# **Original Research Article**

# Detecting blindsight in the clinical practice: a case-miniseries investigation.

#### **ABSTRACT**

**Aims:** After occipital damage, hemianopic patients may respond to targets presented in their non-responsive hemifield even if they are unaware of their presence. This phenomenon, called blindsight, is probably mediated by an extrastriate route and can be revealed by forced-choice response paradigms. Since blindsight can help patients avoid obstacles and prevent collisions, and can be trained, identifying blindsight subjects within the clinical setting deserves consideration. This preliminary investigation assesses the effectiveness of a simple and fast procedure devised on purpose in four well-studied hemianopic patients.

Study design: Case-series.

**Place and Duration of Study:**University of Turin, Dept of Ophthalmology, between February 2023 and July 2024.

**Methodology:**Flickering sinusoidal gratings (temporal frequency: 10 Hz) were briefly presented in the blind hemifield above or below the horizontal meridian. Fixation was strictly monitored. Subjects had to report, according to a 2AFC (Alternative-Forced Choice) temporal paradigm, in which interval the stimulus was presented.

**Results:**A proportion of correct responses significantly above chance was recorded in subject DC (60.94%,  $\chi^2 = 6.08$ , p: .013) with selective deafferentiation of the striate cortex whereas the performance of the other patients, with more extensive damage, was at a chance level.

**Conclusion:**In line with the literature, blindsight depends on the extent of the cerebral injury. This simple and fast procedure (examination time: approximately 20 minutes) seems promising for selecting, within the clinical setting, patients who should be further investigated for blindsight. Within a translational framework, the aim is to customize and optimize rehabilitation programs.

Keywords: blindsight; hemianopia; sinewave grating; stroke; alternative forced-choice

#### 1. INTRODUCTION

Since the fifties, a bulk of research has shown that, in the presence of striate deafferentiation or destruction, unconscious visual functions may persist in the affected region of the visual field: this phenomenon is known as blindsight. Indeed, after occipital or geniculostriate infarction or surgical ablation a proportion of patients may respond to targets presented in their blind hemifield, even if they are not consciously aware of their presence. This ability has been revealed by laboratory studies where observers with blindsight proved to guess well above chance in psychophysical forced-choice response paradigms: this is the case of detection of brief flashes of light or sinewave gratings in the blind hemifield [1-3], discrimination of color and colored stimuli [4-6], orientation [6,7], form [1,8], and especially motion [9-11]. When asked, subjects state that they see nothing so they cannot but guess: and yet, the proportion of correct responses is above chance.

Although blindsight has been supposed to be a mere effect of light scattering [12] or spared islands of intact function in area 17 [13-16] that go unnoticed with conventional perimetry, both hypotheses seem unlikely [17-20].

Blindsight is spatiotemporally modulated: In a patient with left striate lesion and right homonymous hemianopia, Barbur and colleaguesestimated the sensibility of the hemianopic field to Gabor patches with spatial frequency of 1.1 c/deg flickering at different temporal frequencies and, in turn, of Gabor patches with different spatial frequencies flickering at 6 Hz. They found a a bandpass response extending from 0.3 to 8 c deg-1 with a peak of sensitivity at approximately 1.2 c deg-1 and 10 Hz. [2]

Similarly,in two patients with homonymous hemianopia, Sahraie[3] found that when using gratings with spatial frequencies below 3.5 c deg-1 and a temporal frequency of 10 Hz, detection was well above chance, with the best performance at about 1 c deg-1. It was concluded that the best stimulus configuration to elicit blindsight has a temporal frequency higher than 3.7 Hz and a spatial frequency below 8 c deg-1 whereas stimulus duration is irrelevant [21].

There is consistent evidence that "blind vision" is mediated by extrastriate circuits, namely a geniculate extrastriate route [22,23] and/or a retinotectal route that passes through the superior colliculi [6,24]. A geniculate-extrastriate pathway that projects directly to V2, V3, V4, and to the medial-temporal region (MT or V5) has been documented in the macaque monkey [e.g., 22; 25]. This neuronal circuit can elucidate the perception of orientation, form, and color in the hemianopic field of blindsight subjects [6, 26].

Furthermore, the contribution of retinotectal (extrageniculate) projections that reach the extrastriate cortex via the superior colliculus has been suggested [6,24]: Standage and Beneventodescribed in the macague monkey a retino-collicular pathway that projects to the pulvinar and reaches V5 bypassing V1 [27], and it is noteworthy that activity in the superior colliculus has been observed in human subjects with unconscious perception of stimuli moving in the hemianopic field [28]. The potential routes are not restricted to pre-existing (and potentially enhanced) extrastriate pathways but may involve novel connections that are absent in normal subjects and in patients who do not exhibit blindsight. Specifically, there is evidence of crossed projections from the ipsilesional MT+/V5 to the contralesional lateral geniculate nucleus [29]. Because blindsight may help hemianopic subjects unconsciously avoid obstacles and prevent collisions [30], and it can be trained [31-32], the routine detection of this function in subjects with retrogeniculate lesions deserves consideration. Nevertheless, this phenomenon remains unobserved within the clinical practice and is not subjected to investigation. Testing blindsight, indeed, requires strictly controlled environmental conditions and sophisticated psychophysical procedures, and is time-consuming because of the high number of trials that must be administered. In a study by Weiskrantz, for example, the assessment of residual functions related to blindsight in a patient with surgical removal of an arterovenous malformation required several days of testing [1]. More recently, the group of Sahraie administered several hundred presentations to assess blindsight over a range of spatial frequencies [3] and Azzopardi and Cowey made use of up to 100 trials repeated for different stimulus conditions to investigate blindsight-related motion discrimination in a sample of patients [33]. It is worth considering if it is possible to detect blindsight within the clinical setting, where there is a necessity for a limited number of trials and non-expensive, user-friendly technologies. Indeed, this preliminary, exploratory study aims to probe this possibility by administering a simple, fast, and inexpensive test to four patients with wellcharacterized cerebral lesions. To anticipate, the results obtained in one case are suggestive for the diagnosis. However, a more extensive and rigorous examination of this patient is required to confirm the presence of the phenomenon.

#### 2. MATERIAL AND METHODS

#### 2.1Subjects

The four patients were recruited from the Neuro-Ophthalmology department of the University of Turin.

GO is a male, 81 years old. His clinical history included ischemic coronaropathy with atrial fibrillation, hypercholesterolemia, and hypertension. He suffered from a cerebrovascular infarction in October 2022 (time since onset: 6 months). The lesion involved the left temporo-occipital region and two small ischemic regions at the pons due to occlusion of the left posterior cerebral artery; perimetry showed right homonymous hemianopia with macular sparing (figure 1). GO was emmetropic with VA (Visual Acuity) 1.0 and a slight exophoria of 4D for near distance. According to the patient, the accident has not consistently affected his quality of life: navigation was normal and he did not complain about his visual field loss. He retained his driving license and adopted compensatory head movements behind the wheel.

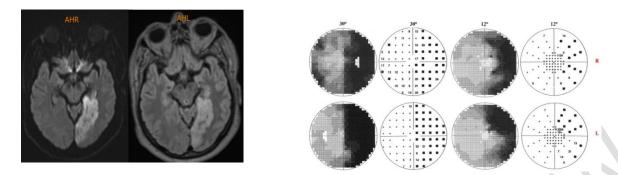


Fig. 1.GO: (left) The lesion at MRI; (right) The perimetric defect: 30 and 12 central degrees. Octopus, Haag-Streit Perimeter, grayscale and comparison maps.

PB is a male, 70 years old. His clinical history includes hypertension and bilateral cataract surgery 10 years earlier. In 1999 an ischemic infarction following right internal carotid dissection damaged his right frontal-insular-temporal-parietal region. The visual field showed left homonymous hemianopia with macular sparing (figure 2). BCVA (Best Corrected Visual Acuity) was 1.0 with a myopic correction of -2.50 spherical equivalent (RE, right eye) and -2.25 spherical equivalent (LE, left Eye). PB has an exophoria of 14 D for far distance and exophoria/tropia for near distance. In addition, the patient suffers from complete left hemiparesis and uses a cane to navigate. When interviewed, PB complained about the reduction of his quality of life due to navigation and motor problems and to his visual field loss that made him struggle to watch the TV, at the cinema, and when reading. Once hemianopia had been ascertained, his driving license was withdrawn. The patient, highly motivated, did not show evident compensatory head movements.

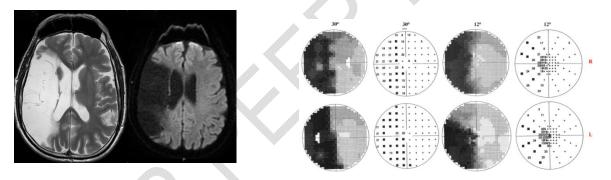
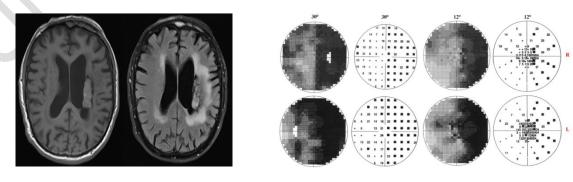


Fig. 2. PB: (left) The lesion at MRI; (right) The perimetric defect: 30 and 12 central degrees

AF is a male, 67 years old. His clinical history includes a pacemaker. A cerebral hemorrhage took place in January 2020: the lesion involved the left nucleo-capsular region extending to the omolateralponto-mesencephalic region and the left occipital region. The visual field showed right homonymous hemianopia with macular sparing. In addition, the patient suffers from glaucoma (cup-disk ratio: 0.8) in therapy with timolol+dorzolamide bid, with a superior and inferior fascicular defect in the spared hemifield (more evident in the left eye: figure 3). His VA was 0.8 (RE, Right Eye) and 0.7 (LE, Left Eye) with no correction. AF has complete right hemiparesis and uses a wheelchair. When interviewed, AF reported the sensation of a severe reduction in his quality of life but did not complain about his visual problem. And yet AF, who did not show compensatory head movements, was quite motivated to improve his visual condition.



#### Fig. 3. AF: (left) The lesion at MRI; (right) The perimetric defect: 30 and 12 central degrees

DC is a woman, 48 years old. A cerebral meningioma was removed on August, 30th, 2022. The lesion (5 cm in size with perilesional edema) was located in the right frontal-parietal region with the involvement of the right lateral geniculate nucleus. After surgery, the visual field showed left superior homonymous quadrantanopia with macular sparing (figure 4). The clinical history was silent. VA was 1.0 with no correction in both eyes. She does not complain about quadrantanopia per se but suffers from a moderate reduction of quality of life for her psychological conditions. DC does not use compensatory head movements and appears highly motivated and collaborative during the testing session.

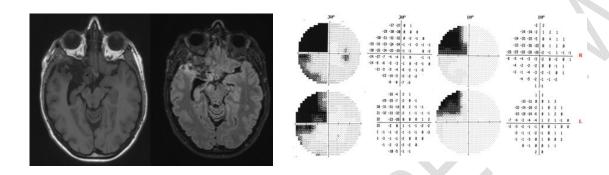


Fig. 4. DC: (left) The lesion at MRI; (right) The perimetric defect: 30 and 12 central degrees

The orthoptic and refractive assessment of the four subjects is summarized below (table 1).

1.0

Refr RE, LE **BCVA** Cover test Cover test Near Stereopsis (sph eq) RE, LE far distance near distance conv point (Lang test II) GO 1.0, Exophoria 4 D ortophoria 6 cm present 1.0 PB 1.0, exoph 14 D -2.50, -2.25 exophoria/tropia 10 cm absent 1.0 AF - ,-0.8, ortophoria orthophoria 7 cm absent 0.7 DC 1.0, ortophoria exophoria 6 cm present

Table 1. Refraction and orthoptic situation in the four patients.

## 2.2Stimuli and procedure

The procedure was similar to that used in previous studies [2,35]. Stimuli were generated on a PC screen (18", Prechen HD-190, resolution 1289 x 1024) using an SVGA graphic card at a 60 Hz refresh rate. The screen, 32°x 21° wide at a viewing distance of 70 cm, was enclosed in a wooden frame, with its borders and the inside covered with black matt felt to neutralize light scattering from the surroundings.

As a fixation reference, a black cross (24.6 arc min wide) was steadily presented at the extreme left or right (depending on the side of the defect) border of the screen. Fixation was constantly monitored via an infrared micro camera (Sony CCD)

placed on a trial frame to the side of the non-examined eye. When an even small eye movement was noticed during the examination or when the subjects reported voluntarily a shift of fixation, the trial was discarded. After each presentation subjects were asked to report if they had "sensed" the onset of the stimulus.

Since there is evidence that blindsight is spatiotemporally modulated with a peak of response at 0.5-1 c/deg and temporal frequency of 10 Hz [2,3,34] sinusoidal gratings enclosed in Gabor patches (8° in size, spatial frequency of the carrier: 1.25 c/deg, temporal frequency: 10 Hz) with contrast 98% and average luminance of 22.5 cd m-2 were presented on a dark background (luminance 0.13 cd m-2). The stimuli were displayed for 2 seconds in the superior or inferior quadrant of the blind hemifield, with the edge placed 20 deg from the vertical meridian intercepting the fixation point and 1.6 degrees respectively above or below the horizontal meridian.

To avoid the biasing effect of light scattering from the stimulus, the fixation point was surrounded by a white field (53 cd m<sup>2</sup>) extending 9.8 deg towards the blind region (figure 5) [2,34].

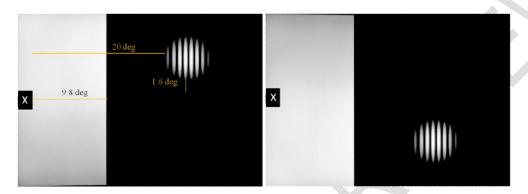


Fig. 5. The stimulation with the sinusoidal patch presented in the superior or inferior quadrant, deep inside the blind region.

Testing was monocular. Which eye (left or right) was examined depended on the side of the loss, being the one whose blind hemifield did not contain the blind spot (left eye in right hemianopia, right eye in left hemianopia). Fixation was constantly monitored via an infrared microcamera placed on a trial frame in front of the non-examined eye. The head of the patient was firmly placed on a chinrest 2.6 feet from the screen.

Each trial consisted of two presentations, one containing the Gabor patch and the other made of a blank stimulus (in randomized order). An acoustical signal (100 msec) preceded each presentation, separated by a time interval of 4 seconds. The position of the stimuli (upper or lower blind quadrant) was randomized. The ratio between the trials containing the signal in the first vs. second interval was 1.0; the same applied to the ratio between the trials with the signal in the upper vs. lower quadrant. The examination was split into two sessions, 64 trials per session (total: 128 trials), with a resting period of 10 min in between.

After each trial, the patient had to report verbally the interval containing the signal according to a 2AFC response model, temporal paradigm. The test was performed in a completely dark room, as recommended by Bender and Krieger [35]. At the end of the examination, the proportion of correct responses in the blind hemifield (superior quadrant, inferior quadrant, and cumulative responses) was computed. The procedure is available on request to the corresponding author. The authors hereby declare that the experiment has been examined and approved by the ethics committee as part of a bachelor's degree (ID nb 926006) and has therefore been conducted under the ethical standards laid down in the 1964 Declaration of Helsinki. Informed consent for publication was obtained from the subjects who underwent the experiment after the explanation of the aims of the study.

# 3. RESULTS

The procedure required on average 20 minutes to be completed, depending on the reaction time of the patient. Table 2 and figure 6 show the results in the four subjects. AF refused to continue the test at the end of the first phase because he felt tired. In DC, who had a superior quadrantanopia, the stimulation of the lower quadrant served as a control and yielded a hit rate of 100%.

Table 2.Proportion of correct responses in the four patients; cl: chance level.

Pat TOT Chi-squared UPPER QUADR Chi-squared LOWER QUADR Chi-squared

	prop		prop corr		prop corr	
	corr					
GO	49.22	$\chi^2 = 0.99$ , p: .31	53.13	$\chi^2 = 0.49$ , p: .48	45.31	cl
PB	51,56	$\chi^2 = 0.99$ , p: .31	53.13	$\chi^2 = 0.49$ , p: .48	50	cl
AF	45,31	cl	37.50	cl	53.13	$\chi^2 = 0.49, p: .48$
DC	60.94	$\chi^2 = 6.08$ , p: .013	60.94	$\chi^2 = 6.08$ , p: .013	=	-

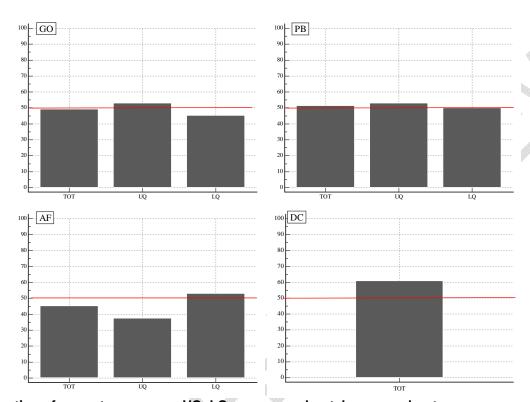


Fig. 6. The Proportion of correct responses. UQ, LQ: upper quadrant, lower quadrant.

A hit rate above chance was observed only in DC (60.94%, $\chi^2$ = 6.08, p: .013). In GO and PB the correct responses were not significantly different from chance irrespective of the location of the stimulus. The score of AF, lower than chance, suggests an incorrect comprehension of the task.

To further investigate whether the trend of responses in DC shows stationary behavior, the proportion of correct responses has been analyzed as a function of test progression. The total number of presentations has been subdivided into 13 subsequent blocks and the proportion of correct responses in each block has been computed. No change in the proportion of correct responses was observed as the examination went on, thereby confirming the stationarity of the performance.

## 3. DISCUSSION

After damage to the optic radiations or occipital infarction, a limited proportion of subjects may retain the ability to localize targets in their blind hemifield.

This ability can be revealed psychophysically with forced-choice response paradigms when patients are asked to guess about the presence of a stimulus in their blind region.

Since blindsight is spatiotemporally modulated, the optimal stimulus configuration should have a temporal frequency higher than 3.7 Hz and a spatial frequency below 8 c deg-1 whereas stimulus duration is irrelevant [2, 3, 21].

Blindsight is supposed to rely on an extrastriate visual pathway which projects from the lateral geniculate nucleus or the superior colliculus to the lateral occipital and temporal cortex. More specifically, blindsight can be mediated by pre-existing pathways that project directly to the extrastriate regions bypassing V1, like a geniculo-extrastriate pathway or a tecto-extrastriate pathway, or by novel connections like cross-projections descending from the extrastriate region toward the contralateral lateral geniculate nucleus. The role of the extrastriate cortex for unconscious visual processing remains to be ascertained. The high variability of this phenomenon, with a prevalence ranging from 5%[36] to 64% [37], depends on the criterion adopted for diagnosing this condition, the size and localization of the lesion, and the different patterns of plastic

neuronal reorganization [18,24]. Of the four patients examined, only one, DC, showed an experimental behavior suggestive of blindsight, thus warranting further investigation. Due to the stimulus parameters and experimental conditions (size, luminance, position in the blind hemifield, and background luminance), the effect of light scattering on the AFC response in this patient can be ruled out [2]. Contrary to the others, who suffered from lesions involving the whole right or left posterior cerebral region, the brain injury of DC was more localized and anterior so that the lateral portion of the occipital cortex, corresponding to the extrastriate areas, was completely spared. This finding complies with the notion that a preserved extrastriate function is necessary for blindsight. The group of Morland [38], for example, tested motion discrimination in 7 hemianopic patients with lesions involving or sparing the lateral occipital (extrastriate) areas. They found that a spared lateral (extrastriate) occipital cortex is necessary for motion discrimination, as documented in two patients of the recruited sample. In these subjects, indeed, extrastriate activity was observed at fMRI during the stimulation. Similar results were reported by Barton and Sharpe [39], who used random dots instead of sinusoidal gratings to measure motion direction discrimination. However, since in our study not only DC but also the other patients retained their extrastriate function and yet did not perform above chance, this condition is presumably not sufficient. It should be considered that our experiment differs from the abovementioned ones as static rather than dynamic stimuli were presented in the blind region.

An interesting question is whether blindsight shows a positional bias, in other terms if it is more evident in the superior quadrant compared to the ipsilateral inferior quadrant or vice versa. In GO and PB the proportion of correct responses was slightly higher in the superior quadrant, that is in the affected quadrant of DC, where, as reported, a hit rate above chance has been recorded. In this patient the comparison of the performance between the superior and inferior quadrants was not possible as the sensibility in the inferior quadrant was normal. A more consistent amount of data and a larger sample are needed to assess whether blindsight shows an anisotropic behavior across the visual field.

This study suffers from some limitations. As suggested by a reviewer, in DC the bottom edge of the stimulus may have been slightly visible to the participant: the 30-2 program at standard automated perimetry has a resolution of 6 deg, and as such the first point tested above the horizontal meridian falls at 3 deg. The bottom edge of the Gabor patch extends up to 1.6deg from the meridian. We were comforted by the fact that trial after trial DC never reported the slightest sensation of the presence of the stimulus, but this is not enough to rule out the possibility of an involuntary minimal stimulation at the extreme superior boundary of the lower quadrant. In the next studies we plan to employ additional testing, for example the high-resolution perimetry (HRT) devised by Kasten and associates [40] and/or use smaller stimuli.

When testing for blindsight, stimuli are displayed on a background with a certain level of luminance, be it the conventional background luminance used during perimetric examinations or a pre-defined value when stimuli are displayed on a screen. In this experiment we opted for a dark background instead of a light one: a recent study showed that a dark background helps detection via pupil dilation compared to a clear pedestal [41]. We understand this choice does not mitigate the biasing effect of light scattering, but, on the contrary, tends to favor it. It should be considered, however, that the spared hemifield in our experiment was flooded with a bright background exactly to avoid this unwanted effect. Patients, especially DC who showed above chance rate of correct responses, never reported awareness of the stimulations. Repeating the experiment using an isoluminant background is necessary to confirm our result.

The use of a completely dark room for testing may increase the risk of light scatter. The use of a completely dark room was originally recommended by Bender and Krieger [35] so we followed their indication. We understand that the subsequent research may have questioned this experimental condition. Again, we aim to repeat the test in a dimly illuminated room to verify our results

The stability of fixation has been checked with a camera monitored in real-time by a second operator, in line with other studies on blindsight [42, 43, 44, 45]. This is not the most suitable and rigorous solution and the use of an eye tracker would be desirable. However, we aimed to provide a tentative indication of the presence of blindsight in patients with hemianopia within the clinical setting, where sophisticated procedures of eye tracking are hardly available, and by no means to discriminate this condition with the highest sensibility and specificity as possible, as it happens in strictly controlled experimental conditions.

A higher number of trials could increase the proportion of correct responses of DC, thereby statistical significance. However, as a first attempt the number of presentations adopted in this preliminary experiment seems to us a good trade-off between efficiency and examination time. It should be noted that during the examination the performance in DC did not show any improvement as a function of the number of trials: in other terms the function in the short term is stationary. Yet, there is consistent evidence that blindsight can be trained [31, 32] so to improve the unconscious ca-pacity of hemianopic subjects who show this phenomenon to avoid obstacles and prevent collisions [30]. Indeed, from a translational perspective identifying patients with blindsight is for rehabilitative purposes. The group of Hyvärinen and Raninen [46,47], for example, showed that blindsight can be trained using flickering discs, then letters, presented on an equiluminant background at different temporal frequencies. In a previous study, Zihl and von Cramon presented pairs of spots of light. Patients had to fixate on a central red spot and report if they saw one or two stimuli after each presentation. At the end of the training, 96% of the patients who complained of severe to moderate problems in avoiding obstacles dropped to 49% and those with reading difficulties decreased from 58% to 22% [43].

#### 4. CONCLUSION

In conclusion, our preliminary results suggest that blindsight can be suspected within the clinical setting using simple, user-friendly, and fast psychophysical procedures. Targeting patients suspected to have this "hidden" function is the first step before proceeding with second-level, more rigorous examinations, with the final aim of devising customized visual rehabilitation programs.

#### CONSENT

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

## **ETHICAL APPROVAL**

All authors hereby declare that the experimenthas been examined and approved by the appropriate ethics committee as part of a bachelor's degree (ID nb 926006) and has therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

#### REFERENCES

- 1. Weiskrantz, L., Warrington, E.K., Sanders, M.D.,&Marshall, J. (1974). Visual capacity in the hemianopic field following a restricted occipital ablation. Brain, 97(4),709-728. doi: 10.1093/brain/97.1.709.
- 2. Barbur, J.L., Harlow, A.J., & Weiskrantz, L. (1994). Spatial and temporal response in a case of hemianopia. Philos Trans R Soc Lond B Biol Sci.343(1304).157–166. doi: 10.1098/rstb.1994.0018.
- 3. Sahraie, A., Weiskrantz, L., Trevethan, CT., Cruce, R.,& Murray, A.D. (2002). Psychophysical and pupillometric study of spatial channels of visual processing in blindsight. Exp Brain Res, 143(2),249–256. doi: 10.1007/s00221-001-0989-1.
- 4. Stoerig, P., & Cowey, A. (1992). Wavelength discrimination in blindsight. Brain;115(pt2),425–444.doi: 10.1093/brain/115.2.425.
- 5. Stoerig, P.,&Cowey, A. (1995). Visual perception and phenomenal consciousness. Behav Brain Res, 71(1-2), 147-156. doi: 10.1016/0166-4328(95)00050-x.
- 6. Boyer, J.L., Harrison, S., &Ro, T. (2005). Unconscious processing of orientation and color without primary visual cortex. Proc Natl Acad Sci USA, 102(46), 16875-16879. doi: 10.1073/pnas.0505332102.
- 7. Weiskrantz, L. (1986). Blindsight: a case study and implications. Oxford, Clarendon Press.
- Weiskrantz, L. (1987). Residual vision in a scotoma. A follow-up study of 'form' discrimination. Brain,110(pt 1),77-92. doi: 10.1093/brain/110.1.77.
- 9. Barbur, J.L., Ruddock, K.H.,&Waterfield, V.A. (1980). Human visual responses in the absence of the geniculo-calcarine projection. Brain,103(4),905–928. doi: 10.1093/brain/103.4.905.
- 10. Ptito, A., Lepore, F., Ptito, M., &Lassonde, M. (1991). Target detection and movement discrimination in the blind field of hemispherectomized patients. Brain, 114, 497–512. doi: 10.1093/brain/114.1.497.
- 11. Perenin, M.T. (1991). Discrimination of motion direction in perimetrically blind fields. Neuroreport, 2(7), 397–400. doi: 10.1097/00001756-199107000-00011.
- 12. Campion, J., Latto, R.,&Smith, Y.M. (1983). Is blindsight an effect of scattered light, spared cortex and near-threshold vision? The Behavioral and Brain Sciences,6(3), 423–486. doi.org/10.1017/S0140525X00016861.
- 13. Celesia, G.G., Bushnell, D., Toleikis, S.C., & Brigell, M.G. (1991). Cortical blindness and residual vision: Is the second visual system in humans capable of more than rudimentary visual perception? Neurology, 41(6), 862–869. doi: 10.1212/wnl.41.6.862.

- Fendrich, R., Wessinger, C.M.,&Gazzaniga, M.S. 1992). Residual vision in a scotoma: Implications for blindsight. Science.258(5087),1489-1491.doi: 10.1126/science.1439839.
- 15. Wessinger, C.M., Fendrich, R.,&Gazzaniga, M.S. (1997). Islands of residual vision in hemianopic patients. J CognNeurosci,9(2),203–221. doi: 10.1162/jocn.1997.9.2.203.
- 16. Wessinger, C.M., Fendrich, R., &Gazzaniga, M.S. (1999). Variability of residual vision in hemianopic subjects. Restor Neurol Neurosci, 15(2-3), 243–253.
- Lutzemberger, L., Marzi, C.A.,&Tassinari, G. (1985). On inferring blindsight from normal vision. BBS, 8(4),754-755. doi.org/10.1017/S0140525X00046112.
- 18. Kentridge, R.W., Heywood, C.A.,&Weiskrantz, L. (1997). Residual vision in multiple retinal locations within a scotoma: Implications for blindsight. J CognNeurosci, 9(2),191-202. doi: 10.1162/jocn.1997.9.2.191.
- 19. Stoerig, P., Kleinschmidt, A.,&Frahm, J. (1998). No visual responses in denervated V1: high-resolution functional magnetic resonance imaging of a blindsight patient. Neuroreport,9(1),21-25. doi: 10.1097/00001756-199801050-00005.
- Barbur, J.L., Watson, J.D., &Frackowiak, R.S., Zeki, S. (1993). Conscious visual perception without V1. Brain,116(pt 6),1293-1302. doi: 10.1093/brain/116.6. 1293.
- 21. Weiskrantz L., Harlow A., &Barbur L. (1991). Factors affecting visual sensitivity in a hemianopic subject. Brain, 114:2269-82.
- Yukie, M., &lwai, E. (1981). Direct projection from the dorsal lateral geniculate nucleus to the prestriate cortex in macaque monkeys. J Comp Neurol, 201(1), 81-97. doi: 10.1002/cne.902010107.
- 23. Bridge, H., Thomas, O., Jbabdi, S., & Cowey, A. (2008). Changes in connectivity after visual cortical brain damage underlie altered visual function. Brain, 131(Pt 6), 1433-1444. doi: 10.1093/brain/awn063.
- 24. Ro, T., Shelton, D., Lee, OL., & Chang E. (2004). Extrageniculate mediation of unconscious vision in transcranial magnetic stimulation-induced blindsight. PNAS,101(26), 9933-9935.doi: 10.1073/pnas.0403061101.
- 25. Fries, W. (1981). The projection from the lateral geniculate nucleus to the prestriate cortex of the macaque monkey. Proc R Soc Lond B Biol Sci, 213(1190),73-86. doi: 10.1098/rspb.1981.0054.
- 26. Schmid, MC., Mrowka, S.W., Turchi, J., Saunders, R.C., Wilke, M., Peters, A.J., et al. (2010). Blindsight depends on the lateral geniculate nucleus. Nature, 466:373-7.
- 27. Standage, G.P., &Benevento, L.A. (1983). The organization of connections between the pulvinar and visual area MT in the macaque monkey. Brain Res; 262:288-94.
- 28. 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; 10:339-46. Doi: 10.1006/nimg.1999.0474.
- 29. Silvanto, J.; Walsh, V.; &Cowey, A. (2009). Abnormal functional connectivity between ipsilesional V5/MT+ and contralesional striate cortex (V1) in blindsight. Exp Brain Res. 2009; 193:645-50.
- 30. Striemer, C.L., Chapman, C.S., &Goodale, M.A. (2009). "Real-time" obstacle avoidance in the absence of primary visual cortex. Proc Natl Acad Sci USA,106(37),15996-16001. doi: 10.1073/pnas.0905549106.
- Sahraie, A., Trevethan, C.T., MacLeod, M.J., Murray, A.D., Olson, J.A., & Weiskrantz, L. (2006). Increased sensitivity after repeated stimulation of residual spatial channels in blindsight. Proc. Natl Acad. Sci. USA, 103(40), 14971–14976. doi: 10.1073/pnas.0607073103.
- 32. Chokron, S., Perez, C., Obadia, M., Gaudry, I., Laloum, L., & Gout, O. (2008). From blindsight to sight: cognitive rehabilitation of visual field defects. Restor Neurol Neurosci, 26(4-5), 305-320.
- 33. Azzopardi, P., &Cowey, A. (2001). Motion discrimination in cortically blind patients. Brain, 124:30-46.
- 34. Sahraie, A., Trevethan, C.T., Weiskrantz, L., Olson, J., MacLeod, M., Murray, A., et al. (2003). Spatial channels of visual processing in cortical blindness. Eur J Neurosci, 18(5),1189 –1196. doi: 10.1046/j.1460-9568.2003.02853.x.
- 35. Bender, M.B., & Kreiger, H.P. (1951). Visual function in perimetrically blind fields. AMA Arch NeurPsych,65(1), 72–79. doi: 10.1001/archneurpsyc.1951.02320010078009.

- 36. Marzi, C.A., Tassinari, G., Aglioti, S., &Lutzemberg, L.(1986)Spatial summation across the vertical meridian in hemianopics: a test of blindsight. Neuropsychologia, 24(6),749-758. doi: 10.1016/0028-3932(86)90074-6.
- 37. Weiskrantz, L. Varieties of residual experience. (1980). Q J Exp Psychol, 32(3), 365-386. doi: 10.1080/14640748008401832.
- 38. Morland, A.B., Le, S., Carrol, E., et al. (2004). The role of spared calcarine cortex and lateral occipital cortex in the responses of human hemianopes to visual motion. J Cogn Neurosci,16(2),204–218. doi: 10.1162/089892904322984517.
- Barton, J.J.S., &Sharpe, J.A. (1997). Motion direction discrimination in blind hemifields. Ann Neurol, 41(2), 255-264. doi: 10.1002/ana.410410217.
- 40. Kasten, E., Strasburger, H., &Sabel, B. A. (1997). Programs for diagnosis and therapy of visual field deficits in vision rehabilitation. Spatial Vision, 10: 499–503.
- 41. Mathot, S., &Ivanov, Y. (2019). The effect of pupil size and peripheral brightness on detection and discrimination performance. Peer J, Dec 19;7:e8220. doi: 10.7717/peerj.8220.
- 42.Zihl, J. (1981). Recovery of visual functions in patients with cerebral blindness. Effects of specific practice with saccadic localization. Experimental Brain Research, 44, 159–169.
- 43.Zihl, J.,&von Cramon, D. (1985). Visual field recovery from scotoma in patients with postgeniculate damage. A review of 55cases. Brain, 108: 335–365.
- 44. Widdig, W., Pleger, B., Rommel, O., Malin, JP.,&Tegenthoff, M. (2003). Repetitive visual stimulation: a neuropsychological approach to the treatment of cortical blindness. NeuroRehabilitation, 18:227-237.
- 45. Huxlin, K.R., Riley, M.E., Martin, T., Kelly, K.N., Friedman, D.I., Burgin, W.S., et al. (2009). Perceptual relearning of complex visual motion after V1 damage in humans. Journal of Neuroscience, 29: 3981–3991.
- 46. Hyvärinen, L., Raninen, A.N.,&Näsänen, RE. (2002). Vision rehabilitation in homonymous hemianopia. Neuro-Ophthalmology, 27:97-102.
- 47. Raninen, A.V., Vanni, S., Hyvärinen, L.,&Näsänen, RE.(2007). Temporal sensitivity in a hemianopic visual field can be improved by long-term training using flicker stimulation. J Neurol Neurosurg Psychiatry, 78,66–73.doi: 10.1136/jnnp.2006.099366.