Chapter-5.

CORTICAL BLINDNESS AND RESIDUAL VISUAL CAPACITIES

Dr Partha Chowdhury, Dr Brinda Shah

ABSTRACT

Cortical blindness is a severe neurological condition caused by bilateral damage to the primary visual cortex, resulting in complete loss of conscious visual perception. Interestingly, many individuals retain a surprising degree of residual visual processing, a phenomenon known as blindsight. This challenges the traditional hierarchical model of vision and highlights the importance of alternative subcortical and extrastriate pathways that bypass the striate cortex. Blindsight extends beyond simple detection and may include motion perception, spatial localization, orientation discrimination, and emotional responses to visually salient stimuli. Clinically, cortical blindness presents significant diagnostic and rehabilitative challenges, particularly when associated with denial of visual loss, as seen in Anton's syndrome. Emerging approaches such as visual retraining, virtual reality–based rehabilitation, and non-invasive brain stimulation aim to enhance recovery. Overall, this condition provides a powerful model for understanding the dissociation between visual processing and conscious awareness.

Keywords: Cortical blindness; Blindsight; Visual cortex; Neurorehabilitation

5.1 INTRODUCTION

The human visual system is among the most complex neural architectures, involving parallel pathways, specialized cortical hubs, and dynamic feedback loops. Damage to the primary visual cortex (V1), particularly when bilateral, disrupts conscious visual perception, giving rise to cortical blindness (CB). Distinct from ocular blindness, where the impairment lies in the eyes or optic nerve, cortical blindness results from cortical disconnection: the optic nerve and retina remain structurally intact, yet the cortical substrate essential for conscious vision is compromised (Weiskrantz, 2009).

For decades, cortical blindness was regarded as a complete condition with no possibility of preserved function. Patients appeared fully blind, unable to consciously perceive or respond to visual stimuli. However, revolutionary work in the late 20th century demonstrated that many patients, when carefully tested, could still detect or discriminate stimuli presented within their blind visual fields despite reporting no awareness of having seen anything (Weiskrantz, 2009). This dissociation between awareness and performance gave rise to the concept of *blindsight* and has since transformed us understanding of vision from a unidirectional, serial process into a parallel, distributed, and redundant network. It thus provides not only a clinical challenge but also a powerful model for investigating the neural correlates of consciousness (Cowey, 2010).

5.2 NEUROBIOLOGICAL BASIS

The neurobiology of cortical blindness is fundamentally linked to the disruption of the geniculostriate pathway, the canonical route by which visual information travels from the retina to the lateral geniculate nucleus (LGN) of the thalamus and subsequently to V1. Destruction of V1 interrupts this stream, rendering patients cortically blind. Yet, other circuits remain operational and capable of mediating unconscious vision (Leh et al., 2006). One of the most significant alternative pathways is the retinotectal pathway, in which retinal ganglion cells project directly to the superior colliculus. From there, visual information is relayed to the pulvinar nucleus of the thalamus and onward to extrastriata

visual areas such as area MT/V5, which is specialized for motion detection (Pitto & Leh, 2007). This pathway helps explain why patients with CB may accurately guess the direction of a moving stimulus or localize a target without awareness (de Gelder et al., 2008). Another bypass mechanism includes a subset of LGN neurons that project directly to extrastriata cortices, avoiding V1 entirely, allowing for limited form and orientation processing (Bridge et al., 2010). Moreover, the pulvinar-amygdala network enables rapid, unconscious responses to emotionally salient stimuli like threatening faces. This provides a substrate for preserved affective responses, even when subjects deny any conscious perception of the triggering stimulus (Tamietto & de Gelder, 2010). Beyond these anatomical pathways, neuroplasticity plays a crucial role in shaping residual vision. Following damage, the brain engages compensatory mechanisms like cross-modal plasticity, where somatosensory or auditory cortices recruit occipital regions. This reorganization can improve spatial navigation and environmental awareness, further underscoring the scope of non-conscious processing in CB (Pascual-Leone et al., 2005; Merabet et al., 2005).

4.3 CLINICAL MANIFESTATIONS

Cortical blindness presents with a range of clinical manifestations that extend beyond the mere absence of vision. The defining feature is a complete loss of conscious visual perception, typically encompassing both hemifields due to bilateral occipital lobe damage. Patients often report an inability to perceive light, shapes, or movement, despite intact pupillary light reflexes. This dissociation highlights the distinction between ocular function and cortical processing, emphasizing that blindness in CB arises from cortical dysfunction rather than optic nerve or retinal pathology (Bergsma & van der Wildt, 2008). A primary striking clinical phenomenon is blindsight residual visual capacity expressed without conscious awareness. Patients reliably deny seeing stimuli yet are able to perform visual tasks at accuracy levels far above chance. These tasks may include localizing a light source, detecting motion, or discriminating orientation (Sahraie et al., 2006). For example, a patient unable to verbally report the presence of a moving object may nevertheless orient their eyes or head toward it. Correspondingly, they may navigate around obstacles in an unfamiliar environment without recognizing having seen them (de Gelder et al., 2008). Such dissociations form the cornerstone of our understanding that unconscious vision can persist despite cortical destruction. Beyond basic visuospatial functions, patients with CB may retain affective and emotional reactivity to visual input. Studies show that fearful or threatening stimuli presented in the blind visual field can produce autonomic responses, such as increased heart rate or galvanic skin conductance, despite the patient's lack of conscious recognition. Functional neuroimaging shows activation of the amygdala and pulvinar in such scenarios, demonstrating preserved pathways for affective visual processing (Tamietto & de Gelder, 2010). This highlights that cortical blindness does not equate to a complete absence of visual influence on behaviour but rather a loss of conscious access to visual signals. Another distinctive presentation is Anton's syndrome, characterized by cortical blindness associated with denial of blindness (anosognosia) and confabulation. Patients insist they can see, often providing fictitious descriptions of their surroundings. This condition illustrates the brain's attempt to generate a coherent narrative in the absence of visual input, further emphasizing the disconnection between sensory processing and conscious awareness.

4.4 DIAGNOSTIC APPROACHES

The diagnosis of cortical blindness requires careful neuroimaging and clinical assessment to distinguish it from ocular and optic nerve pathologies. Standard visual field evaluations often reveal total deficits, but these alone cannot capture residual capacities. More nuanced psychophysical paradigms are required. Forced-choice guessing tasks, in which patients are asked to guess the direction, location, or presence of stimuli, often reveal performance significantly above chance, revealing blindsight (Bergsma & van der Wildt, 2008). Eye tracking provides another window into covert processing: patients may generate accurate saccades towards unseen targets. Functional neuroimaging, including fMRI, reliably demonstrates activation of extrastriata visual areas during visual tasks, even in the absence of V1 activity (Bridge et al., 2010). Diffusion tensor imaging further shows preserved connectivity between the pulvinar, LGN, and extrastriata cortices, supporting the anatomical basis of blindsight. Together, these diagnostic tools provide evidence not only of blindness but also of residual visual processing, informing therapeutic decision-making.

4.5 Rehabilitation and Residual Capacities

Historically, rehabilitation for cortical blindness depended primarily on compensatory approaches aimed at teaching patients to consciously redirect gaze and enhance other sensory inputs. The main approach is scanning training, where patients are trained to make systematic saccadic eye movements

into their blind field. This improves environmental awareness, safer mobility, and reading speed by encouraging exploration beyond the intact hemifield. While these approaches do not restore vision *per se*, they can significantly reduce functional disability and improve quality of life. Informed by the detection of residual capacities like blindsight, modern rehabilitation has shifted towards vision restitution therapies that aim to stimulate and strengthen spared neural pathways. These therapies are based on the principle of perceptual learning, wherein repeated stimulation of blind field regions improves sensitivity through cortical plasticity (Huxlin et al., 2009; Sahraie et al., 2006).

For example, presenting stimuli near the blind-intact field border can expand visual detection zones over time. Controlled trials show modest but clinically meaningful recovery, with improvements in detection thresholds and reaction times (Cavanaugh & Huxlin, 2017). This emphasizes that the damaged visual system is not functionally silent but capable of reorganization under systematic training. A stimulating frontier is the deliberate use of blindsight as a therapeutic target. Training protocols encourage patients to respond to visual stimuli within their blind field using forced-choice tasks, even when they deny conscious perception. Over time, some patients report a gradual feeling of visual presence, signifying a partial transition from non-conscious to conscious vision. This has led to the concept of "blindsight to sight" rehabilitation, where structured practice strengthens alternate pathways, potentially restoring fragments of awareness. Neuroplasticity shows a central role in functional adaptation post-CB.

Evidence from neuroimaging shows that auditory and somatosensory cortices can recruit occipital regions in the absence of visual input, facilitating improved non-visual perception (Merabet et al., 2005). Rehabilitation approaches increasingly exploit this plasticity by designing multisensory integration tasks, like pairing auditory cues with visual field stimulation. This reorganization indicates that rehabilitation should not focus only on vision restitution but also on enhancing compensatory neural adaptations across sensory systems.

Latest developments in digital technology have transformed therapeutic approaches. Virtual reality platforms allow for immersive and controlled stimulation of the blind field while concurrently engaging multisensory feedback. Eye-tracking-integrated systems provide real-time feedback on scanning strategies. Moreover, non-invasive brain stimulation techniques like transcranial magnetic stimulation (TMS) are being explored to potentiate neuroplasticity and enhance the effects of perceptual training.

4.6 FUTURE DIRECTIONS

While complete visual recovery remains rare, rehabilitation focused on residual capacities produces meaningful functional gains. Patients often achieve improved environmental awareness, better obstacle avoidance, and increased reading fluency. Equally important are psychological benefits: structured rehabilitation reduces helplessness, increases independence, and mitigates the risk of depression. Cortical blindness is both a profound clinical condition and a fertile ground for scientific discovery. Future research must refine our understanding of residual pathways and identify biomarkers predictive of rehabilitation outcomes. Integrating multimodal imaging, electrophysiology, and computational modelling will clarify how conscious and unconscious visual processing diverge (Ffytche & Zeki, 2011; Zeki & Ffytche, 1998). Therapeutically, innovation will increasingly combine digital technologies, adaptive learning algorithms, and neuromodulation to personalize rehabilitation. At a conceptual level, the study of cortical blindness will continue to illuminate the neural correlates of consciousness, probing the boundary between awareness and perception.

4.7 CONCLUSION

Cortical blindness, far from being vision loss, demonstrates the flexibility and complexity of the visual system. The perseverance of residence visual capacities shows the strength of parallel processing pathways and highlight the brain’s remarkable ability to adapt after injury. Clinically, these perceptions provide a foundation for advanced rehabilitation approaches aims at harnessing unconscious vision to improve functional outcomes. Scientifically, cortical blindness encounters our most fundamental assumptions about consciousness, perception and the architecture of the human brain. As both a model system and clinical syndrome, it continues to advance our understanding of how we see and what it means to be aware of seeing.

4.8 BIBLIOGRAPHY

1. Bridge H, Hicks SL, Xie J, Okell TW, Mannan S, Alexander I, Cowey A, Kennard C. Visual activation of extrastriate cortex in the absence of V1 activation. Neuropsychologia. 2010;48(13):4148–4154.

2. Cowey A. The blindsight saga. Exp Brain Res. 2010;200(1):3–24.
3. de Gelder B, Tamietto M, van Boxtel G, Goebel R, Sahraie A, van den Stock J, et al. Intact navigation skills after bilateral loss of striate cortex. Curr Biol. 2008;18(24):R1128–R1129.
4. Ffytche DH, Zeki S. The primary visual cortex, and feedback to it, are not necessary for conscious vision. Brain. 2011;134(Pt 1):247–257.
5. Huxlin KR, Martin T, Kelly K, Riley M, Friedman DI, Burgin WS, Hayhoe M. Perceptual relearning of complex visual motion after V1 damage in humans. J Neurosci. 2009;29(13):3981–3991.
6. Leh SE, Johansen-Berg H, Ptito A. Unconscious vision: new insights into the neuronal correlate of blindsight. Annu Rev Neurosci. 2006;29:195–212.
7. Pascual-Leone A, Amedi A, Fregni F, Merabet LB. The plastic human brain cortex. Annu Rev Neurosci. 2005;28:377–401.
8. Pitto M, Leh SE. Neural substrates of blindsight after hemispherectomy. Neuroscientist. 2007;13(5):506–518.
9. Sahraie A, Trevethan CT, MacLeod MJ, Murray AD, Olson JA, Weiskrantz L. Increased sensitivity after repeated stimulation of residual spatial channels in blindsight. Proc Natl Acad Sci U S A. 2006;103(40):14971–14976.
10. Tamietto M, de Gelder B. Neural bases of the non-conscious perception of emotional signals. Nat Rev Neurosci. 2010;11(10):697–709.
11. Weiskrantz L. Blindsight: A case study spanning 35 years and new developments. 2nd ed. Oxford: Oxford University Press; 2009.
12. Zeki S, ffytche DH. The Riddoch syndrome: insights into the neurobiology of conscious vision. Brain. 1998;121(1):25–45.
13. Merabet LB, Rizzo JF, Amedi A, Somers DC, Pascual-Leone A. What blindness can tell us about seeing again: merging neuroplasticity and neuroprosthetics. Nat Rev Neurosci. 2005;6(1):71–77.
14. Cavanaugh MR, Huxlin KR. Visual discrimination training improves Humphrey perimetry in chronic cortically induced blindness. Neurology. 2017;88(19):1856–1864.
15. Bergsma DP, van der Wildt GJ. Properties of the intact visual system in cerebral blindness. Exp Brain Res. 2008;191(1):53–66.