The Neuroanatomy Of Phenomenal Vision

Phenomenal vision is the sight of “colors, brightness, depth, shades, and motion”, generated by light distributed on the retina (Stoerig, 2001). Undeniably, there are phenomenally unconscious visual mechanisms occurring within an individual's retinal processes and occipital lobe activity does not correlate with visual awareness. Research into the study of vision and whether the phenomenal vision is mandatory to be aware of visual stimuli has extensively been researched in blindsight patients (Brogaard, 2011). Specifically, striate damage results in profound homonymous loss, resulting in cortical blindness. Extrastriate damage affects specific losses, color vision, and motion abnormalities. Subcortical damage involves processing impediments before signals reach the cortex (Wurtz and Albano, 1980). Notably, type 1 blindsight, reveals individuals are unaware of any stimuli but can predict aspects like location or movement of a stimulus. Type 2 blindsight holds some awareness but no visual perception (Fuchs, 1921). Blindsight occurs when damage is present to the primary V1; potentially resulting in blindness across the full visual field however, some are still capable of predicting the location and motion of visual stimuli. The primary visual pathway derives from the retina and projects to the occipital lobe via the lateral geniculate nucleus. Visual information reaches the extrastriate cortex along the ventral and the dorsal stream. (Braddick and Atkinson, 2011). Subcortical sites are associated and transmit direct projections to the extrastriate visual cortex. Damage to structures in the dorsal and ventral stream can impair visual perception while leaving some mechanisms intact (Diederich et al, 2014).

Gelder et al, 2008, demonstrated phenomenal vision is needless regarding the awareness of a visual stimulus. Brain imaging of a patient with bilateral damage to the primary V1 has suffered complete clinical blindness, a lack of functioning in the visual cortex, and a loss of posterior fibers however could successfully complete a ‘residual visual abilities test’. This demonstrates extra-striate pathways can sustain visuospatial, locomotion, and navigation skills in the absence of perceptual awareness. Conversely, Stoerig, 2001 states: absolute blindness is the absence of all visual qualia due to damage of visual structures and cortical areas that need to be activated in both veridical and non-veridical phenomenal vision. A unilateral lesion affects the visual field in one eye and both eyes if it is behind the chiasma (Mazzi et al, 2019). Patients who have experienced the destruction of visual regions are blind, do not see stimuli present in the blind field neither ‘after-images’. Magnetic brain imaging simulation provides support, demonstrating the association between primary V1 inactivity and visual blindness (Burton, 2003)

Arguably, Overgaard et al, 2008 improved methods for detecting awareness in blindsight patients report that visibility of stimulus correlates with accuracy in both healthy and blindsight individuals indicating blindsight is essentially a ‘highly degraded conscious vision’ confirming awareness of visual stimuli are still intact. She and Von Cramon, 1979 provided further clinical support. Those suffering from postchiasmatic visual field defects were trained using light-difference thresholds. Improvements in contrast sensitivity, visual acuity, color perception as well as visual field enlargement were obtained. Likewise, Balliet et al, 1985, undertook 89,000 visual field training trials for patients with homonymous, hemianopia or quadrantanopia caused by occipital lesions. Tests for light sensitivity demonstrated patients, did not have significant visual field differences when compared to controls as they reported no change in vision suggesting lesions do not necessarily result in a complete loss of function to the visual system.

Nevertheless, Sahraie et al, 2003 undertook research on trans-neuronal retrograde degeneration. Evidence portrayed that a lesion cause's lack of neuronal communication, therefore, cells anterior to the lesion degrade, and damage extending to the thalamus disrupts transmission from LGN through fibers, therefore, no residual visual ability is present. This indicates these elements are essential in visual awareness and damage to brain structure involved in visual processing can cause permanent visual impairment.

Contrariwise, Weiskrantz, 1986 'commentary key paradigms' provides support for awareness without phenomenal vision. Blindsight patients have revealed, discrimination is possible for certain visual events. They are not aware, however, to achieve this through a temporal guessing task. When patient's photosensitive retinas were stimulated, they could identify, localize, and distinguish targets when forced to guess whether, where, or which stimulus had been presented to their blind field. Kentridge, 1999 case study shows, patients reported awareness of salient stimuli presented to their blind field depending on stimulus contrast. Vague sensations of visual awareness were reported at '75% contrast' indicating phenomenal vision is meaningless regarding visual awareness. Huxlin et al, 2009 used moving, 'complex global dots' and 'simple local gratings' to test visual awareness in blindsight subjects. Additionally, initial blindsight monkey studies revealed pupil dilation in response to a motion supporting this concept (Humphrey, 1974). Although V1 damage impaired visual awareness, repetitive training improved direction discrimination. Nonetheless, Sahraie et al, 2010 conducted regular blind field stimulations for hemianopia patients including several sessions and thousands of trials. Findings indicated that both accuracy and awareness improved, again, providing support.

The study of phenomenal vision in the visually impaired has been a controversial subject. While numerous studies argue to have provided conclusive evidence for the presence of blind field qualia, others often reveal difficulties in interpreting results. From previously discussed literature, it is evident there is more empirical support regarding the idea: we do not need phenomenal vision in order to be visually aware. Many studies reveal, those who are visually impaired or experience blindsight still express awareness to visual stimuli, yet in the absence of phenomenal vision. Empirical evidence overrides the opposing argument, however, a limitation, is the use of case studies conducted on single patients. Findings cannot be generalized neither applied reliably unless replications have produced similar valid results. Conversely, some have undertaken numerous trials that overcome this. Future studies should be directed into examining, brain regions and actively involved in those with impaired vision. Furthermore, how the brain adapts via plasticity to compensate for V1 lesions. Furthermore, studying why there are incidents of phenomenal awareness even in the absence of the primary visual brain regions; especially as the visual cortex persists to unconsciously process visual information, would be an interesting future focus.