Editorial

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Neurosciences Reflect all the Brain Pathways in Normal and Pathological Manifestations

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Introduction

"Neurosciences must change the philosophy of mind, and to a great extent has already done so" [1]. Problem of vision is the problem of knowledge, knowledge about the external acquired through the sense of vision. One cannot unravel the first process, that of seeing, in any profound sense unless one unravels the second process, that of understanding what is seen, because there is no real division between the two. In other words, seeing is understanding, and color vision happens to be a perfectly good example of this. The study of color vision has thus been instrumental in modifying the views on the cerebral processes involved in vision. Indeed, it has provided us with powerful insight into brain function. Understanding the role of the cortex in color vision has therefore philosophical and epistemological implications which go beyond understanding the detailed physiological mechanisms underlying the perception of colors. The study of colors gives us a vision of how the visual cortex works. The study of the visual cortex in turns gives us a vision of how the brain works. The new insights into the role of the cerebral cortex in vision have not been obtained by studying color in isolation, but rather in relation to how the cerebral cortex handles other attributes of vision, such as form, motion and depth. To grasp this requires a fairly detailed, though not exhaustive, description of the anatomy and physiology of the visual pathways. Central to this description is the theory of functional specialization [2].

This theory supposes that different attributes of the visual scene are processed simultaneously, in parallel, but in anatomically separate parts of the visual cortex. The study of color vision of motion provided the cornerstone on which the theory of functional specialization in the visual cortex is based and have thus us some insights into how the brain is organized to acquire its knowledge of the visual world. The determination of the color of every point in the field of view by the retina, the transmission of that color impression to the cortical retina in the well documented point by point system connecting the two structures, and the fact that a small lesion anywhere along this pathway led to a total blindness for a small part of the field of view (a scotoma) were all strong arguments in favor of this simple analytic doctrine of vision, including color vision, or so it seemed at the time.

Anatomical, Clinical Aspects of the Brain Pathway in Acquired Color Vision

The retina can also be subdivided into central and peripheral portions; the central retina is that part of it with which one fixates and sees detail. Structurally, it consists of a highlight sensitive region, the foveolar, which lies at the center of the foveal pit. The foveolar contains only the receptors for daylight vision, the cones; it is consequently known as the rod-free area of the retina, the rods being the receptors which are active at night in low levels of illumination. The fibers of the optic nerve, which carry the impulses from the retina, cross over at the optic chiasm in a very specific way. Beyond the optic chiasm the visual pathway becomes known as the optic tract. This relays visual signals to a subdivision on the subcortical thalamus entitled the lateral geniculate nucleus. Nerve impulses signaling color formation are relayed from the lateral geniculate nucleus via the visual radiations to the main visual areas of the brain, the striate cortex. The principal zones were designated area 17 by Brodman [3]. The axons of the lateral geniculate nucleus travel until the striate cortex, at the end; and each part of the retina, according with a topographical map, is represented in a given part of the primary visual cortex [4], and circumscribed cortical lesions can cause partial and selective visual deficits. The cortical color processing system brain consists of several stages extending from V1 to V4, partly directly but mainly through V2 [5] and beyond that to the inferior temporal cortex [1,2]. Lesion restricted to V1 lead to a specific loss of conscious color vision.

An Example of Color Vision Brain Pathway Loss: Epilepsy

Consciousness is lost when the function of both cerebral hemispheres or the brainstem reticular activating system is compromised. Episodic dysfunction of these anatomic regions presents transient, and often recurrent loss of consciousness. Seizures are disorders characterized by temporary neurologic signs or symptoms resulting from abnormal, paroxysmal, and hypersynchronous electrical neuronal activity in the cerebral cortex.

Epilepsy, a group of disorders characterized by recurrent, is a common cause of episodic loss of consciousness. Seizures can result from either primary Central Nervous System dysfunction or an underlying metabolic derangement or systemic disease. This distinction is critical, because therapy must be directed at the underlying disorder as well as at seizure control [6].

We investigated 46 epileptic patients, in total:

- 12 patients with temporal lobe epilepsy, 6 out of these showing an impair color vision and 6 out of these showing normal color vision;
- 30 patients with focal epilepsy, 12 out of these showing impair color vision, and 18 out of these showing normal color vision;
- 2 patients with secondary epilepsy, all showing impair color vision;
- 2 patients with congenital epilepsy, all showing normal color vision.

Epileptic patients who suffer from the above kinds of epilepsy show the normal color vision, showed for the most brain gaps in the white matter, to Magnetic Resonance Imaging. Moreover, diffuse subarachnoid large spaces and bad hippocampal rotation are present. All the frontal and parietal punctiform images are not present in occipital brain area.

Discussion and Conclusion

Epilepsy is a symptom of many diseases. Before a diagnosis of idiopathic epilepsy can be made primary metabolic disorders or intracranial lesions must be excluded. A diagnosis can usually be made after a careful history and examination, but simple outpatient investigations such as electroencephalography, radiographs of the skull, and biochemical tests are often helpful. Occasionally full inpatient studies are necessary. If there is an underlying metabolic disorder or intracranial lesion, such a tumor, the primary condition must be treated. In the management of epilepsy there are two major considerations. The first is the control of the seizures. Drug therapy should aim to prevent the attacks without producing disabling unwanted effects. Second is the social care of the of the patient, for children education may be difficult, and for adults there are often problems with employment and personal life [7]. In the normal brain, dentate granule cells block seizure from entorhinal cortex to the hippocampus. A hypothesis is that granule cell dispersion may disrupt the normal massy fiber pathway connecting granule cells CA3 pyramidal cells leading to massy fiber sprouting and new excitatory networks capable of generating seizures [8]. Cortical dysplasia is a brain malformation may cause abnormal cortical layers (dyslamination), occur with abnormal neurons (dysmorphic neurons, ballon cells) and may occur with a brain tumor or vascular malformation [6]. It is very pleasure for us to insert a beautiful sentence by Sir William Turner [9].

Just as it is desirable before studying the geology of a district to have the surface carefully surveyed and accurately mapped out, so it is advisable that the topography of the convolutions of the human cerebrum should be satisfactorily ascertained before an analysis of the intimate structure and deep connexons of the gray matter can be put forth with the necessary exactness.

Conflicts of Interest

All the Authors declare no Conflicts of Interest.

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FONDAZIONE

CASSA DI RISPARMIO DI CALABRIA E DI LUCANIA

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