Editorial

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Occipital Lobe Epilepsy and Color Vision

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Introduction

The occipital lobe is the smallest of the four lobes of the cerebral hemisphere. It is present posterior to the parietal and temporal lobes, and thus it forms the caudal part of the brain. The paired occipital lobes are separated from each other by a cerebral fissure. The occipital lobe is primarily responsible for visual processing. It contains the primary and association visual cortex. The cerebral surface of the occipital lobe irregularly molds into eminences called gyri and is separated by depressions called sulci: the intra occipital sulcus, the transverse occipital sulcus, and the lateral occipital sulcus. The medial surface of the occipital lobe has a characteristic calcarine sulcus or calcarine fissure. It extends from the parieto-occipital sulcus to the occipital pole. The upper, and lower banks of calcarine sulcus house the primary visual cortex. Each primary visual cortex receives visual information from the controlateral half of the brain [1].

Brain Occipital Lobe and Vision

The occipital lobe is the visual processing area of the brain. It is associated with visual-spatial processing, distance and depth perception, color determination, object and face recognition, and memory formation.

The primary visual cortex, also known as V1 or Brodmann area 17, surrounds the calcarine sulcus on the occipital lobe's medial aspect. It receives the visual information from the retina via the thalamus. The secondary visual cortex, also known as V2, V3, V4, V5 or Brodmann areas 18 and 19, surrounds the primary cortex and receives information from it. The primary visual cortex transmits information through two pathways: the dorsal and the ventral stream. The dorsal stream is associated with object location and carries visual information to he parietal lobe. The ventral stream has associations with object recognition and transmits visual information to the temporal lobe [2].

Clinical Influence on Brain Occipital Lobe

Injury to the occipital lobe can occur due to vascular insults, neoplastic lesions, trauma, infections, and seizures. Depending on the type and location of the injury, specific neurological deficits can occur. Unilateral occipital lobe lesions causes "controlateral homonymous hemianopia". It is a visual field defect on the same side of both eyes controlateral to the site of the lesion, lesions of the occipital lobe due to the posterior cerebral artery infarct cause "homonymous hemianopia with macular sparing". Macular sparing is due to the dual blood supply of the occipital pole by middle and posterior cerebral arteries. Lesions of the posterior occipital lobe may cause "homonymous hemianopia with sparing of crescent-shaped temporal vision". The posterior lobe lesions spare the anterior striate cortex, which controls temporal vision. Bilateral occipital lesions cause "bilateral complete hemianopia", also known as "cortical blindness". "Anton syndrome" is sometimes present in patients with cortical blindness. It occurs in cases of insult to the occipital lobe. The patient persistently denies loss of vision and is unaware of the visual deficit, despite evidence of cortical blindness. Confabulation often accompanies this deficit [3].

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Another rare syndrome associated with occipital lobe injury is "Riddock syndrome". The subject is only able to see moving objects in the blind field, while non-moving objects are invisible. The subject has motion perception while unable to perceive shape or color [4].

"Occipital lobe epilepsy" is relatively uncommon but often presents with specific neurological findings. Seizures originating in the occipital lobe are associated with visual hallucinations, blurring or loss of vision, and rapid eye blinking or fluttering of eyelids. The seizures usually occur after a bright visual image or flicker stimulus [5]. Generally, lobe lesions can cause visual hallucinations, color agnosia, or agraphia.

Anton syndrome

Patients showing the black/white vision can have a compromise of the visual pathways from V1 to V4 in the middle brain. The very compromise of the primary visual area V1 does not allow that the visual stimulus can arrive to V4. The clinical evidence makes such a plausible suggestion because the integrity of both areas is critical to be consciously aware of having seen the colors. In these patients miss the intact return pathways from V4 to V1. Patients showing the color vision deficiency both on red/green, and/or on blue axis, can have no a great compromise of the V1 primary visive area. But, very likely both great or small compromise of the visive areas is related only to V4 color vision area. Within this group, the return pathways from V4 back to V1 showed to be critical for the conscious awareness of the color attributes of vision. The operational connections between the two areas are not very compromised, and it have restored after the surgical ventricular-peritoneal shunt [6].

Occipital Lobe Epilepsy

In an our recent investigation, we study a patient with diagnosed occipital lobe epilepsy, let him reading three color vision tests: Ishihara test, Farnsworh Dicotomous D15 test, the City University test (Figure 1).

1. **Ishihara Test**

Patient did not read any table, confounding him as an inherited colorblind subject.

2. **Farnsworth Dicotomous D15 test**

Patient made deutan, and tritan errors reading the tables.

3. **The City University Test**

Patient confirmed the deutan, and tritan errors reading the tables.

Thus, we confirmed in this preliminar work, as an impair color vision pathway placed within the occipital lobe can reflect the damage due to same pathological event in this brain area.

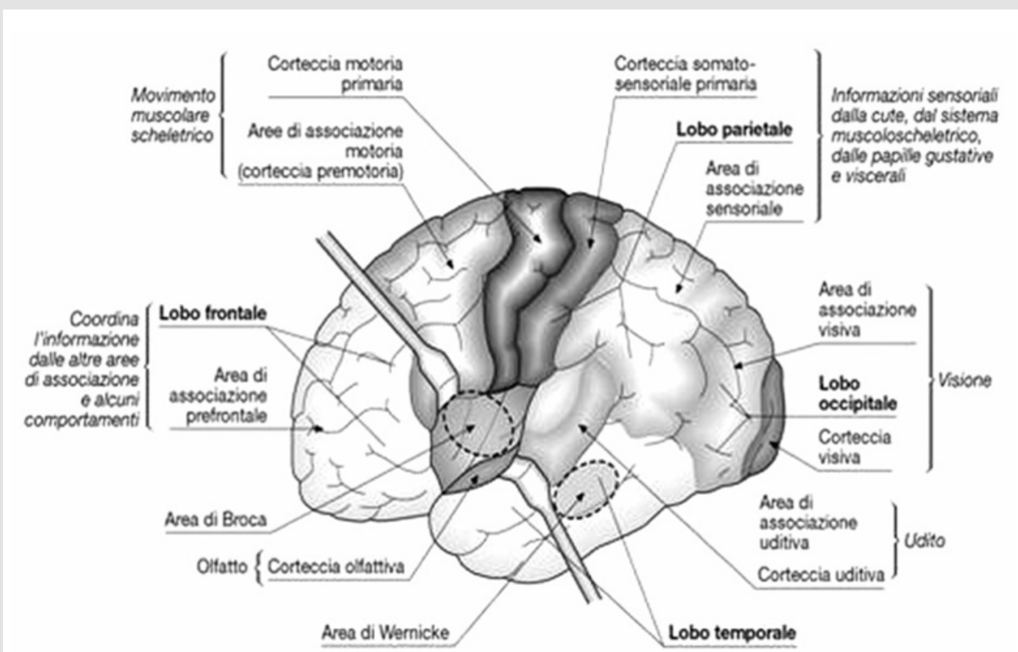

Figure 1.

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FONDAZIONE

CASSA DI RISPARMIO DI CALABRIA E DI LUCANIA

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