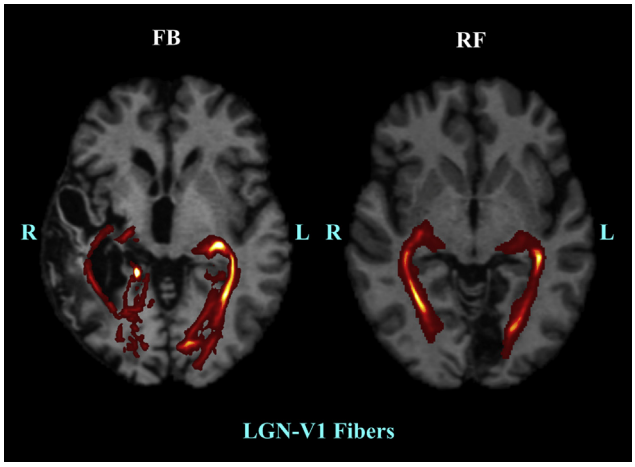
Appendix Table 2 – Percentage of brain damage of single patients and group mean for 30 ROI areas taken from the Harvard–Oxford Atlas

Harvard–Oxford ROI	Group 1			Group 2				Group 3						Group 4				Group 5			
	GB	RF	mean	AP	DD	LF	mean	GA	AM	HE	ML	SL	mean	LB	BC	GS	mean	FB	AN	LC	mean
Intracalcarine	65.25	58.51	61.88	.13	9.04	14.06	7.74	8	16.09	46.67	63.59	63.02	39.47	69.55	83.36	41.95	64.95	12.59	1.05	.69	4.78
Supracalcarine	26.27	28	27.14	0	0	.72	.24	9.46	.38	32.87	14.56	39.34	19.32	84.40	92.23	52.62	76.42	20.55	4.60	2.49	9.21
Lateral Occipital inferior	.31	1.93	1.12	30.34	34.76	0	21.70	32.44	1.26	.05	15.69	4.91	10.87	1.36	1.32	0	.89	61.64	19.37	35.31	38.77
Lateral Occipital superior	0	0	0	.01	.23	0	.08	38.23	0	.09	0	1.40	7.94	9.16	5.72	.03	4.97	60.36	41.32	13.50	38.39
Occipital Fusiform Gyrus	27.50	19.76	23.63	48.14	68.99	.05	39.06	22.79	21.37	.14	77.58	22.70	28.92	1.07	16.17	0	5.75	11.59	.03	11.21	7.61
Occipital Pole	9.88	12.24	11.06	1.04	19.77	.29	7.03	60.65	.43	5.95	37.36	16.92	24.26	36.03	31.69	.62	22.78	11.63	4.28	.21	5.37
Lingual Gyrus	62.99	50.47	56.73	8.96	32.55	8.30	16.60	4.28	30.89	12.79	79.20	30.59	31.55	13.06	33.08	12.54	19.56	3.23	0	1.37	1.53
Cuneus	5.80	8.49	7.15	0	0	.01	0	41.98	0	13.35	.76	29.21	17.06	86.98	79.85	22.29	63.04	15.49	2.75	.89	6.38
Precuneus	3.78	6.79	5.29	0	0	1.01	.34	21.75	1.35	2.33	3.10	7.31	7.17	28.73	29	13.20	23.64	16.08	10.37	1.54	9.33
Temporal Occipital Fusiform	27.29	.42	13.86	40.62	59.72	0	33.45	0	13.24	0	4.13	19.88	7.45	0	0	0	0	8.87	0	27.92	12.26
Angular Gyrus	0	0	0	0	0	0	0	2.32	0	0	0	0	.46	0	0	0	0	100	66.35	24.37	63.57
Cingulate Gyrus posterior	4.32	7.73	6.03	0	0	.44	.15	1.28	3.35	.67	1.93	2.65	1.98	5.58	7.46	6.44	6.49	4.02	8.95	.05	4.34
Heschl Gyrus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	93.45	16.06	.75	36.75
Insular	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	51.37	.32	.58	17.42
Inferior Temporal Gyrus anterior	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	17.05	0	19.47	12.17
Inferior Temporal Gyrus posterior	.09	0	.05	.37	9.45	0	3.27	0	0	0	0	.02	0	0	0	0	0	30.08	0	33.05	21.04
Superior Temporal Gyrus anterior	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	100	0	33.20	44.40
Superior Temporal Gyrus posterior	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	98.57	39.71	55.86	64.71
Temporal Fusiform anterior	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7.69	0	5.41	4.37
Temporal Fusiform posterior	7.90	0	3.95	.44	25.61	0	8.68	0	.96	0	0	5.76	1.34	0	0	0	0	6.83	0	15.59	7.47
Temporal Gyrus posterior	.09	0	.05	.37	9.45	0	3.27	0	0	0	0	.02	0	0	0	0	0	30.08	0	33.05	21.04
Temporal pole	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	54.82	0	5	19.94
Middle Temporal Gyrus anterior	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	79.72	0	65.29	48.34
Middle Temporal Gyrus posterior	0	0	0	0	.03	0	.01	0	0	0	0	0	0	0	0	0	0	85.26	15.56	71.77	57.53
Middle Temporal Gyrus temporo-occipital	0	0	0	5.78	6.90	0	4.23	0	0	0	0	0	0	0	0	0	0	100	40.34	83.69	74.68
Parietal Operculum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	95.57	65.93	2.39	54.63
Planum Temporale	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	89.41	59.75	5.52	51.56
Superior Parietal Lobule	0	0	0	0	0	0	0	52.55	0	0	0	0	10.51	0	0	0	0	64.86	13.86	1.46	26.73
Supramarginal Gyrus anterior	0	0	0	0	0	0	0	8.21	0	0	0	0	1.64	0	0	0	0	88.72	36.95	0	41.89
Supramarginal Gyrus posterior	0	0	0	0	0	0	0	7.75	0	0	0	0	1.55	0	0	0	0	95.77	64.56	18.37	59.57
Total Mean	8.05	6.48	7.26	4.54	9.22	.83	4.86	10.39	2.98	3.83	9.93	8.12	7.05	11.20	12.66	4.99	9.62	50.51	17.07	19.00	28.86

Appendix C. hMT + Functional Localizer

The hMT + localizer consisted of the assessment of the hemodynamic response (blood oxygen level dependent – BOLD) during the presentation of a moving versus stationary stimulus. A block design paradigm with 2 runs (each lasting 350 sec), one presented in the blind and the other in the sighted visual field, was used. Each run was composed by 12 stimulus presentation and 13 rest blocks of 14 sec alternating among rest, moving and stationary blocks in a fixed order. Stimulation was generated using Matlab (version R2013b TheMathWorks Inc., Natick. MA, United States) and consisted of 300 black randomly moving or stationary dots, shown within an aperture of 4° in either the blind or sighted hemifield. Patients were asked to fixate a central dot during the entire session without giving any response. Functional images were acquired covering almost the whole brain by recording from slices parallel to the calcarine scissure. One hundred volumes were acquired (T2*-weighted echo-planar imaging, 32 slices acquired in an ascending order, repetition time = 2000 msec, echo time = 35 msec, field of view = 230 × 230, FA = 30°) and for each run 4 dummy scans were added at the beginning in order to avoid T1 saturation. Preprocessing and statistical analyses were performed by using tools from FSL and consisted of non-brain tissue extraction using BET (Brain Extraction Tool); motion correction using MCFLIRT (FMRIB Linear Image Restoration Tool with Motion Correction); spatial smoothing using a Gaussian kernel of FWHM of 5 mm and a high-pass temporal filtering; and finally the functional images were registered to both high-resolution structural images using FLIRT after applying BET and to a standard MNI brain template using both FLIRT and FNIRT (FMRIB Nonlinear Image Registration Tool). Localization of the functional hMT + areas was performed for each single participant. BOLD time course data were analyzed using a General Linear Model (GLM) approach with motion and static conditions as regressors. A moving > stationary dots GLM contrast was computed by applying a cluster thresholding ($z = 2.3$, $p < .001$) with the corresponding cluster defining threshold ($p = .05$). The final unthresholded image was masked with the hMT + ROI extracted from area V5 in the Juelich Atlas to delimit the borders of functional area hMT + to be used in the following analyses. Finally, a mean hMT + ROI was calculated and used separately for each hemisphere. The above described procedure was performed with patients AM, AP, BC, DD, HE, FB, LF, SG, AN and SL. The other patients did not attend the hMT + Localizer session. For statistics, an overall group mean hMT + ROI was calculated and used separately for each hemisphere.

Appendix D. Example of Optic Radiation Fibers in two hemianopic patients



Appendix Fig. 1 – Example of LGN-V1 fibers from patient FB (Group 5) with massive lesion of the right Optic Radiation and patient RF (Group 1) with intact Optic Radiation. Right and left are radiologically oriented.

REFERENCES

Ajina, S., & Bridge, H. (2017). Blindsight and unconscious vision: What they teach us about the human visual system. *The Neuroscientist: a Review Journal Bringing Neurobiology, Neurology and Psychiatry, 23*(5), 529–541. https://doi.org/10.1177/1073858416673817

Ajina, S., & Bridge, H. (2018). Blindsight relies on a functional connection between hMT+ and the lateral geniculate nucleus, not the pulvinar. *Plos Biology, 16*(7). https://doi.org/10.1371/journal.pbio.2005769

Ajina, S., Kennard, C., Rees, G., & Bridge, H. (2015). Motion area V5/MT+ response to global motion in the absence of V1 resembles early visual cortex. *Brain, 138*(1), 164–178. https://doi.org/10.1093/brain/awu328

Ajina, S., Pestilli, F., Rokem, A., Kennard, C., & Bridge, H. (2015). Human blindsight is mediated by an intact geniculo-extrastriate pathway. *ELife, 4*(2015), 1–23. https://doi.org/10.7554/elife.08935

Alvarez, I., Parker, A. J., & Bridge, H. (2019). Normative cerebral cortical thickness for human visual areas. *NeuroImage, 201*, 116057. https://doi.org/10.1016/j.neuroimage.2019.116057

Andersson, P., Ragni, F., & Lingnau, A. (2019). Visual imagery during real-time fMRI neurofeedback from occipital and

superior parietal cortex. *NeuroImage, 200*, 332–343. https://doi.org/10.1016/j.neuroimage.2019.06.057
Antal, A., Baudewig, J., Paulus, W., & Dechent, P. (2008). The posterior cingulate cortex and planum temporale/parietal operculum are activated by coherent visual motion. *Visual Neuroscience, 25*(1), 17–26. https://doi.org/10.1017/S0952523808080024
Barbur, J. L., Watson, J. D. G., Frackowiak, R. S. J., & Zeki, S. (1993). Conscious visual perception without VI. *Brain, 116*(6), 1293–1302. https://doi.org/10.1093/brain/116.6.1293
Behrens, T. E. J., Berg, H. J., Jbabdi, S., Rushworth, M. F. S., & Woolrich, M. W. (2007). Probabilistic diffusion tractography with multiple fibre orientations: What can we gain? *NeuroImage, 34*(1), 144–155. https://doi.org/10.1016/j.neuroimage.2006.09.018
Behrens, T. E. J., Woolrich, M. W., Jenkinson, M., Johansen-Berg, H., Nunes, R. G., Clare, S., et al. (2003). Characterization and propagation of uncertainty in diffusion-weighted MR imaging. *Magnetic Resonance in Medicine, 50*(5), 1077–1088. https://doi.org/10.1002/mrm.10609
Bittar, R. G., Ptito, M., Faubert, J., Dumoulin, S. O., & Ptito, A. (1999). Activation of the remaining hemisphere following stimulation, of the blind hemifield in hemispherectomized subjects. *NeuroImage, 10*(3 I), 339–346. https://doi.org/10.1006/nimg.1999.0474
Bouwmeester, L., Heutink, J., & Lucas, C. (2007). The effect of visual training for patients with visual field defects due to brain damage: A systematic review. *Journal of Neurology, Neurosurgery, and Psychiatry, 78*(6), 555–564. https://doi.org/10.1136/jnnp.2006.103853
Bridge, H., Thomas, O., Jbabdi, S., & Cowey, A. (2008). Changes in connectivity after visual cortical brain damage underlie altered visual function. *Brain, 131*(6), 1433–1444. https://doi.org/10.1093/brain/awn063
Brogaard, B. (2015). Type 2 blindsight and the nature of visual experience. *Consciousness and Cognition, 32*, 92–103. https://doi.org/10.1016/j.concog.2014.09.017
Campion, J., Latto, R., & Smith, Y. M. (1983). Is blindsight an effect of scattered light, spared cortex, and near-threshold vision? *Behavioral and Brain Sciences, 6*(3), 423–448. https://doi.org/10.1017/S0140525X00016861
Cavanna, A. E., & Trimble, M. R. (2006). The precuneus: A review of its functional anatomy and behavioural correlates. *Brain, 129*(3), 564–583. https://doi.org/10.1093/brain/awl004
Celeghin, A., Bagnis, A., Diano, M., Méndez, C. A., Costa, T., & Tamietto, M. (2019). Functional neuroanatomy of blindsight revealed by activation likelihood estimation meta-analysis. *Neuropsychologia, 128*, 109–118. https://doi.org/10.1016/j.neuropsychologia.2018.06.007
Celeghin, A., Barabas, M., Mancini, F., Bendini, M., Pedrotti, E., Prior, M., et al. (2015). Speeded manual responses to unseen visual stimuli in hemianopic patients: What kind of blindsight? *Consciousness and Cognition, 32*, 6–14. https://doi.org/10.1016/j.concog.2014.07.010
Celeghin, A., Diano, M., De Gelder, B., Weiskrantz, L., Marzi, C. A., & Tamietto, M. (2017). Intact hemisphere and corpus callosum compensate for visuomotor functions after early visual cortex damage. *Proceedings of the National Academy of Sciences of the United States of America, 114*(48), E10475–E10483. https://doi.org/10.1073/pnas.1714801114
Cowey, A. (2010). The blindsight saga. *Experimental Brain Research, 200*(1), 3–24. https://doi.org/10.1007/s00221-009-1914-2
Dale, A. M., Fischl, B., & Sereno, M. I. (1999). Cortical surface-based analysis: I. Segmentation and surface reconstruction. *NeuroImage, 9*(2), 179–194. https://doi.org/10.1006/nimg.1998.0395
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*(7), 1035–1046. https://doi.org/10.1016/j.neubiorev.2005.02.001
Danckert, J., Tamietto, M., & Rossetti, Y. (2019). Definition: Blindsight. *Cortex, 119*, 569–570. https://doi.org/10.1016/j.cortex.2019.01.027
de Groot, M., Vernooij, M. W., Klein, S., Ikram, M. A., Vos, F. M., Smith, S. M., et al. (2013). Improving alignment in Tract-based spatial statistics: Evaluation and optimization of image registration. *NeuroImage, 76*, 400–411. https://doi.org/10.1016/j.neuroimage.2013.03.015
Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods, 134*(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009
Dundon, N. M., Bertini, C., Làdavas, E., Sabel, B. A., & Gall, C. (2015). Visual rehabilitation: Visual scanning, multisensory stimulation and vision restoration trainings. *Frontiers in Behavioral Neuroscience, 9*(192), 1–14. https://doi.org/10.3389/fnbeh.2015.00192
Fendrich, R., Wessinger, C. M., & Gazzaniga, M. S. (2001). Speculations on the neural basis of islands of blindsight. *Progress in Brain Research, 134*, 353–366. https://doi.org/10.1016/S0079-6123(01)34023-2
Field, D. T., Inman, L. A., & Li, L. (2015). Visual processing of optic flow and motor control in the human posterior cingulate sulcus. *Cortex, 71*, 377–389. https://doi.org/10.1016/j.cortex.2015.07.014
Fischl, B., & Dale, A. M. (2000). Measuring the thickness of the human cerebral cortex from magnetic resonance images. *Proceedings of the National Academy of Sciences of the United States of America, 97*(20), 11050–11055. https://doi.org/10.1073/pnas.200033797
Fischl, B., Sereno, M. I., & Dale, A. M. (1999). Cortical surface-based analysis: II. Inflation, flattening, and a surface-based coordinate system. *NeuroImage, 9*(2), 195–207. https://doi.org/10.1006/nimg.1998.0396
Fischl, B., Van Der Kouwe, A., Destrieux, C., Halgren, E., Ségonne, F., Salat, D. H., et al. (2004). Automatically parcellating the human cerebral cortex. *Cerebral Cortex, 14*(1), 11–22. https://doi.org/10.1093/cercor/bhg087
Foley, R. (2015). The case for characterising type-2 blindsight as a genuinely visual phenomenon. *Consciousness and Cognition, 32*, 56–67. https://doi.org/10.1016/j.concog.2014.09.005
Garric, C., Sebaa, A., Caetta, F., Perez, C., Savatovsky, J., Sergent, C., et al. (2019). Dissociation between objective and subjective perceptual experiences in a population of hemianopic patients: A new form of blindsight? *Cortex, 117*, 299–310. https://doi.org/10.1016/j.cortex.2019.05.006
Georgy, L., Celeghin, A., Marzi, C. A., Tamietto, M., & Ptito, A. (2016). The superior colliculus is sensitive to gestalt-like stimulus configuration in hemispherectomy patients. *Cortex, 81*, 151–161. https://doi.org/10.1016/j.cortex.2016.04.018
Georgy, L., Lewis, J. D., Bezgin, G., Diano, M., Celeghin, A., Evans, A. C., et al. (2020). Changes in peri-calcarine cortical thickness in blindsight. *Neuropsychologia, 143*, 107463. https://doi.org/10.1016/j.neuropsychologia.2020.107463
Goodwin, D. (2014). Homonymous hemianopia: Challenges and solutions. *Clinical Ophthalmology, 8*, 1919–1927. https://doi.org/10.2147/OPTH.S59452
Hervais-Adelman, A., Legrand, L. B., Zhan, M., Tamietto, M., de Gelder, B., & Pegna, A. J. (2015). Looming sensitive cortical regions without V1 input: Evidence from a patient with bilateral cortical blindness. *Frontiers in Integrative Neuroscience, 9*(OCTOBER), 1–12. https://doi.org/10.3389/fnint.2015.00051
Hurme, M., Koivisto, M., Henriksson, L., & Railo, H. (2020). Neuronavigated TMS of early visual cortex eliminates unconscious processing of chromatic stimuli.

Neuropsychologia, 136, 107266. https://doi.org/10.1016/j.neuropsychologia.2019.107266

Jenkinson, M., Beckmann, C. F., Behrens, T. E. J., Woolrich, M. W., & Smith, S. M. (2012). FSL. *NeuroImage, 62*(2), 782–790. https://doi.org/10.1016/j.neuroimage.2011.09.015

Jones, D. K., Knösche, T. R., & Turner, R. (2013). White matter integrity, fiber count, and other fallacies: The do's and don'ts of diffusion MRI. *NeuroImage, 73*, 239–254. https://doi.org/10.1016/j.neuroimage.2012.06.081

Joseph, R. (1988). The right cerebral hemisphere: Emotion, music, visual-spatial skills, body-image, dreams, and awareness. *Journal of Clinical Psychology, 44*(5), 630–673. https://doi.org/10.1002/1097-4679(198809)44:5<630::AID-JCLP2270440502>3.0.CO;2-V

Kaas, J. H., & Lyon, D. C. (2007). Pulvinar contributions to the dorsal and ventral streams of visual processing in primates. *Brain Research Reviews, 55*(2 SPEC. ISS.), 285–296. https://doi.org/10.1016/j.brainresrev.2007.02.008

Kavcic, V., Triplett, R. L., Das, A., Martin, T., & Huxlin, K. R. (2015). Role of inter-hemispheric transfer in generating visual evoked potentials in V1-damaged brain hemispheres. *Neuropsychologia, 68*, 82–93. https://doi.org/10.1016/j.neuropsychologia.2015.01.003

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. https://doi.org/10.1016/j.concog.2014.08.005

Khalsa, S., Mayhew, S. D., Chechlacz, M., Bagary, M., & Bagshaw, A. P. (2014). The structural and functional connectivity of the posterior cingulate cortex: Comparison between deterministic and probabilistic tractography for the investigation of structure-function relationships. *NeuroImage, 102*(P1), 118–127. https://doi.org/10.1016/j.neuroimage.2013.12.022

Kinoshita, M., Kato, R., Isa, K., Kobayashi, K., Kobayashi, K., Onoe, H., et al. (2019). Dissecting the circuit for blindsight to reveal the critical role of pulvinar and superior colliculus. *Nature Communications, 10*(1), 1–10. https://doi.org/10.1038/s41467-018-08058-0

LaFleur, B. J., & Greevy, R. A. (2009). Introduction to permutation and resampling-based hypothesis tests. *Journal of Clinical Child and Adolescent Psychology, 38*(2), 286–294. https://doi.org/10.1080/15374410902740411

Leech, R., & Sharp, D. J. (2014). The role of the posterior cingulate cortex in cognition and disease. *Brain, 137*(1), 12–32. https://doi.org/10.1093/brain/awt162

Leh, S. E., Johansen-Berg, H., & Ptito, A. (2006). Unconscious vision: New insights into the neuronal correlate of blindsight using diffusion tractography. *Brain, 129*(7), 1822–1832. https://doi.org/10.1093/brain/awl111

Ludbrook, J., & Dudley, H. (1998). Why permutation tests are superior to t and F tests in biomedical research. *American Statistician, 52*(2), 127–132. https://doi.org/10.1080/00031305.1998.10480551

Lyon, D. C., Nassi, J. J., & Callaway, E. M. (2010). A disynaptic relay from superior colliculus to dorsal stream visual cortex in macaque monkey. *Neuron, 65*(2), 270–279. https://doi.org/10.1016/j.neuron.2010.01.003

Marzi, C. A. (1999). The Poffenberger paradigm: A first, simple, behavioural tool to study interhemispheric transmission in humans. *Brain Research Bulletin, 50*(5–6), 421–422. https://doi.org/10.1016/S0361-9230(99)00174-4

Mazzi, C., Bagattini, C., & Savazzi, S. (2016). Blind-sight vs. degraded-sight: Different measures tell a different story. *Frontiers in Psychology, 7*(901), 1–11. https://doi.org/10.3389/fpsyg.2016.00901

Mazzi, C., & Savazzi, S. (2019). The glamour of old-style single-case studies in the neuroimaging era: Insights from a patient with hemianopia. *Frontiers in Psychology, 10*(956), 1–5. https://doi.org/10.3389/fpsyg.2019.00965

Mazzi, C., Savazzi, S., & Silvanto, J. (2019). On the "blindness" of blindsight: What is the evidence for phenomenal awareness in the absence of primary visual cortex (V1)? *Neuropsychologia, 128*, 103–108. https://doi.org/10.1016/j.neuropsychologia.2017.10.029

Mazzi, C., Tagliabue, C. F., Mazzeo, G., & Savazzi, S. (2019). Reliability in reporting perceptual experience: Behaviour and electrophysiology in hemianopic patients. *Neuropsychologia, 128*, 119–126. https://doi.org/10.1016/j.neuropsychologia.2018.01.017

Nelles, G., de Greiff, A., Pscherer, A., Forsting, M., Gerhard, H., Esser, J., et al. (2007). Cortical activation in hemianopia after stroke. *Neuroscience Letters, 426*(1), 34–38. https://doi.org/10.1016/j.neulet.2007.08.028

Nelles, G., Widman, G., De Greiff, A., Meistrowitz, A., Dimitrova, A., Weber, J., et al. (2002). Brain representation of hemifield stimulation in poststroke visual field defects. *Stroke, 33*(5), 1286–1293. https://doi.org/10.1161/01.STR.0000013685.76973.67

Overgaard, M. (2011). Visual experience and blindsight: A methodological review. *Experimental Brain Research, 209*(4), 473–479. https://doi.org/10.1007/s00221-011-2578-2

Overgaard, M., Fehl, K., Mouridsen, K., Bergholt, B., & Cleeremans, A. (2008). Seeing without seeing? Degraded conscious vision in a blindsight patient. *Plos One, 3*(8), Article e3028. https://doi.org/10.1371/journal.pone.0003028

Overgaard, M., & Mogensen, J. (2015). Reconciling current approaches to blindsight. *Consciousness and Cognition, 32*, 33–40. https://doi.org/10.1016/j.concog.2014.08.003

Phillips, I. (2019). *Making sense of blindsense: A commentary on Garric et al., 2019*. Cortex. https://doi.org/10.1016/j.cortex.2019.11.016

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

Pustina, D., Avants, B., Faseyitan, O. K., Medaglia, J. D., & Coslett, H. B. (2018). Improved accuracy of lesion to symptom mapping with multivariate sparse canonical correlations. *Neuropsychologia, 115*, 154–166. https://doi.org/10.1016/j.neuropsychologia.2017.08.027

Roalf, D. R., & Gur, R. C. (2017). Functional brain imaging in neuropsychology over the past 25 years. *Neuropsychology, 31*(8), 954–971. https://doi.org/10.1037/neu0000426

Sahraie, A., Hibbard, P. B., Trevethan, C. T., Ritchie, K. L., & Weiskrantz, L. (2010). Consciousness of the first order in blindsight. *Proceedings of the National Academy of Sciences of the United States of America, 107*(49), 21217–21222. https://doi.org/10.1073/pnas.1015652107

Sanchez-Lopez, J., Pedersini, C. A., Di Russo, F., Cardobi, N., Fonte, C., Varalta, V., et al. (2019). Visually evoked responses from the blind field of hemianopic patients. *Neuropsychologia, 128*, 127–139. https://doi.org/10.1016/j.neuropsychologia.2017.10.008

Sanchez-Lopez, J., Savazzi, S., Pedersini, C. A., Cardobi, N., & Marzi, C. A. (2020). Neural bases of unconscious orienting of attention in hemianopic patients: Hemispheric differences. *Cortex, 127*, 269–289. https://doi.org/10.1016/j.cortex.2020.02.015

Schmid, M. C., Mrowka, S. W., Turchi, J., Saunders, R. C., Wilke, M., Peters, A. J., et al. (2010). Blindsight depends on the lateral geniculate nucleus. *Nature, 466*(7304), 373–377. https://doi.org/10.1038/nature09179

Smits, A. R., Seijdel, N., Scholte, H. S., Heywood, C. A., Kentridge, R. W., & de Haan, E. H. F. (2019). Action blindsight and antipointing in a hemianopic patient. *Neuropsychologia, 128*, 270–275. https://doi.org/10.1016/j.neuropsychologia.2018.03.029

Thomas, J. O., & Barrett, A. M. (2019). Right brain stroke syndromes. In R. Wilson, & P. B. T.-S. R. Raghavan (Eds.), *Stroke rehabilitation* (pp. 71–89). Elsevier. https://doi.org/10.1016/b978-0-323-55381-0.00005-6.

Tomaiuolo, F., Ptito, M., Marzi, C. A., Paus, T., & Ptito, A. (1997). Blindsight in hemispherectomized patients as revealed by spatial summation across the vertical meridian. *Brain, 120*(5), 795–803. https://doi.org/10.1093/brain/120.5.795

Tran, A., MacLean, M. W., Hadid, V., Lazzouni, L., Nguyen, D. K., Tremblay, J., et al. (2019). Neuronal mechanisms of motion detection underlying blindsight assessed by functional magnetic resonance imaging (fMRI). *Neuropsychologia, 128*, 187–197. https://doi.org/10.1016/j.neuropsychologia.2019.02.012

van den Berg, E., & Ruis, C. (2017). Space in neuropsychological assessment. In A. Postma, & I. J. M. van der Ham (Eds.), *Neuropsychology of space: Spatial functions of the human brain* (pp. 361–378). Elsevier Inc. https://doi.org/10.1016/B978-0-12-801638-1.00010-0.

Vogt, B. A., & Devinsky, O. (2000). Topography and relationships of mind and brain. In E. A. Mayer, & C. B. Saper (Eds.), *Progress in brain research* (Vol. 122, pp. 11–22). Elsevier Science BV. https://doi.org/10.1016/S0079-6123(08)62127-5.

Wang, R. W. Y., Chang, W. L., Chuang, S. W., & Liu, I. N. (2019). Posterior cingulate cortex can be a regulatory modulator of the default mode network in task-negative state. *Scientific Reports, 9*(1), 1–12. https://doi.org/10.1038/s41598-019-43885-1

Weiskrantz, L. (1996). Blindsight revisited. *Current Opinion in Neurobiology, 6*(2), 215–220. https://doi.org/10.1016/S0959-4388(96)80075-4

Weiskrantz, L., Barbur, J. L., & Sahraie, A. (1995). Parameters affecting conscious versus unconscious visual discrimination with damage to the visual cortex (V1). *Proceedings of the National Academy of Sciences of the United States of America, 92*(13), 6122–6126. https://doi.org/10.1073/pnas.92.13.6122

Weiskrantz, L., Warrington, E. K., Sanders, M. D., & Marshall, J. (1974). Visual capacity in the hemianopic field following a restricted occipital ablation. *Brain, 97*(1), 709–728.

Werring, D. J., Toosy, A. T., Clark, C. A., Parker, G. J., Barker, G. J., Miller, D. H., et al. (2000). Diffusion tensor imaging can detect and quantify corticospinal tract degeneration after stroke. *Journal of Neurology, Neurosurgery, and Psychiatry, 69*(2), 269–272.

Whitlock, J. R. (2017). Posterior parietal cortex. *Current Biology, 27*(14), R691–R695. https://doi.org/10.1016/j.cub.2017.06.007

Woolrich, M. W., Jbabdi, S., Patenaude, B., Chappell, M., Makni, S., Behrens, T., et al. (2009). Bayesian analysis of neuroimaging data in FSL. *NeuroImage, 45*, S173–S186. https://doi.org/10.1016/j.neuroimage.2008.10.055

Yushkevich, P. A., Piven, J., Hazlett, H. C., Smith, R. G., Ho, S., Gee, J. C., et al. (2006). User-guided 3D active contour segmentation of anatomical structures: Significantly improved efficiency and reliability. *NeuroImage, 31*(3), 1116–1128. https://doi.org/10.1016/j.neuroimage.2006.01.015

Zeki, S., & Ffytche, D. H. (1998). The Riddoch syndrome: Insights into the neurobiology of conscious vision. *Brain*. https://doi.org/10.1093/brain/121.1.25

Zhang, X., Kedar, S., Lynn, M. J., Newman, N. J., & Biousse, V. (2006). Homonymous hemianopias: Clinical-anatomic correlations in 904 cases. *Neurology, 66*(6), 906–910. https://doi.org/10.1212/01.wnl.0000203913.12088.93

Zihl, J. (2010). Rehabilitation of visual disorders after brain injury. In *Rehabilitation of visual disorders after brain injury* (2nd ed.). Psychology Press. https://doi.org/10.4324/9780203843253.