



## Mini Review Article

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# Different Kind of Epilepsy, and different Kind of Color Vision Impairment: Neurophysiological Implications

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## Abstract

The study on color vision in epileptic patients representing the most important different kinds of this neurologic disorder, is a new, and original result at the Literature in the field, to-day. The also subtle, and some time hidden impair brain pathways in epilepsy highlighted by a impair color vision which resulted to be again a biological marker.

## General Knowledge on Consciousness Status

Consciousness is lost when the function of both cerebral hemispheres or the brainstem reticular activating system is compromised. Episodic dysfunction of these anatomic regions produces 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 [1]. Syncope is loss of consciousness due to reduced blood flow to both cerebral hemispheres or the brainstem. It can result from pan-cerebral hypoperfusion caused by vasal-vagal reflexes, orthostatic hypotension, decreased cardiac output, or from selective hypoperfusion of the brainstem resulting from vertebral-basilar ischemia. Seizures, and syncope are the most important causes for consciousness loss,

have different causes, but diagnostic approaches, and treatment. If all spelling are identical for these two disorders, then a single pathophysiologic process can be assumed [2].

## Epilepsy: General Tasks

Epilepsy, a group of disorders characterized by recurrent, is a common cause of episodic loss of consciousness; the prevalence of epilepsy in the general population is about 1% and the lifetime probability of a seizure is approximately 10%. 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. The age of the patient may

help in establishing the cause of seizure. The genetic contribution to epilepsy and its response to treatment is complex. 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.

### Epilepsy Kind

**Events at onset of Spell of Generalized Epilepsy:** The often brief, stereotyped premonitory symptoms, as aura, at the onset of some seizures may localize the central nervous system abnormality responsible for seizures. A sensation of fear, olfactory or gustatory hallucinations, or visceral or déjà vu sensations are commonly associated with seizures originating in the temporal lobe. Progressive light-headedness, dimming of vision and faintness suggest decreased cerebral blood flow.

**Events after the Spell of Generalized Epilepsy:** Recovery from a simple faint is characterized by a prompt return to consciousness, with full lucidity, within 20 to 30 seconds. A period of confusion, disorientation, or agitation follows a generalized tonic-clonic seizure. The period of confusion usually lasts only minutes. Although such behavior is often strikingly evident to witness, it may not be recalled by the patient. Prolonged alteration of consciousness may follow status epilepticus. It may also occur after a single seizure

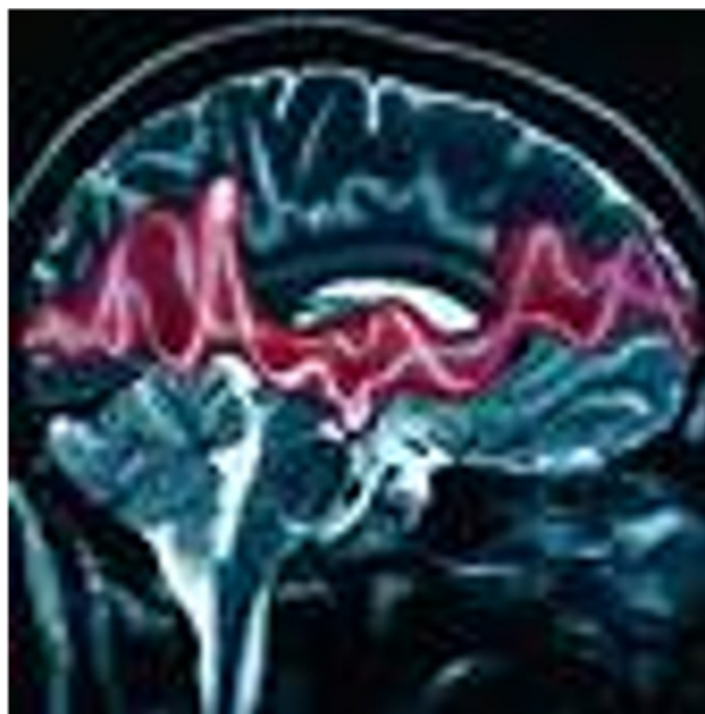
in patients with diffuse structural disease (for example, dementia, cognitive impairment), metabolic encephalopathy, or continuing non convulsive seizures.

### Cryptogenic Seizures

These account for 2/3 of new onset seizures in the general population. The age range is broad, from the second to the seventh decade. The risk of recurrence in the next 5 years is approximately 35% after a first unprovoked seizure. 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 e CACNB4 calcium channel subunits.

### Temporal Lobe Epilepsy

Temporal lobe epilepsy is an enduring brain disorder that causes unprovoked seizures from the temporal lobe. Memory and psychiatric comorbidities may occur. 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 present in seizure. Lateral temporal lobe seizures arising from the temporal-parietal lobe junction may cause complex visual hallucinations [3].



Figures 1: Epilepsy.

## Color Vision and Epilepsy in the present work [4]

Temporal Lobe	Impair Color Vision = 6/12 patients
Epilepsy	Normal Color Vision = 6/12 patients
Focal Epilepsy	Impair Color Vision = 12/30 patients
	Normal Color Vision = 18/30 patients
Secondary Epilepsy	Impair Color Vision = 2/2 patients
	Normal Color Vision = 0/0 patients
Congenital Epilepsy	Impair Color Vision = 0/0 patients
	Normal Color Vision = 2/2 patients
Total patients = 46	
Total Impair Color Vision patients = 20	
Total Normal Color Vision patients = 26	

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 present not in occipital brain area. So, we can think that the color vision area is intact. Epileptic patients who suffer by the above kinds of epilepsy showing the impair color vision, as the above, showed for the most, asymmetric brain space, and sulci, at the Magnetic Resonance Images. Callosus corpus is altered, the punctiform areas are present in T2, and FLAIR at Magnetic Resonance Imaging. And, in these patients, the brain occipital lobe shows hypodensities. So, we can think that the color vision area is not intact.

## Discussion

Hippocampal sclerosis occurs with severe CA1 and less CA3 and CA4 neuronal loss, following a N-methyl-D-aspartate receptor (NMDA). Repetitive seizures irreversibly damage inter-neurons leading to persistent loss of recurrent inhibition. Damage of GABAergic inter-neurons lead to loss of inhibition, uncontrolled neuronal firing, leading to seizures [5]. The other secondary epileptic hypothesis is that repetitive seizures lead to inter-neuron loss, loss of glutamatergic principal neurons, axonal sprouting, and formation of new recurrent glutamatergic excitatory circuits leading to a more severe epilepsy [5]. The KCC2 mutation prevents subiculum neurons from potassium and chloride ion extrusion, leading to in-

tracellular chloride accumulation, and positive GABA mediated currents. Accumulated chloride efflux through GABA receptors leads to neuronal depolarization, increased neuronal excitability and ultimately seizures [5]. Dentate gyrus granule cell dispersion refers to a granule cell layer that is widened, poorly demarcated, or accompanied by granule cells outside the layer (ectopic granule cells) [6]. 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 mossy fiber pathway connecting granule cells and CA3 pyramidal cells leading to mossy fiber sprouting and new excitatory networks capable of generating seizures [7]. A 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 [8].

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## Conflict of Interest

There are no conflicts of interest.

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