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Color Vision as a Biological and Prognostic Marker in Ophthalmology, Along with Related Neurological Disorders

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Abstract

Color Vision resulted to be a useful biological marker asserting the course and developing the most part of neurological disorders. The analysis of the impairment pathway of color vision from V1, brain primary visive area, to V4, brain color vision area allows us to better understand the impar brain pathways implied in different neurological disorders.

Keywords: Color Vision; Neurological Disorders; Biological Marker; Prognostic Marker; Normal Pressure Hydrocephalus; Epilepsy Kinds; Neurophysiology

Introduction

Neurosciences must change the philosophy of mind, and to a great extent has already done so [1]. The problem of vision is the problem of knowledge, knowledge about the external world 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 it 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.

Color Vision: Neurophysiology from the Eye to the Brain

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 insights into brain function. Understanding the role of the cortex in color vision has therefore philosophical and epistemological implications which go far 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 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 has provided the cornerstone on which the theory of functional specialization in the visual cortex is based and has thus us some insights into how the brain is organized.

Light traverses the neural layers of the retina to the photoreceptors which are in the innermost region (posterior) and is adsorbed by the photo-pigments contained within the receptors; subsequently a neural discharge is initiated by which the signal is transferred to the brain. The retina has been shown to play a major role in separating out the colored elements of the stimulus from brightness characteristics and coding these two attributes. Little is known about the mechanisms converting the photochemical change in the sensory receptors to an electrical potential. It involves complex internal chemical reactions in which changes in potassium and sodium play a major part. Pharmacological agents known as neurotransmitters act to carry the coded signal form one neuron to the next as they are released form neurons at the synaptic junction. Nerve impulses signaling

color information are relayed from the lateral geniculate nucleus via visual radiation, to the main visual areas of the brain, the striate cortex. The principal zones were designated Area 17 by Brodman in his classical division, and then onwards to Area 18 (the occipital or para-striate cortex) and Area 19 (the pre-occipital cortex).

Some not color fibers concerned with eye movements project to the superior colliculus and pre-tecta regions. The visual areas maintain strong focal representation having specific location within the region for each part of the visual field. Retinal organization or spatial mapping is thus preserved and fibers conveying signals from corresponding points of the two retinal remain close. Foveal regions are spatially magnified in the lateral geniculate nucleus and striate cortex to ensure maximum resolution in this region [2-4]. 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 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 (scotoma), were all strong arguments in favor of this simple analytic doctrine of vision, including color vision, or so it seemed at the time.

Acquired Color Vision Impairment

There exists a wide group of color vision disturbances which are acquired during life, predominantly the result of ocular or general disease. Color vision changes provide a valuable means of monitoring the progress of a disease, or the toxic effects of a chemical substance, whether exposure is deliberate for therapeutic purpose or unintentional in the case of an industrial hazard. The effectiveness of treatment can be assessed by the continued monitoring of recovery of an acquired color vision disturbance. Renewed interest has thus been concentrated around the use of color vision as a diagnostic tool although there have been differing estimates of the efficiency of color vision test as the earliest index of malfunction [5-7]. Many pathological conditions involving damage to the nervous system give acquired color vision impairment progressing from normal trichromatism to anomalous trichromatism on to a dichromatic stage and even to monochromatism. Which are the characteristics of the acquired color vision than that inherited? They are, differences in color perception between eyes; color loss may be accompanied by deficiencies in other visual areas; disturbances of blue-green-yellow vision are common; females are affected in the same proportion as men; the elderly are particularly susceptible; above all, severity of the defect is variable according to the progression of disease and the disease's degree, and it can be showed a transient chromatopsia; colors can often identified correctly, and color perception improved when all the external conditions are improved; an acquired defect may be superimposed on an inherited defect.

Patients

During the studies, an Ophthalmologist examined all patients and controls in order to rule out diabetic retinopathy, cataracts, senile maculopathy, or ocular fundus anomalies. Fixed sampling of males allows to avoid the genetic Lyon phenomenon [8], which is present only in the heterozygous females for X- linked diseases such as colorblindness (the inherited red-green color vision deficiency). The acquired color vision deficiency is real in the male cohort because they have not the compensation presence by second X chromosome.

Clinical Tests for Color Vision Identification in Research

There are a number of clinical color vision tests which aim to identify, classify and grade the severity of color deficiency and are designed to determine occupational suitability. Screening tests identify people with normal or abnormal color vision. Grading tests estimate the severity of color deficiency. Some tests have both screening and grading functions. Most tests aim to classify protan, deutan and tritan color deficiency. The efficiency of most tests in current use has been established in clinical trials with previously diagnosed normal and color deficient observers.

In our research, all patients and controls underwent the following test: Ishihara test [9] which is the most reliable among the pseudo-isochromatic tests to identify inherited colorblind subjects. Patients who made more than five errors reading the first 17 plates were diagnosed as being colorblind. The type of colorblindness was determined by reading the last four tables. Farnsworth Dichotomous D-15 test [10] identified both the greatness of color vision deficiency by the high number of errors (maximum value, n. 15) and the type (deutan, protan, tritan). The City University test [11] identified (both binocularly and monocularly) people with different types and degrees of the acquired red-green deficiency (maximum value of errors number, n. 6) and blue-yellow color vision deficiency (maximum of errors number, n. 3).

Normal Pressure Hydrocephalus Neurophysiology

Normal Pressure Hydrocephalus (NPH) is a neurological disease characterized by an expansion of the ventricles of the brain against the background of normal intra-cranic pressure values and manifested by a specific triad or symptoms including gait disturbance, cognitive disorders and dysuria (primarily urinary incontinence) [12]. They paid special attention to the possible reversibility of clinical manifestations in this syndrome by adequate surgical with ventricular-peritoneal shunting. There are few reasons for a significant (up to 80%) under diagnosis of this disorder. First, the main difficulty is the differential diagnosis of Normal Pressure Hydrocephalus and similar diseases, including neurodegenerative ones (Alzheimer Disease, Parkinson Disease, vascular Dementia, dementia with Levy Bodies). Secondly, the

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diagnosis of NPH is laborious because the dementia symptom. The main theory that precise the development of NPH concept proposed by Greitz. According to it, the imbalance between secretion and reabsorption of brain-spinal fluid is a key link of NPH pathogenesis. Fundamentally, this disease is a brain-spinal pathways disorder, with metabolic and neurodegenerative factors and heredity.

Depending on the detection of the immediate cause of the disease, NPH is divided into two subtypes, a) secondary NPH and primary or idiopathic NPH, detected in approximately 40-60% of cases, more than ten in older patients when a history there is no indication of any clear cause underlying the development of the disease. The most common reasons for NPH include intra-cranic hemorrhage, traumatic brain injury, purulent inflammatory processes in the cranial cavity, surgical operations on the brain, ventricular-peritoneal bypass, are the better method in nature to improve a symptoms' regression.

Epilepsy Neurophysiology

Consciousness is loss when the function of both cerebral hemispheres or the brainstem reticular activation system is compromised. Episodic dysfunction of these anatomic regions precedes 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 [13]. Epilepsy, a group of disorders characterized by recurrent seizures, 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. A single epileptic syndrome (for example, juvenile myoclonic epilepsy), can result from mutations in several different genes and, conversely, mutations in a single gene (for example, SCN1A, sodium channel subunit) can cause several epilepsy phenotypes. Genes implicated in susceptibility to epilepsy include those coding for sodium, calcium, potassium, and chloride channels, nicotinic cholinergic, GABA, and G protein-coupled receptors, and enzymes.

Cryptogenic Seizures

Cryptogenic seizures, account for 2/3 of new onset seizures in the general population. The age range is broad, from the second to seventh decade. A second seizure increases the risk of recurrence to approximately 75%. Genes implicated in cryptogenic epilepsy include the mitochondrial NAD-dependent malic enzyme ME2 and the CACNA1A and CACNB4 calcium channel subunits.

Temporal Lobe Epilepsy

Temporal lobe epilepsy is an enduring brain disorder that causes unprovoked seizures from the temporal lobe. Medial temporal lobe epilepsy arises from the inner part of the temporal lobe that may involve the hippocampus, para-hippocampal gyrus or amygdala gyrus or amygdala, the lateral temporal lobe arises from the outer temporal lobe. In medial temporal lobe epilepsy can occur repeated stereotyped motor behaviors, automatism and dystonic posture is seizure. Lateral temporal lobe seizures arising from the temporal-parietal lobe junction may cause complex visual hallucinations [14].

Results in the present Research

Normal Pressure Hydrocephalus and Color Vision

The results have obtained analyzing 28 Calabria male patients (age range 51-84 years; mean age, 73.2 ± 1.57 years) showing a mean disease duration of 4.4 ± 0.91 years (range, 1.0-23 years). 7/28 patients showed normal color vision with all intact visive areas. 10/28 patients made the surgical ventricular-peritoneal shunt: 5/10 restored their normal color vision, and 5/10 retain their both black/white vision without any color, and color vision deficiency protan/deutan/tritan, and its miscellaneous. 11/28 patients were without surgical ventricular-peritoneal shunt: they show both black/white vision, and color vision deficiency.

Epilepsy and Color Vision

Our research on 46 epileptic patients showed the following results, referring to different epilepsy kind: 26 patients showed a normal color vision; temporal lobe patients showed 6/12 impair color vision; focal epilepsy patients showed 12/30 with impair color vision (18/30 normal); secondary epilepsy patients showed 2/2 impair color vision; congenital epilepsy patients showed no impair color vision. Epileptic patients who suffer from the above kinds of epilepsy showing the normal color vision, have for the most brain gaps in the white matter in resonance Imaging [15]. Moreover, diffuse subarachnoid large spaces and bad hippocampal rotation are present. All the frontal and parietal punctiform images are present not in occipital brain area. So, we can think that the color vision area is intact.

Discussion and Conclusion

In the group of patients showing the black/white vision, very likely we have a compromise of the visual pathways from V1 primary visive area to V4 color vision area in the middle brain. The very compromise of the primary visual area V1 does not allow that the visual stimulus can arrive to V4 area. And the clinical evidence makes such a suggestion plausible because the integrity of both above areas is critical to see and be consciously aware of having seen the colors. Evidently, in these patients miss the intact return pathways from V4 to V1. This integrity is restored in the patient who has a new normal color vision after the surgical ventricular-peritoneal shunt.

In the group of patients showing the color vision deficiency both on red (green, and/or on blue/yellow axis), very likely there

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is no a great compromise of the V1. But, very likely both great and small compromise of the visive areas is related only to V4. 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 re not very compromised, and it has been restored after the surgical ventricular-peritoneal shunt.

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 and CA3 pyramidal cells leading to massy fiber sprouting and new excitatory networks capable of generating seizures [15]. Cortical dysplasia is a brain malformation that may cause focal or lobe temporal epilepsy. This malformation may cause abnormal cortical layers (dyslamination), occur with abnormal neurons (dysmorphic neurons, balloon cells), and may occur with a brain tumor or vascular malformation.

References

1. Hondrick T (1987) Mind, brain and self-conscious mind. In: "Mindwayes". Blakemore C, Greenfield S, eds. Oxford: Basil Blackwell pp. 445-460.
2. Zeki S (1993) A vision of the brain. Oxford: Blackwell Scientific Publication.
3. Zeki S (1973) Colour coding in rhesus monkey prestriate cortex. Brain Res 53(2): 422-427.
4. Zeki S (1980) The representation of colours in the cerebral cortex. Nature 284(5755): 412-418.
5. Dubois-Pulsen A (1952) Colour vision in brain lesions. In: "Color vision deficiencies". Verriest G, ed. The Hague: Junk, pp. 429-440.
6. Fletcher R, Voke J (1985) Defective color vision. Fundamentals, Diagnosis and Management. Bristol: A. Hilger.
7. Lyle WM (1974) Diseases and conditions (Part II of drugs and conditions which may affect color vision). J Am Optom Assoc 45: 173-182.
8. Lyon MF (1962) Sex chromatin and gene action in the mammalian X chromosome. Am J Hum Genet 14(2): 135-148.
9. Ishihara S (1998) Ishihara's test for colour deficiencies. Tokyo: Kanehara and Co. LTD.
10. Farnsworth D (1943) The Farnsworth-Munsell 100 hue and dichotomous test for color vision. J Opt Soc Am 33: 568-578.
11. Fletcher R (1975) The City University Colour Vision Test. London: Keeler Instruments.
12. Adams RD, Fisher CM, Hakim S, Ojemann RG, Sweet WH (1965) Symptomatic occult hydrocephalus with normal cerebrospinal fluid pressure: a treatable syndrome. N Eng J Med 272: 117-126.
13. AminoffM, Greenberg DA, Simon RP (2015) Clinical Neurology. Bristol and Boston: Mc Graw-Hill.
14. Salmeupera TM, Duncan JS (2005) Imaging in epilepsy. J Neurol Neurosurg Psichiatry 76(3): iii2-iii10.
15. Rusu V, Chatoux F, Landre E, Bouillert V, Nataf F, et al. (2005) Dystonic posturing for seizures of mesial temporal origin: in electroclinical and metabolic patterns. Neurol 65(10): 1612-1619.

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