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ملخص

عمى الألوان أو خلل تمييز الألوان (CVD) يشير إلى عدم القدرة على التمييز بوضوح بين الألوان في ظروف الإضاءة الطبيعية. قد يكون خلقيًا أو مكتسبًا، حيث ينتج الخلقي عن طفرة جينية. وعلى العكس، فإن خلل تمييز الألوان المكتسب، ينتج عن أمراض عينية أو جهازية أو مرتبطة بالأدوية، قد يكون أيضًا علامة مبكرة على مرض كامن.

كان الهدف من هذه الدراسة تقييم حالات خلل تمييز الألوان المكتسب الناتجة عن أسباب عينية أو جهازية أو مرتبطة بالأدوية باستخدام اختبار D-15 المزدوج، وهو الجمع بين اختبار فانسورث D-15 المشبع وغير المشبع .

شملت العينة المدروسة 40 مريضًا موزعين بالتساوي بين الذكور والإناث؛ كل واحد منهم كان يعاني إما من أحد الأمراض العينية أو من أمراض جهازية مرتبطة مع الأمراض العينية. أظهرت النتائج سيادة واضحة للأسباب العينية (92%)، حيث كان الجلوكوما واعتلال الشبكية السكري أكثر الأمراض شيوعًا. تلتها الأمراض الجهازية والأسباب المرتبطة بالأدوية. كما لوحظ أيضًا أن محور الأزرق-الأصفر الناتج عن الأسباب العينية سائد بشدة، وهو ما يتماشى مع البحوث المتداولة حول خلل تمييز الألوان المكتسب.

تؤكد هذه النتائج أن خلل تمييز الألوان المكتسب يُعد مظهرًا سريريًا شائعًا ومتكررًا لمجموعة متنوعة من الحالات المرضية، مما يوضح أيضًا القيمة التشخيصية للجمع بين اختبار D-15 المشبع وغير المشبع في تقييمات الرؤية اللونية السريرية.

الكلمات المفتاحية: عمى الألوان، خلل مكتسب في تمييز الألوان، اختبار D-15 غير المشبع، اختبار D-15 المشبع، المحور الأزرق-الأصفر، CVD

Résumé

Le daltonisme, ou dyschromatopsie (CVD), désigne l'incapacité à distinguer clairement les couleurs dans des conditions d'éclairage normales. Il peut être congénital ou acquis, le congénital résultant d'une mutation génétique. En revanche, le CVD acquis, causé par des pathologies oculaires, systémiques ou liées aux médicaments, peut également constituer un signe précoce d'une maladie sous-jacente.

L'objectif de cette étude était d'évaluer les cas de vision des couleurs acquise présentés pour des raisons oculaires, systémiques ou médicamenteuses en utilisant le double test D-15, qui est la combinaison des deux tests du Farnsworth Panel D-15 désaturé et saturé.

L'échantillon étudié comprenait un total de 40 patients avec une répartition égale entre hommes et femmes ; chacun d'eux présentait soit une des affections oculaires, soit des affections systémiques associées aux maladies oculaires.

Les résultats ont montré une nette dominance des étiologies oculaires (92 %), avec le glaucome et la rétinopathie diabétique comme pathologies les plus fréquentes. Suivies par les maladies systémiques et les causes liées aux médicaments. Une prédominance de l'axe bleu-jaune causée par les étiologies oculaires a également été observée, ce qui est cohérent avec la littérature établie sur le CVD acquis.

Ces résultats confirment que les déficiences acquises de la vision des couleurs sont une manifestation clinique courante et fréquente de diverses conditions pathologiques, démontrant la valeur diagnostique de la combinaison des tests D-15 désaturé et saturé dans les évaluations cliniques de la vision des couleurs.

Mots-clés : Daltonisme, Déficiency acquise de la vision des couleurs, CVD, D-15 désaturé, D-15 saturé, Axe bleu-jaune

Abstract

Color blindness, or dyschromatopsia (CVD), refers to the inability to clearly, distinguish between colors under normal lighting conditions. Which may be congenital or acquired, with the congenital resulting from a genetic mutation. On the contrary, the acquired CVD, caused by ocular, systemic, or drug-related pathologies, may also be an early sign of underlying disease.

The objective of this study was to evaluate acquired color vision cases presented with ocular, systemic, or drug-related reasons using the dual of D-15 tests, which is the combination of the two tests of Farnsworth Panel Desaturated and Saturated D-15.

The studied sample included 40 patients in total with an equal distribution between males and females; each one of them either presented with one of the ocular conditions or associated systemic conditions with the ocular diseases.

The results showed a clear dominance of ocular etiologies (92%), with glaucoma and diabetic retinopathy as the most frequent pathologies. Followed by systemic diseases and drug-related reasons. With also an observed predominance with Blue – Yellow axis caused by the ocular etiologies, which is coherent with established literature on, acquired CVD.

These findings confirm that acquired color vision deficiencies are a common and frequently clinical manifestation of various pathological conditions, demonstrating the diagnostic value of combining the Desaturated and Saturated D-15 tests in clinical color vision assessments.

Keywords: color blindness, acquired color vision deficiency, CVD, Desaturated D-15, Saturated D-15, blue–yellow axis.

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List of abbreviation

AMD	Age-Related Macular Degeneration
AO H-R-R	American Optical Hardy, Rand, and Rittler
AR	Augmented Reality
B-Y	Blue–Yellow
CCT	Cambridge Color Test
CNGA3	Cyclic Nucleotide-Gated Channel Alpha 3
CNGB3	Cyclic Nucleotide-Gated Channel Beta 3
CSC	Central Serous Chorioretinopathy
CUT	City University Test
CVD	Color Vision Deficiency
CVTME	Color Vision Test Made Easy
D-15	Panel D-15 (Farnsworth)
DR	Diabetic Retinopathy
ERG	Electroretinogram
FM 100	Farnsworth-Munsell 100 Hue Test
GNAT2	Guanine Nucleotide-Binding Protein Alpha Transducin 2
LHON	Leber Hereditary Optic Neuropathy
MS	Multiple Sclerosis
ON	Optic Neuritis
OPN1LW	Long Wavelength-Sensitive Opsin 1 Gene
OPN1MW	Medium Wavelength-Sensitive Opsin 1 Gene
OPN1SW	Short Wavelength-Sensitive Opsin 1 Gene
PD	Parkinson's Disease
PDE5	Phosphodiesterase Type 5
R-G	Red–Green
RHO	Rhodopsin
RP	Retinitis Pigmentosa
RPE	Retinal Pigment Epithelium
UV	Ultraviolet
UX	User Experience

Introduction

Color vision is essential for the human visual perception, giving him the ability to identify and distinguish colors. Therefore, a category of individuals suffers from color blindness. Which is a disability to differ between colors, that can be present at birth or be an acquired condition resulted from many reasons, with affecting the individuals' daily life.

Color vision deficiency (CVD), can be caused by genetic mutation in one of the cones responsible for color perception, or by abnormalities present in the retina and diseases that impair color vision. Moreover, multiple diagnostic methods were made to detect and determine the various types of color vision deficiencies, with the many different approaches to treat color vision deficits.

This manuscript consists of two parts (theoretical and practical), while the theoretical section talks about color vision deficiency globally, delving into its complexities, exploring its origins within the human body, the various causes and types, several tests for diagnosis, and potential solutions to aid daily life. The practical section is a cross-sectional clinical study about cases with acquired color vision deficiencies using the dual color vision tests (the Desaturated and Saturated D-15), with the purpose of identifying the common acquired CVD types according to etiological reasons and detecting the underlying pathologies at a pre-diagnostic stage.

Theoretical section

Chapter I: Color Blindness

I. Introduction

Color vision is important in the human life, presented by three types of cone photoreceptors. In this chapter, we'll dive into the importance of color vision to individuals and how does it affect people, resulting to color blindness.

In addition to, understanding the anatomy of the retina by also differentiating between rods and cones. Thus, figuring out how the perception of color work in the human eye.

Concluding the chapter with branching to the etiology of color blindness and discovering the reasons that may cause color vision deficiencies.

II. Definition of color blindness

Color blindness, or dyschromatopsia (CVD), which refers to the inability to clearly distinguish between colors under normal lighting conditions. According to the Young–Helmholtz trichromatic theory of normal color vision, there are three types of cones, each containing a different photopigment that is maximally sensitive to one of the three primary color ranges: short wavelengths (blue), medium wavelengths (green), and long wavelengths (red) [1].

John Dalton being the first scientist to give a clear description of his own affliction of color blindness in 1798 explaining it as an anomaly or an absence in one of the cones. Color blindness can therefore result from a genetic mutation affecting cone pigments, or it may be acquired due to abnormalities in the retina or diseases that impair color vision[2]. In addition, CVD is significantly more common in males than in females, affecting approximately 8% of men compared to about 0.5% of women [3].

To simplify, color blindness can be either congenital or acquired, which result to a difficulty in identifying colors, whichever makes the individual suffer in his daily life [3].



Figure 1: Color Blindness [3]

III. Anatomy of the retina

1. The retina

The retina, a delicate and transparent layer derived from the neuroectoderm, represents the innermost nervous tunic of the eye. It initiates vision through its sensory neurons, and is divided into two distinct regions, separated by the ora serrata. This neural retina consists of ten distinct layers: the outer and inner segments of photoreceptor cells (rods and cones), the external limiting membrane, the outer nuclear layer, the outer plexiform layer, the inner nuclear layer, the inner plexiform layer, the ganglion cell layer, the nerve fiber layer, and the internal limiting membrane [4], [5].

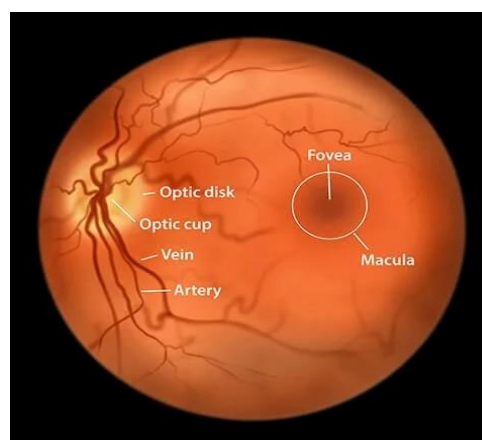


Figure 2: The Retina [65]

Moreover, it is presented by two zone:

- **Optic disc:** The optic disc, or optic papilla, is a round area of approximately 3 mm², where the axons of retinal ganglion cells converge to create the optic nerve. Moreover, at this site, the central retinal artery enters and the retinal vein exits the eye. Thus, the normal optic disc appears orange to pink in color. Because this region lacks photoreceptors (rods and cones) and other retinal layers, it is insensitive to light and corresponds to the physiological blind spot in the visual field. Nevertheless, the brain compensates for this gap by including information from the other eye, so the blind spot is usually invisible in daily vision [6] [7].
- **The macula:** The macula lutea, in other words the yellow spot. Therefore, the region of the retina responsible for the sharpest vision. Thus, it measures nearly 5 mm and includes the highest concentration of cone photoreceptors, making it important for color perception. Moreover, at its center lies the fovea, which represents the area of maximal visual acuity due to the densest packing of photoreceptors in the eye. When an image falls on the macula, its most detailed representation is projected onto the fovea [7].

In addition, the macula lutea is the retinal region primarily responsible for color perception, owing to its high density of cone photoreceptors. These cones—classified as short (S), medium (M), and long (L) wavelength receptors—enable the discrimination of blue, green, and red hues, respectively, thereby supporting fine chromatic vision i.e. the trichromatic theory [8] .

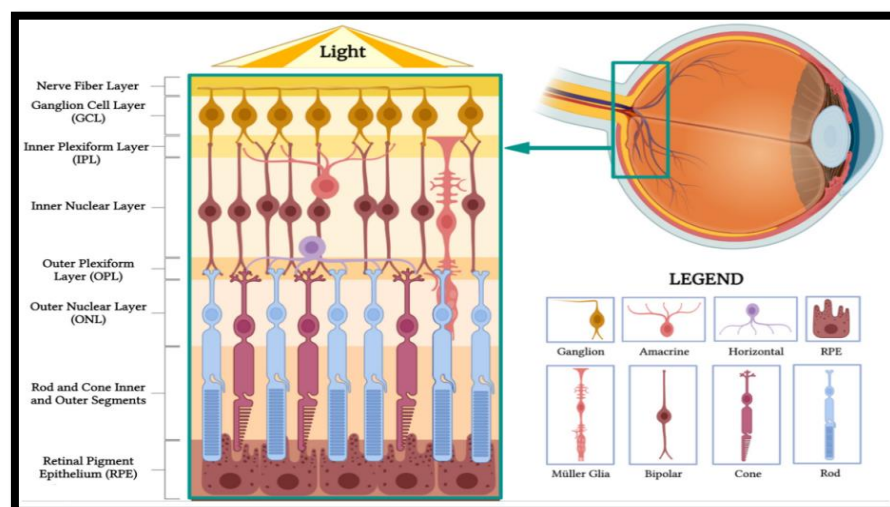


Figure 3: Anatomy of the Retina [5]

2. Photoreceptors

Photoreceptors consisting of rods and cones, characterized by their morphology, patterns of gene expression, sensitivity to light and functional properties. Located in the outermost retinal layer adjacent to the retinal pigment epithelium (RPE) [4], [9].

2.1. Rods

Rod photoreceptors function under dim illumination and are involved in motion detection. They predominate in the peripheral retina, with approximately 92 million rods present.

Furthermore, the spectral sensitivity of each photoreceptor type is determined by the expression of specific opsin proteins. Opsin photopigments are light-sensitive G-protein-coupled receptors covalently bound to an 11-cis-retinal chromophore via a Schiff base. Rods contain rhodopsin (RHO), which has a maximal absorption at 498 nm [9], [10].

2.2. Cones

Cone photoreceptors function under ambient and bright illumination, exhibiting rapid responses to changes in light intensity, with being responsible for high-acuity daytime vision and trichromatic color perception.

Moreover, in humans the three cone subtypes are defined by the expression of opsin proteins that are optimally sensitive to distinct wavelengths of light. While, cones contain either short-wavelength opsins (S/blue cones), medium-wavelength opsins (M/green cones), or long-wavelength opsins (L/red cones). They express S-, M-, or L-opsin (OPN1SW/MW/LW), with peak absorption at 420 nm for S-opsin, 534 nm for M-opsin, and 564 nm for L-opsin.

Additionally, the human retina contains about 4.6 million cones (range 4.08–5.29 million) [9], [11].

In addition, each of the cones follows the principle of Univariance, which explained as the absorption of long wavelength (low frequency, low energy), is the same influence on a receptor as the absorption of a short wavelength (high frequency, high energy). Thus, it is the possibility of absorption that changes photoreceptors sensitivity [12].

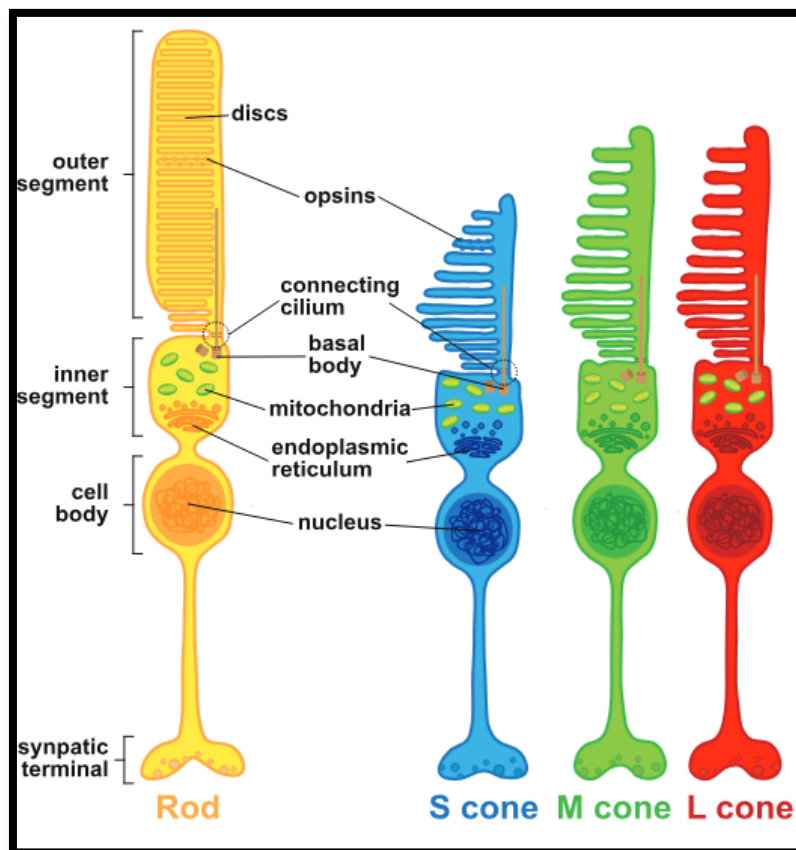


Figure 4: Retina' Photoreceptors [8]

Cone type	Name	λ range (nm)	Peak λ (nm)	Color
Small S	Blue β	400 - 500	420 – 440	Violet
Medium M	Green γ	450 - 630	534 – 545	Green
Large L	Red α	500 - 700	564 - 580	Yellow

Table 1: Cone cell types, ranges and peak sensitivities of the human eye [13]

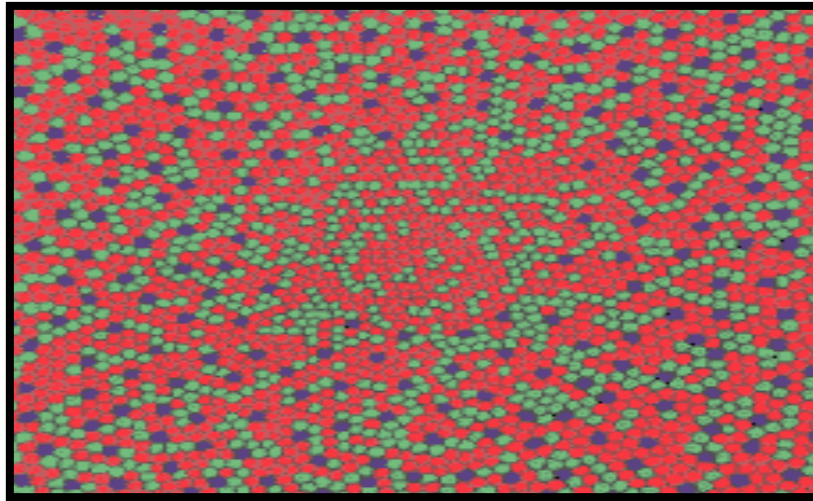


Figure 5: Cone distribution in the Macula [64]

IV. Etiology of color blindness

1. Genetic etiology

Congenital defects appear in two forms, total and partial. The former is rarer and generally associated with nystagmus and a central scotoma, with all colors manifesting as shades of greys with varying brightness.

The latter form (partial) with being the most common type of color vision deficiencies, primarily inherited and transmitted through the mother (and still unaltered) [14]. Resulting from mutations in the genes coding for cone photopigments in the retina.

The mutations often affect the longwave-sensitive opsin -1 gene (OPN1LW) and the mediumwave-sensitive opsin – 1 gene (OPN1MW) because it is located on the X chromosome; On the other hand, the shortwave-sensitive opsin 1 gene (OPN1SW) is autosomal (non-sexual chromosome) [15].

Congenital color vision deficiencies affect 0.5–8% of the population, with red–green color blindness as the most common variant, influencing roughly 8% of males and 0.35% of females due to its X-linked recessive inheritance pattern. Depending on whether cone type is compromised, the insufficiency shows as protanopia (loss of L-cones), deutanopia (loss of M-cones), or tritanopia (loss of S-cones). These congenital abnormalities are bilateral, stationary, and non-progressive, resulting in lifelong problems in differentiating colors, particularly in the red/green spectrum [15] [16].

2. Acquired etiology

The most common type of color vision deficiency is congenital, which means it is passed down through families and is present at birth. However, color vision defects can also be acquired, most often due to ocular diseases affecting the retina [17].

When the optic nerve and its pathways are damaged, individuals typically lose sensitivity to red–green colors. Meanwhile, damage to the outer retinal layers usually results in a blue–yellow deficiency [14].

Abnormalities of color vision may sometimes act as early warning signs of ocular damage resulting from irregularities of the optic nerve, retinal problems, or brain disorders [2].

In addition, acquired defects are more complex and should not be classified by the affected photoreceptor, but instead as a variable loss along a particular color axis: blue–yellow axis, red–green axis, or a nonspecific loss of color vision. It may also modify in severity with time, given that it is more likely to be asymmetric.

Moreover, the majority of acquired color vision deficiencies are tritan, with having an equal percentage in male and females, and are typically accompanied by a reduction in visual acuity [17].

Aspect	Acquired color-vision defects	Inherited color-vision defects
Onset	Not present at birth	Present at birth
Progression	May be progressive	Do not change over time (stable)
Diagnostic clarity	Combined defects may occur making diagnosis difficult.	Precise diagnosis of a given type (protan, deutan, tritan)
Ocular involvement	Frequently monocular	Mostly binocular
Visual acuity	Decreased visual acuity	Visual acuity unaffected (except achromatopsia, blue-cone monochromacy)
Predominant type	Predominantly tritan deficiencies	Predominantly red–green deficiencies, rarely tritan
Gender distribution	Equal incidence in males and females	Higher incidence in males
Frequency	1 in 20 individual	1 in 12 males affected with red–green defect

Table 2: Comparative table between acquired and congenital color vision defects [17], [18]

Etiologically, we can divide the acquired CVD to three categories: oculars diseases, systematic diseases plus drugs and toxicities [17]:

2.1. Conditions Resulting in an Acquired Color-Vision Defect

2.1.1. Ocular diseases

Considered the most widespread acquired color defects, which occur from different diseases that could touch the eye in various ways. Thus, ocular opacities and retinal pigment epithelium (RPE), progressive cone dystrophies with abnormalities that involve the retinal neuros (ganglion cells), are provoked by these diseases.

Moreover, the features of these defects are central visual field loss, and/or light sensitivity (photophobia), electroretinogram (ERG) cone abnormality with decreased visual acuity. Furthermore, the intensity of color vision deficit with the age of the individuals and the ratio of progression could change vastly among the various diseases.

However, color vision deficiency is most likely the least worry for these subjects, yet it is connected to the disease (which itself may be an inherited condition), and could sometimes assist with the prognosis [17].

A few of the most common diseases are shown below:

2.1.1.1. Age-related macular degeneration

Age-related macular degeneration (AMD) is portrayed as the most common form of acquired color vision deficits and one of the reasons leading to blindness. Thus, the disorder varies to two forms dry and wet, either of them include a degeneration of the photoreceptors in the central retina (macula), which is the field of which the acuity and color vision are at its peak. That is why patients with AMD have trouble in both.

Moreover, in the slow-progressing dry form of age-related macular degeneration, a yellow deposit known as drusen is produced beneath the macula. Which influences negatively the condition of the retinal pigment epithelium, a layer essential for preserving the normal operational function of photoreceptor cells. As a result, the vision performance declines over time.

On the other hand, tiny blood vessels start to form behind the retina in the direction of the macula, which is in the fast-progressing age-related macular degeneration. Additionally, the weak fragile blood vessels result in leaking, bleeding and scarring, thus damaging the retina with a fast vision loss and irreversible impact.

As the disease progresses and photoreceptors cells are being progressively damaged, color vision gradually deteriorates, but this does not usually contribute to the differential diagnosis [17].

2.1.1.2. Glaucoma

Worldwide, glaucoma is the second major cause of blindness. Thus, it corresponds to a number of handicaps within the optic nerve presented by a loss of retinal ganglion cells with a loss of some or most of the fibers of the optic nerve.

Additionally, in glaucoma the reduction of the visual field appears progressively over time, and may not be realized until the disability is quite severe, with a proportion of 60% of the retinal ganglion cells declining before a perceptual result is determined.

Moreover, critical visual field loss has been suggested to be anticipated by these individuals' decreasing color vision. Thus, the concept that acquired color deficiency in glaucoma occurs by damage to the neural pathways rather than the retinal photoreceptors is proven by the fact that field loss results from diffuse anomalies in the neural retina.

Although these subjects have a normal S-cone electroretinogram (ERG), acquired tritanopia (blue-yellow defects) is usually detected with severity paralleling the visual field loss. Thus, the tritan deficit is consistent with other optic nerve abnormalities, demonstrating the S-cones staying intact, except that the postreceptoral (refers to neural processing that occurs after the photoreceptors (rods and cones) have captured light) mechanism included in the transmission of blue-yellow distinction should be selectively disturbed [17].

2.1.1.3. Cataract

Cataract is an age-related condition, with a defect of the crystalline lens of the eye. Which is the lens hardening, becoming opaque and yellowish.

Furthermore, a regular lens selectively absorbs short-wavelength light, but both normal aging and the progress of cataracts ameliorate this impact and result in trouble distinguishing in the blue-green part of the spectrum. Thus, it is called xanthopsia (Yellow Vision) and relates to the abundance of yellow whilst dominating the visual space.

However, patients often observe Cyanopsia, or a blue-dominated visual perspective, after cataract surgery, in which their natural lens is removed and replaced with a clear plastic lens. The cause of this cyanopsia is that the brain applies a blue tint to the visual scene to compensate for the perceptual yellow tint displayed by the cataract, and it takes some time for the brain to adapt to the new chromatic input once the yellow lens is removed. Hence, after cataract removal, the mild-blue perception may remain for weeks or even months, but normal color vision eventually recovers, showing the adult visual system's notable chromatic plasticity [17].

2.1.1.4. Retinitis pigmentosa

Retinitis pigmentosa (RP) is considered as a hereditary retinal dystrophy with anomalies in rods or the retinal pigment epithelium (RPE) of the retina, leading to progressive visual loss. Additionally, patients first face nocturnal blindness and defective dark adaptation, with decreases in periphery vision (tunnel vision), and followed sometimes with a loss in central vision after the progression of the disease.

Furthermore, it is proven that the ongoing degeneration of the rods can result in a loss of cones (a result of reduced rod-derived cone viability factors). Thus, as much as the cones are degenerating, there will be a disturbance in distinguishing colors, also resulting in patients suffering severe tritan defects or pseudoprotanomaly (a mild red-green defect).

Eventually, subjects diagnosed with RP go blind; therefore, again the color-vision defect is of relatively minor importance to the individual [17].

2.1.1.5. Diabetic retinopathy

Diabetes, a systemic disease presented by high blood sugar levels that can result in severe changes in the retinal blood vessels affecting patients' vision, which manifests as an ocular disease known as Diabetic Retinopathy (DR).

Additionally, the neovascularization and edema in new vessels in the eyeball can lead to bleeding, blurred vision, with degeneration of the retina, by failing to provide sufficient oxygen and fulfil the metabolic requirements of the photoreceptors.

Furthermore, visual disturbance starts with blur; however, the pattern of vision loss in these individuals is frequently widespread, uneven, and achromatic, declining vision perception to shades of grey disrupted by scotomas [17].

2.1.1.6. Optic neuritis (Névrite optique)

Partial or complete vision loss can be induced by inflammation of the optic nerve, which is known as optic neuritis (type of Optic neuropathy). Thus, individuals reported sensitivity to light (photophobia), pain while moving the eye, and a deterioration of color vision.

Direct axonal (nerve fibers for the electrical signals) can act as a crucial part in nerve demolishment in many cases. Thus, the weakening of color vision begins with an irregular dimming, and a noticeable increased noise in the visual images, and then escalates to a wide fading or loss of color recognition and blurring throughout the whole visual field. Additionally, in most cases, only a single eye is damaged and patients remain indifferent to the loss of color vision unless the doctor tells them to cover or close the healthy eye.

Generally, these deficiencies resemble deutan deficiency (a form of red–green defect); however, there's also an additional decrease in sensitivity to the shorter wavelengths (blue-yellow).

Up to one-third of optic neuritis is the presenting symptom of multiple sclerosis (MS). Thereby, almost half of all patients with multiple sclerosis (sclérose en plaque) will build an optic neuritis condition. Thus, the destruction of the myelin sheath (gaine de myéline) may cause inflammation of the optic nerve. Patients with multiple sclerosis may experience repeated episodes of optic neuritis, which worsen with time and lead to a permanent loss of color vision [17].

2.1.2. Systematic diseases

2.1.2.1. Multiple sclerosis

Multiple sclerosis (MS) is a chronic demyelinating and axonal degenerative disease of the central nervous system that commonly includes the visual pathways, with optic neuritis (ON) portraying one of the most frequent earliest manifestations.

Although visual acuity often improves after optic neuritis (ON), small visual deficits may remain, including changes in contrast sensitivity and color identification.

Thus, recent research proposes that even with the clinical optic neuritis ON not present, individuals with multiple sclerosis MS could undergo visual pathway involvement due to both diffuse axonal damage and demyelination [19], [20].

2.1.2.2. Parkinson's disease

Parkinson disease (PD) is a neurodegenerative condition, that damage cognitive, motor, and sensory function throughout the progressive loss of dopaminergic neurons.

Thus, abnormal color vision was reported in patients with Parkinson disease, which is resulted from altering the retinal visual processing.

Moreover, Parkinson disease patients are identified by weakening the S-cone photoreceptors, resulting to an acquired Blue – Yellow color deficiencies [17].

2.1.3. Drugs and toxics

Besides the internal impairment that induce color vision anomalies, other various environmental reasons can also change and alter color vision.

Moreover, many therapeutic drugs have been proven to cause color vision abnormalities in patients. Although the majority of these cases have been recorded due to high doses of prescribed medications, some might happen even at recommended dosages; however, most adverse effects are temporal. Multiple research have figured out altered color perception in individuals working in exposed areas to chemicals as well. A potential mechanism for these toxin-induced abnormalities may involve the direct impact of the chemicals on cone performance and/or disruption of neurotransmitter transmission [17].

Examples of drugs and substances that may alter color vision perception: Digitalis (digoxin, digitoxin), PDE5 inhibitors, Chloroquine Hydroxychloroquine, Ethambutol, Ethanol, Methyl-alcohol [16], [17].

Condition	Color vision defect
Optic neuritis	R-G
Stargardt's disease	R-G
Best's disease	R-G
Leber's optic atrophy	R-G
Glaucoma	B-Y
Retinitis pigmentosa	B-Y
Age-related macular degeneration	B-Y
Myopic retinal degeneration	B-Y
Chorioretinitis	B-Y
Diabetic retinopathy	B-Y
Dominant hereditary optic atrophy	R-G
Papilledema	R-G
Tobacco-alcohol or toxic amblyopia	R-G
Methyl alcohol poisoning	R-G

Table 3: Examples of acquired color vision defects in eye disease [18]

V. Conclusion

In this chapter, we discovered the color blindness disorder and we have seen how much the individual suffers in their everyday life.

It was shown what are the various and different reasons that may cause color vision deficiencies especially for the acquired one, with learning the different between the congenital and the acquired. Thus, the causes of the acquired CVD vary to ocular condition, systemic diseases, and drug-related reasons. Opening a new aspect of the different reasons that exist and result to abnormalities within the individual color vision.

Some cases are reversible such as the acquired one with a reducing effect, in the other hand the congenital one can't be cured or reduced until this day, due to its existence from birth.

Chapter II: Classification of Color Vision Deficiency

I. Introduction

Color blindness caused by many different reasons, is classified according to the number and type of functional cone systems present in the retina, resulting to four main categories: trichromacy, anomalous trichromacy, dichromacy, and monochromacy. In addition to, within each category, a specific type of deficit that is identified by which cone is abnormal, absent, or functionally altered. The purpose of this chapter is to outline the main categories and types of color vision deficiency, with describing each condition and the resulted manifestation in color vision.

II. Categories

1. Trichromacy

The retina of the human eye, developed after a few week of his/her birth to consist three types of receptors (cones), giving him/her the opportunity to identify three essential colors Red (long wavelength), Green (medium wavelength), Blue (short wavelength) and mix it in a variou combination which is called trichromacy.

To sum up, trichromacy is the existence of the three types of cones without any anomaly or absence so it gives each individual the ability to have a normal color vision [1], [21].

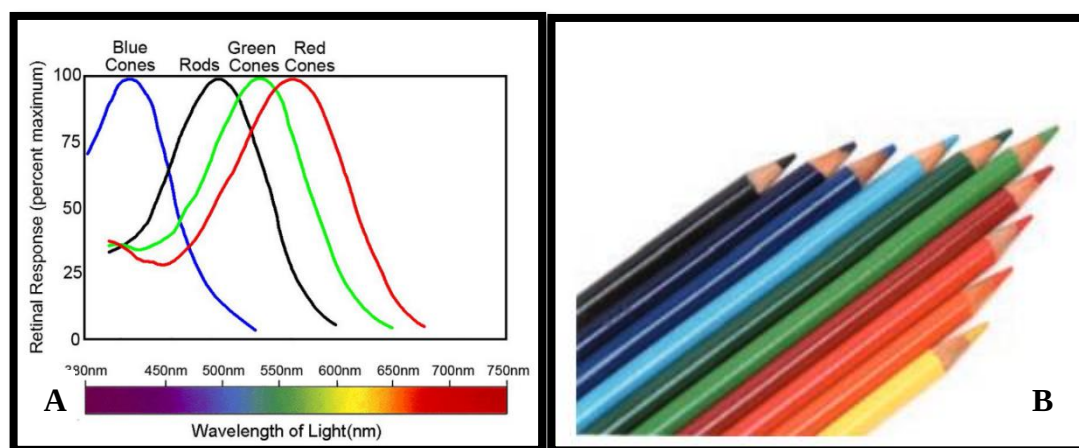


Figure 6: (A): The figure illustrates the normalized spectral absorption peaks of the four cells [7], **(B):** Trichromacy, a normal color vision [18]

2. Anomalous trichromacy

One of the most common categories in color blindness, which is abnormal trichromacy. It is explained as the existence of an irregular cone, i.e. one of those pigments that obtains an altered response sensitivity curve. Depending on which cone pigment is

affected of those three, we have three forms of anomalous trichromatic vision, and it has been termed as protanomaly, deuteranomaly, and tritanomaly [22].

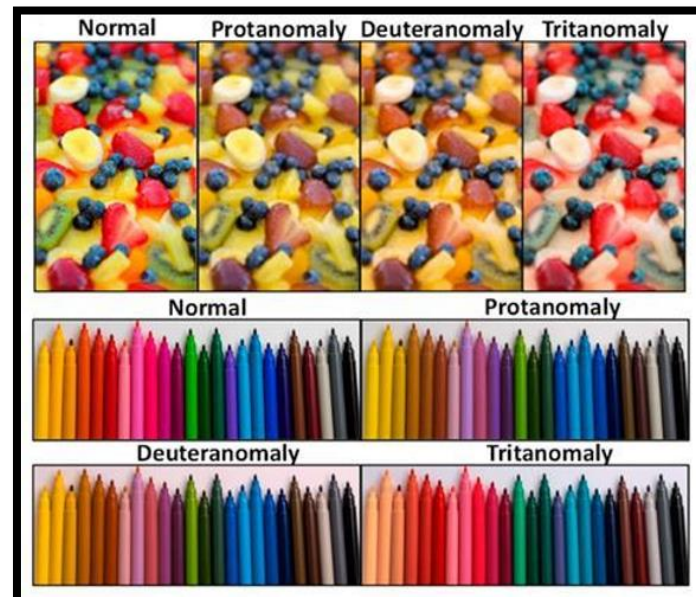


Figure 7: Simulation of Anomalous Trichromacy' types [63]

3. Dichromacy

It is known as the absence of one of the cones, which leave us with two other cones, in other term dichromatic vision. In addition, according to the missing cone system long wavelength, medium wavelength, or short wavelength, we can determine the three types of color dichromacy, and it is termed by protanopia, deuteranopia, and tritanopia respectively. Alas, due to the presence of only two cone pigments, color discrimination will be poor and color perception will be distorted and the patient will suffer from color dichromatic vision [22].

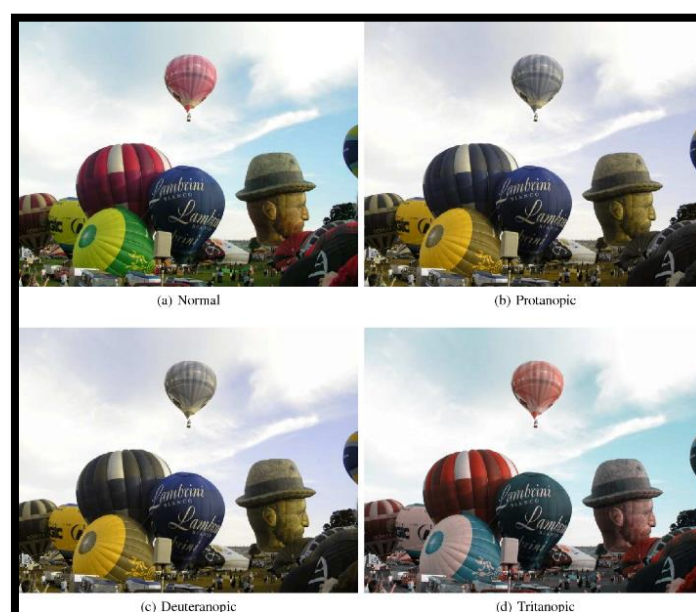


Figure 8: Simulation of normal color vision and dichromacy types [67]

4. Monochromacy

It is referred to as the total (complete) color blindness or achromatopsia. Which means the patient experience difficulties to identify all types of colors in the visible spectrum, resulting a world dressed in dark, white and dim, along with a high sensitivity to light and glare. Along with the distinction between its two types, which are Short cone monochromacy and rods achromatopsia. Furthermore, it is mostly an inherited ocular disease and one of the rarest types of color blindness [3], [23].

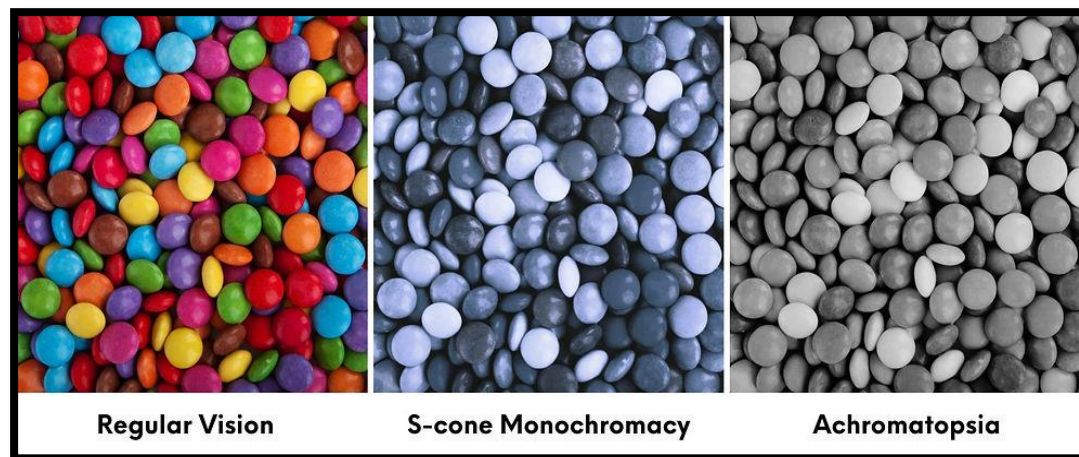


Figure 9: Simulations of Monochromacy types [62]

III. Types of color blindness

1. Red - Green color deficiency

RG color blindness is widely a genetic recessive condition, also a spread one. Identified as a disorder/absence in Long or Medium wavelength cones in the retina, resulting an inability in distinguishing between the colors red and green [23].

1.1. Protanomaly

Potanomaly or red color deficiency, identified as an anomaly in the long wavelength cone pigment. Resulting the patient to see red looks more green in color [3].

1.2. Protanopia

The absence of the long wavelength cone, responsible for the red color. Resulting the individual to be unable to distinguish between red and purple, color seems to be more blue, and it is mostly spread between women [23].

1.3. Deuteranomaly

With the presence of an alerted response sensitivity curve in the Medium cones (pigment responsible for the green color), the patient will see the green color looks redder [3], [23].

1.4. Deuteranopia

The lack of the middle wavelength cone in charge of the green color, affect the eyes of the person to regard the green color same as the red color [23].

2. Blue – Yellow color deficiency

BY color blindness described as anomalous/absence in the Short wavelength cones (blue cones pigment), resulting a difficulty to see the blue color. This kind of disorder grows with aging, exposure to UV light, or the problem that comes with birth [23].

2.1. Tritanomaly

The abnormality of the existed Short cone wavelength (blue color), gives a hardship to the individual to tell the distinction between blue – green and yellow – red [3].

2.2. Tritanopia

Missing the short cone wavelength (blue color), leads to a difficulty to differ between green – purple – red and yellow – pink [3].

3. Monochromacy or achromotopsia

As already mentioned, it is characterized by the inability to recognize any kind of color existing in the visible spectrum, affecting the daily life of the person badly [23].

3.1. Short cone monochromacy

In this, the two cone cell colors (long and middle cone pigment) are not functioning. Individuals with short cone monochromatic disorder struggle with recognizing color on the ground. Their mind needs flags from different cones to understand the shading plus to the suffering from photophobic[3].

3.2. Rod monochromacy achromotopsia

It is present from birth and doesn't have any of the pigments found in cone cells. Unfortunately, this disorder give his individual the cruelty of seeing the world in black [3].

Deficiency	Cones affected	Symptoms
Anomalous trichromacy		Reduced sensitivity to the color corresponding to the cone wavelength.
protanomaly	L	Reduced sensitivity to long wave length (red)
deuteranomaly	M	Reduced sensitivity to medium wave length (green)
Incomplete tritanopia (tritanomaly)	S	Reduced sensitivity to short wave length (blue)
Dichromacy		Inability to see color of the missing cone type.
Protanopia	L	Cannot see long wave length (red)
Deuteranopia	M	Cannot see medium wave length (green)
tritanopia	S	Cannot see short wave length (blue)
Monochromacy		Inability to see any color
L-cone monochromacy	M & S	Inability to see any color
M-cone monochromacy	L & S	
S-cone monochromacy	M & L	
Rod monochromacy & incomplete achromatopsia	L & M & S	

Table 4: difference in affected cones and symptoms among different types of color vision deficiency [24].

IV. Conclusion

Color vision deficiencies is represented by a diverse of conditions unified by the interruption of normal trichromatic cone function. Moreover, understanding that the classification is crucial for a correct interpretation of color vision test' results and to learning how to distinguish between congenital and acquired color vision deficiencies during clinical practice.

Chapter III: Evaluation and Management of Color Vision Defects

I. Introduction

The diagnosis and management of color vision deficiency include various clinical tools and therapeutic approaches each adapted to the nature and severity of the condition.

The present chapter shows the available various diagnostic methods clinically, which vary from rapid screening tools to arrangement and matching tests, as well as objective electroretinography methods. Besides these, for the treatment approaches it includes filtered lenses, gene therapy and digital assistive technologies that were developed to support each individual's needs.

II. Diagnostic methods for color blindness

As we know, methods for diagnosing color blindness are numerous and can be categorized to screening tests, such as pseudo - isochromatic plates, which are created for a quick and reliable identification of a color vision defect. Grading test i.e. arrangement's tests to determine the severity of the disability (FM 100, D - 15). And the classification of the defect (anomaloscope) with the diagnostic tests (matching), plus the vocational tests used in clinical practice such as lanterns which are used neither for screening nor scoring but rather replicate an environment that an individual could encounter at work [18].

1. Pseudo - isochromatic plates

- **Ishihara's test**

Ishihara's pseudo – isochromatic plates can identify red – green deficit, with the design been built on the plates of stilling and being named after Dr. SHINOBU ISHIHARA, who introduced the test in 1917. The figure and background are made up of distinct discs that vary in size and luminance, allowing the figure to be only determined by its chromatic variation from the background and not from a difference in the perceived luminance.

The test is performed at 66 cm, with the combination of 38 plates, and giving the patient a four-second for the detection of the numerals or pathways. Moreover, the plates are categorized into transformation, vanishing, hidden and classification plates. With each, one having its own method for identifying specific color vision-related deficiencies.

Therefore, Transformation plates reveal different figures to individuals with color deficiencies compared to those with normal trichromatic (NT) vision, due to certain chromaticities aligning with dichromatic confusion lines. Vanishing plates are invisible to NTs as they utilize colors from specific confusion zones. Hidden plates present figures unnoticed by NTs, as they can discern chromatic variance that those with red-green defects cannot. Classification plates help identify color vision defects, showing different perceptions based on whether a person has a protan or deutan defect [12], [25].

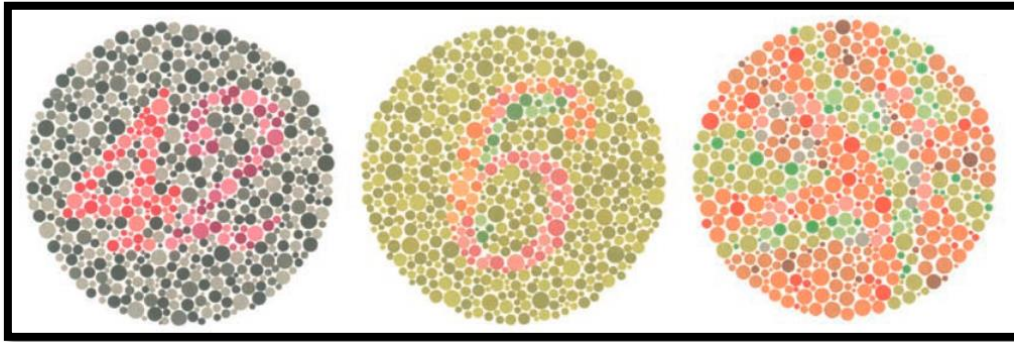


Figure 10: Ishihara' test [18]

- **AO H – R – R**

The American Optical Company Hardy, Rand, and Rittler (AO H-R-R) plates, which are not as good at determining Red-Green deficiencies but offer screening and diagnostic plate for tritan defects in addition to prognosis and grading plates for RG defects, are frequently used in conjunction with the Ishihara tests. Thus, this test detects moderate to severe tritan defects [18].

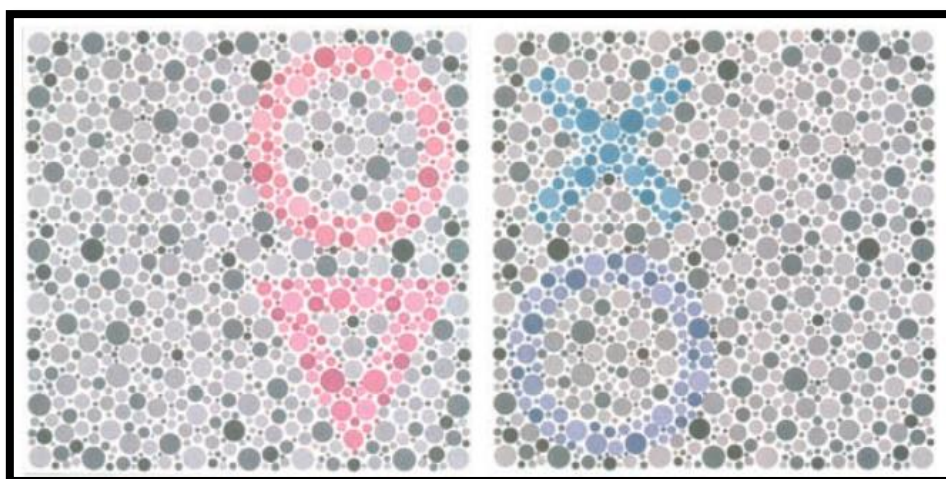


Figure 11: The AO H - R - R plates [18]

- **The Neitz test**

One of the newest methods for detecting color vision deficiencies is the Neitz test. It can uncover more deficiencies because of the identification of both Red-Green and Yellow-Blue classes of defects.

Moreover, it can be administered under diverse illumination settings, including fluorescent and daylight, and requires around 5 minutes for administration and scoring. The Neitz Test uses both vanishing and transformation designs, and there are three different variations of the test. In each one, a neutral background surrounds grayscale and colored dots, and the patient has to correctly match a figure to one of nine potential options [12].

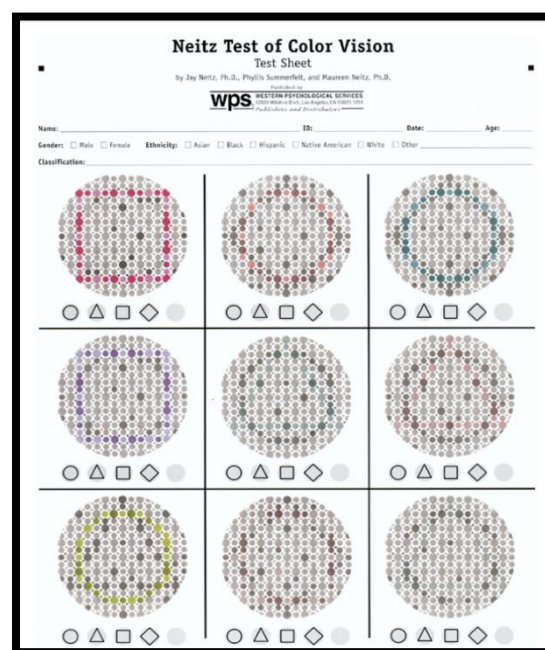


Figure 12: The Neitz test [61]

- **Color vision test made easy**

The Color Vision Test Made Easy (CVTME) is designed to screen color vision deficiencies in individuals with intellectual disabilities by adapting the procedures to be simpler. Due to insufficient comprehension of the testing requirements, patients' answers may be confusing, which makes it difficult to determine whether the patient is manifesting a color vision deficiency or merely not understanding the instructions.

The test uses vanishing pseudo-isochromatic plates with shapes familiar to individuals with intellectual disabilities rather than numbers. In addition, an initial demonstration card is provided to facilitate comprehension and matching, with the purpose of clarifying the instructions and assessing the patient's ability to follow them. The CVTME is intended to detect malingering and random guessing, thereby ensuring the reliability of the results [12].



Figure 13: The Color Vision Test Made Easy [61]

- **The city university test**

The city university test (CUT) was created to reduce the limitations with the D-15 test. This assessment tool contains plates designed to determine protan, deutan and tritan deficiencies. The second of the test comprises 10 pages, each with a central reference color circled by four testing colors. The subject is instructed to select the closest and most similar to the central reference.

Amidst the four surrounding colors, three align with dichromatic confusion lines corresponding to protan, deutan and tritan defects respectively. On the other hand, the fourth color is chosen to be noticeably distinct from the reference for a color normal subject.

The choice of different color combinations in the city university test (CUT) is based on the D-15 test, which allows the CUT to function as an alternative to it.

Nonetheless, the problems with the CUT include the possibility of none of the tests colors are closely similar to the reference color. Moreover, the individual has to physically point to the closest color, it present a challenge to the disabled patients [12].

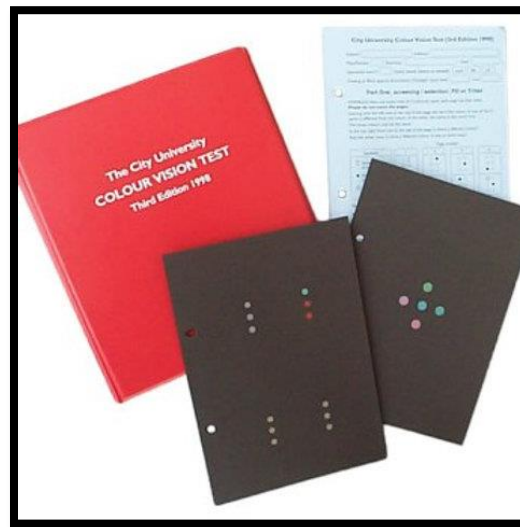


Figure 14: The city university test (CUT) [60]

2. Matching tests

- **Anomaloscope (Nagel Rayleigh / Pickford – Nicholson/ Besancon)**

An anomaloscope serves as a diagnostic instrument for assessing color vision, functioning on the principle of color matching. It is widely regarded as the clinical gold standard for the diagnosis and classification of congenital color vision deficiencies.

Moreover, **the Nagel** (model 1) anomaloscope one of the most accurate and common tests, apply the Rayleigh equation to determine Red-Green color vision deficiencies. This device assesses an individual's Rayleigh matches by determining the specific proportions of red and green light necessary to match a yellow stimulus presented during testing, thus facilitating the identification of red-green color deficiencies. The test involves a vertically divided field with the upper half displaying a mixture of red and green lights, and the lower half-showing yellow light. Both the red-green mixture's proportions and the yellow light's luminance are adjustable, allowing the subject to manipulate these parameters until both halves appear identical.

On the other hand, for a blue defects to be diagnostic, alternative anomaloscopes such as the **Pickford-Nicolson** or the **Besancon** anomalometer are sometimes needed [12], [18].



Figure 15: The Anomaloscope [59]

3. Arrangement tests

- **FM 100 hue**

The Farnsworth-Munsell 100-Hue test (FM 100-Hue), is produced to evaluate hue distinction between individuals with normal color vision and to measure color discrimination loss in patients with congenital and acquired color deficits [18].

The test include four boxes of 21 to 22 caps, with specific hue of paper is mounted to each cap, making a total of 85 colored caps [18].

The FM100 is presented at 50 cm (approximately an arm's length) to the patient with proper illumination. Thus, the subject is instructed organize the caps in order according to color, from first to last. At the end of the test, each result will be recorded on a score

sheet by comparing to the correct arrangement. A higher the score mean an abnormal color discrimination ability [18].

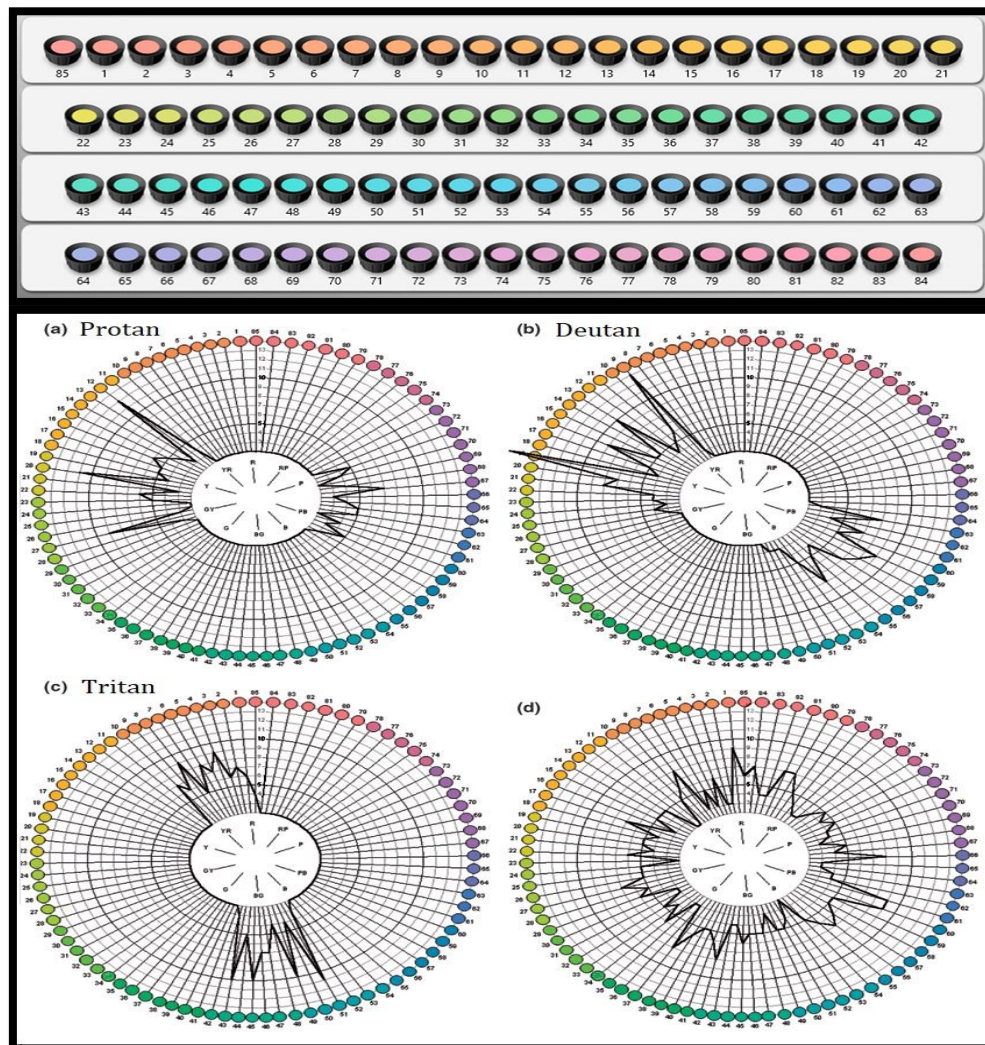


Figure 16: The FM 100 Hue Test [57]

- **D-15 saturated**

The Farnsworth Panel D-15 test is used to identify Red – Green and Blue – Yellow deficits. The test consists of a box with 15 caps, one of each installed with a specific hue. The individual is presented with the test on 50 cm distance, under Standard Illuminant C providing at least 250 lux [18].

Moreover, one pilot cap is fixed at the end of the box. Hence, after randomly placing the caps, the subject is instructed to arrange each cap in an order that seems normal, from first to last [18].

Thus, the arrangement is noted on a special sheet result, with the correct arrangement follows the semi-circular pattern of the graph. Errors show up as crossed lines across the circle. Regularly, a one notable crossover in observed. Protans, deutans, and tritans

make various important crossovers that create a nearly parallel series of lines. The axis of these lines decide the type and the nature of color vision deficits. In addition, if the subject make few minor mistakes, a retest is recommended [18].

However, the test is not effective for screening mild and weak color vision deficiencies, still is a rapid and efficient tool to identify severe color vision abnormalities [18].



Figure 17: The Saturated D-15 test [58]

- **D-15 desaturated**

Lanthony Desaturated Panel D-15 Test (Desaturated Panel D-15) is designed to supplement the Farnsworth Panel D-15. Both tests are alike; nevertheless, the Desaturated Panel D-15 used paler and lighter colored caps compared to the Saturated D-15 test, making the test harder for even the simple anomalous trichromats [18].

Thus, the test is administratively identical to the Saturated D-15. 15 caps are arranged in a natural order, with the individual being instructed to order the caps according to color. Also a scoresheet same as the Saturated D-15 is used to interpret the results of the patient [18].



Figure 18: The Desaturated D-15 test [66]

The Desaturated Panel D-15 was originally created to detect acquired color vision deficits and simple anomalous trichromacy that passed the Saturated D-15, which makes it more precise and accurate than the latter [18].

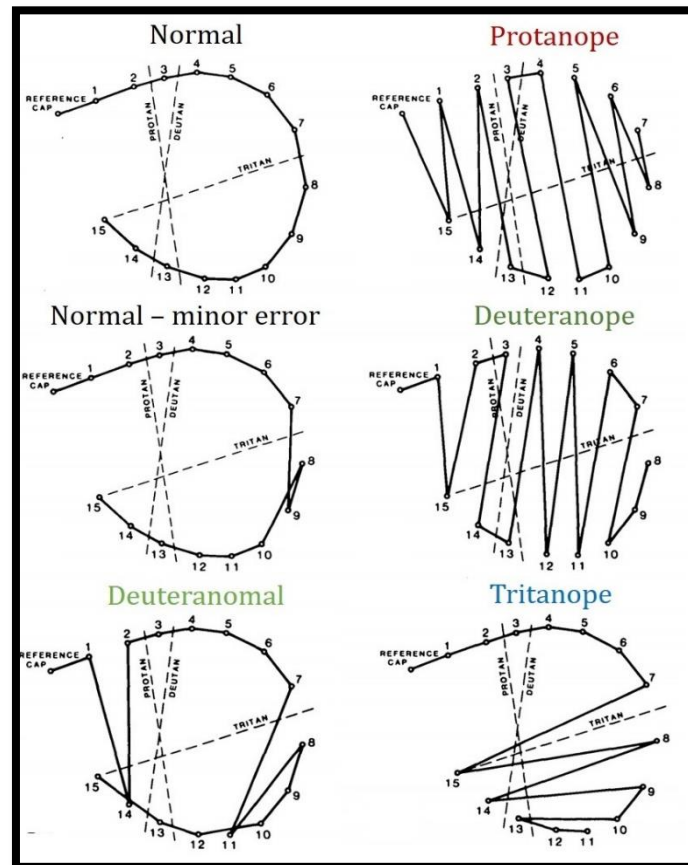


Figure 19: The Desaturated and Saturated tests 'sheet results' [58]

- **Lanthony New Color Test**

The new color test permits the detection of neutral zones (colors mixed up with gray), while also testing the color distinguish ability at four various saturation levels [18].

The test includes four boxes containing 15 colored caps each. While the saturation varies, the hues are all the same in the four boxes, in addition to 10 gray chips of varying lightness [18].

Importantly, this test was especially designed to precisely identify and diagnose the acquired color vision deficiencies [18].

4. Vocational tests

- **Lanterns**

Lantern tests are tools to evaluate the color vision suitability for occupational reasons, particularly in domains such as maritime, aviation, and railway industries. The test is

designed to mimic real life scenarios by displaying one or two colored lights from a distance of 6 meters, requesting from the individual to simply name and identify the color stimuli.

In addition, the number of errors made in recognizing colors determines the pass/fail criteria. Research has shown that the test reliably excludes all dichromats and about 75 % of anomalous trichromats.

Originally, lanterns did not include blue (tritan) stimuli, and thus tritan-related color vision deficiencies are generally not addressed.

To simplify, lanterns are not typically complex, it only demands that the subject name the colors that they see. Two types exist, lanterns that give out single colors and those that give paired colors signals. The accuracy of the lanterns depends on the timing and order in which the colors are presented. In general, the test is not designed for detailed clinical diagnosis or classification of color vision anomalies; even though patients with color vision deficiencies tend to make characteristic mistakes during the test [12], [18].



Figure 20: Lanterns [56]

5. Computerized tests

• Cambridge Color Test

The Cambridge color test (CCT), a computerized color vision test present an advantage of the possibility of adjusting the difficulty that depends on the performance of the individual, along with putting the plates randomly to prevent the patient from remembering formerly seen plates.

Moreover, the CCT design employ a computer adaptation of pseudo-isochromatic plates and the combination of the Principles of Chibret [12].

In addition, the test include a Landolt C, with each stimulus consists of a big colored C defined by a specific hue and luminance portrayed in four different orientations: with the opening directed upward, downward, to the right, or to the left. This letter stimulus is embedded within a background composed of circles that vary in size, color, and luminance. Thus, the background presented in the CCT stimuli exclude any luminance or contours cues, and the figures (the test stimuli of Landolt letters) must be detected only by their hues. The process is combined with non-hue noise, presented by altering the luminance and size of the individual components according to Stilling's Principle. Participants respond by pressing one of four keys on a handheld device to indicate the direction of the openings in successive Landolt C stimuli, rendering the task straightforward in cognitive and simple terms [12].

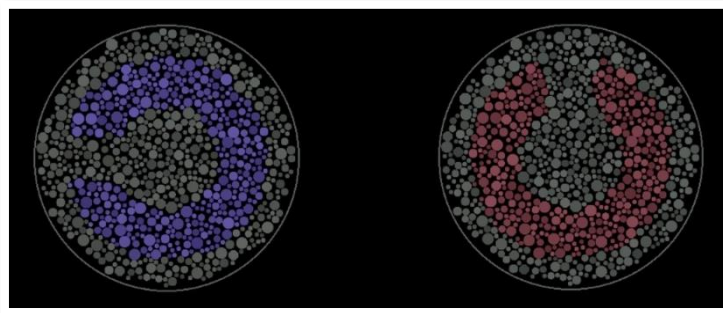


Figure 21: Cambridge Color Test (CCT) [55]

6. Electroretinography

The Electroretinography (ERG) is an objective method to detect color vision defects, unlike the various types of color vision tests that depend on the individual's subjective perception of colors.

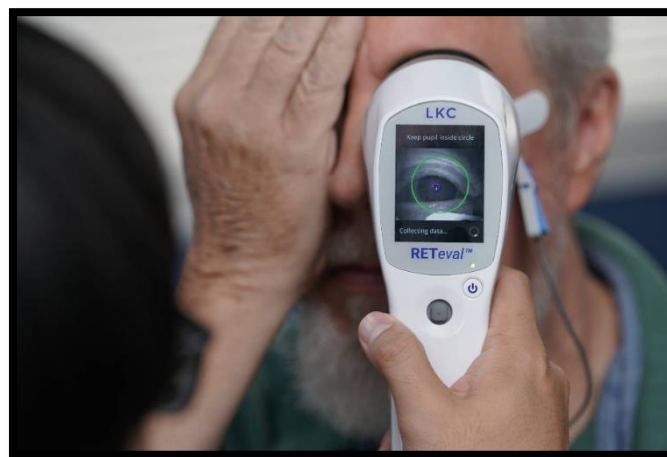


Figure 22: Electroretinography (ERG) [54]

Within the ERG, it utilizes a corneal electrode positioned on an anaesthetized and pharmacologically dilated eye to capture the retinal responses elicited by controlled light stimuli. Color vision defects characterized as deutan and protan can be discriminated by the log ratio of the sensitivity at short (480 nm) and long (620 nm) wavelengths (sensitivity quotient) [12].

7. Holugen wool test

The first commercially available test of color vision, it is a primarily and a historical one, with the test consisting wool skeins of different colors.

Moreover, the patient is introduced with a pile of different colored wool skeins. The individual is to choose the skeins from the pile that are equal to the test skein presented by the clinician. The test does not include standardized scoring guidelines [18].



Figure 23: Holugen wool test [53]

III. Different approaches for treating color blindness

1. Tinted/Filtered Lenses (Glasses & Contacts)

For treating color blindness deficiencies, there are special lenses that are coated and covered with various treatments to selectively filter specific wavelengths of light. The purpose of these inventive lenses is to improve and boost color perception and the full visual encounter for subjects with Red-Green color blindness.

Moreover, besides the filtered glasses, dyed contact lenses have been formed to enhance the color perception of patients, offering benefits of non-toxicity, economic efficiency, and adaptability to specific requirements.

In addition, other studies demonstrated the possibility of contact lenses produced from gold nanoparticles (GNPs) and hydrogel matrix to have the ability to enhance color vision.



Figure 24: Color Blind Contact Lenses [52]

Currently, Enchroma is leading in the color blindness market, which originally launched its products in 2012. Thus, a multi-notch filter is applied to the glasses to erase the overlap of red and green, using Trivex as the lens material. Because it is lighter, thinner, and stronger than commonly used glass materials (CR-39 and polycarbonate), by covering the surface of the lens with a dye with a limited absorption band to prevent impairing the patient's typical color perception.



Figure 25: Enchroma color blindness glasses [51]

However, Blue-yellow blindness has no effective solution. In general, the clinical results of assistive technology, such as glasses or contact lenses have been unsatisfactory. For the reason of the S-cone making, only 5% of all the cone cells, blue-yellow color blindness is relatively mild and has little impact on patients. As a

conclusion, there is a deficit of related research. Instead of designing effective treatments, current research focuses primarily on studying the underlying disease mechanisms [26].

2. Gene Therapy (Experimental — Most Promising)

The gene therapy mean to modify the expression of human genome with the purpose of treating inherited diseases, cancer, and infectious diseases. This approach can be used through many processes: silencing a dysfunctional gene, adding a new or modified gene to cure a disease, and overriding a harmful gene with a correct, healthy gene copy.

Furthermore, it was applied by using human gene modifying technology, viral vectors, bacterial vectors or patient-derived cellular gene therapy products.

In addition, reports about restoring the operational and physiological characteristics of cone system, was established by photopic electroretinograms (ERGs), with subretinal injections.

Restoration of the functional and physiological characteristics of the cone system was reported, as proven by photopic electroretinograms (ERGs), with subretinal injections. Thus, considering the positive results maintained for at least five months under treatment, it was stated that the effect is lasting.

To simplify, gene therapy is an interesting new treatment choice for patients with color vision defects. Hence, the three most common gene of achromatopsia are; CNGB3, CNGA3 and GNAT2, have been the focus of several experimental studies in animal models, as well as a small number of phase I/II clinical trials.

On the other hand, the focus was also on L-opsin and M-opsin gene. An improvement was proved to be durable and maintained in cone cell functionality as illustrated by ERGs and visually elicited behavior. Additionally, it has been shown that intravitreal (intravitréenne) injections of the therapy's genes may be a safer option than sub-retinal (sous-rétine) injections [15].

3. Digital Assistive Technologies

- **Color Blind Pal app**

The Color Blind Pal app is an application to support students with color vision defects. It permits students to aim their cell phone's camera at anything, freeze the screen and change the object on the screen, and receive a text readout of the target's color.

Additionally, the app is accessible on Mac OS for a fee. Hence, the Color Assist Lite app is a substitute technology for the Blind Pal app, with the availability only for Apple products.

Since many students had smartphones but not all of them had Apple devices, it was decided to use the Color Blind Pal app, wanting the application to be accessible to all students in the course.

Moreover, the application also provides individuals with normal color vision the ability to adapt the colors in the camera to mimic different color vision deficiencies. While drafting the curriculum, it was discovered to be peculiarly interesting to view the laboratory assignments from the perspectives of students having color vision deficiencies [27].



Figure 26: Color Blind Pal App [27]

- **Augmented Reality (AR) for color recognition**

Smartphone-based Augmented Reality (AR) application provides new assistance for patients with color vision deficiencies. The crucial purpose is to enhance the 2D color perception of individuals with dichromacy by including an extra dimension of information. Thus, promoting the encoded in a 3D space for dichromats.

The extra dimension is elicited throughout the temporal modulation of colors: the subject interacts with the application by swiping using their fingers. An applied transfer color-space transformation, results in originally unclear colors undergoing distinct color shifts. Therefore, the match of the original two-dimensional color perception with the provoked temporal shift reconstructs a new three-dimensional perceptual space for the patients.

Profoundly, the innovation of a three-dimensional perceptual space allows individuals to comprehend how to identify colors. Over time, operating on the system patients tend to develop an intuitive understanding of how initially confusing colors sustain temporal color shifts. Then, individuals understand how to associate different color names with varied shifts, thus identifying and naming colors.

In addition, it was offered to improve and establish a way that permits patients with dichromacy, whose color vision is originally two-dimensional, to identify perplexing colors. The essential is to trigger an additional dimension throughout the temporal color shifts, while, as well as the primary 2D perception of color, positions colors in a novel 3D space where subjects can determine and distinguish colors.

Moreover, according to psychophysics and a longitudinal study, it was confirmed that the temporal color shift has selective ability and provokes a regular design that can be understood by individuals. Thus, the AR interface was applied on smartphones, which is simple to use and supports patients with color vision deficiencies, identifying colors in two real-world scenarios (building with Lego blocks and interpreting artistic works) [28].

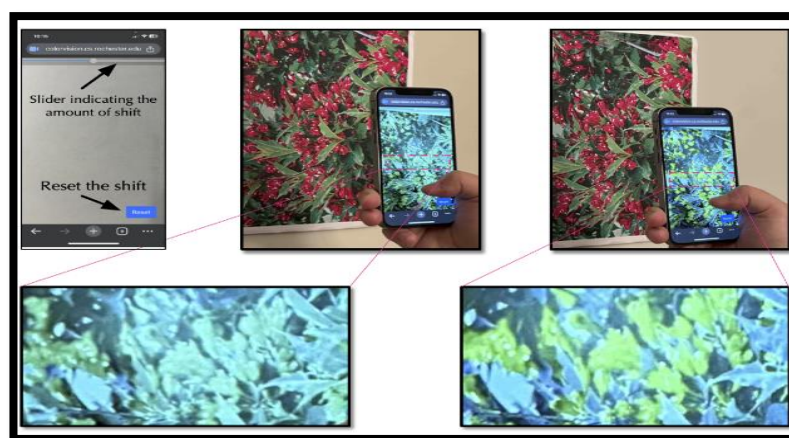


Figure 27: User swipes the finger to shifts colors of objects in the physical world [28]

- **Color enhancement algorithms on smartphones**

The research analyzes color enhancement algorithms built on the neural mechanisms of color deficiencies and color blindness. Thus, the algorithm is employed on smartphones to augment the user experience (UX) of color-enhancing attributes in various top-selling smartphone brands with diverse operating systems (OS).

Moreover, an application program designed for color enhancement was created specifically for individuals experiencing color vision deficiency, covering three distinct features: daily life, information visualization, and videos.

In addition, it was shown to be advantageous for patients with color vision deficiencies using the color-enhancing features when using color-enhancing features, as these provide strong color contrast. Thus, the user preferences are closely linked to perceived color naturalness.

Furthermore, the app program assists individuals with color vision deficiencies to differentiate between colors, while maintaining a great color naturalness level and high color preference.

Research proved that the color-enhancing application program has a positive influence on color vision impairments; by raising, the level of contrast patients with color vision deficiencies could identify color-related information more clearly [29].

IV. Conclusion

As a conclusion, many different tools to diagnose color vision deficiencies has evolved noticeably making it easier to clinicians to interpret CVD more smoothly and precisely.

On the therapeutic side, while assistive solutions such as tinted lenses and digital technologies offer practical support, effective treatments remain limited, and gene therapy is still in the early stages of clinical application.

Practical section

Clinical study of acquired color vision deficiency

I. Introduction

Acquired color vision deficiency considered as frequent functional disorder in patients with ocular and systemic diseases. Thus, the purpose from this work is to interpret and discuss the results obtained from the color vision study of patients present with different ocular, systemic, and drug-related causes, utilizing the dual D-15 protocol combining the Desaturated and Saturated Farnsworth D-15.

Results and discussion included the general characteristics of the sample, the etiological distribution of acquired color vision deficiency, the testing protocol, and the types of color vision deficits related to pathological condition.

In addition to, analyzing and comparing each resultant of the study with available published literature to prove that the results of this research was confirmed. Finally, limitation of the study was also included as any other study with the addition of perspectives that open future research.

II. Objective

Acquired color vision deficiency is widespread among patients with ocular diseases; it often remains underdiagnosed due to the limited availability of standardized testing materials.

The main purpose of this study is to classify the types of acquired color vision deficiency according to their origin and pathology.

It also aims to evaluate the severity of the CVD associated with different ocular pathologies, systematic diseases, and toxics or drug-related causes.

To sum up, the purpose of the examination is:

- Identify acquired CVD types according to etiology.
- Detect underlying pathologies at a pre-diagnostic stage.
- Demonstrate progression phases of color vision loss/regain in relation to disease mechanisms.

III. Methodology

1. Study design

This research represents a cross-sectional analytical study conducted over a three months' period (from February 04th to May 06th), with partial retrospective data collection.

2. Study settings

The study was conducted in the public health establishment ‘‘ EHS Ophthalmology - A- Hammou Boutlelis ex-Couniot ‘’ in Oran state, Algeria, under the supervision of Pr. DERDOUR.

3. Study population

40 patients with different ocular condition, systematic diseases, and drug-related causes were examined over a period of three months. The age of all participants ranged from 18 to 77, with an equal distribution of 20 males and 20 females.

4. Inclusion / Exclusion criteria

4.1. Inclusion

- The patients met the following criteria, leading to their inclusion:
- Age ranging between 18 and 80 years old,
- Patients with confirmed or suspected ocular pathology, systemic disease, or drug-related condition.
- Cooperative patients, with the ability to arrange caps.

4.2. Exclusion

- The patients met the following criteria, leading to their exclusion:
- Children and old people who cannot understand the principle of the tests.
- Non-cooperative patients.
- Patients with non-diseases that affect color vision.
- Patients with congenital color vision deficiencies.

5. Clinical examination

Data was collected during routine ophthalmology consultations using a standardized protocol. Clinical assessment included evaluation of various pathologies. Additional

data on patient history and disease progression were obtained from clinical records and patient interviews.

5.1. Test' conditions

- The examination was performed in the well-lit consultation room, with artificial light equivalent illumination to daylight to ensure accurate color perception.
- The color vision of the patient was examined using Farnsworth Panel D-15 and the Desaturated Panel D-15, consisting of 15 caps arranged correctly, so the patient can figure the principle of the test.

5.2. Procedure

- Each participant was first examined by the ophthalmologist, to check the presence of ocular diseases, including visual acuity and fundus examination,.
- After confirming the presence of ocular pathology, systemic disease, or drug-related condition, patients were submitted for color vision deficiencies' tests.
- Color vision was tested separately for each eye, using both tests to detect unilateral or asymmetric deficiencies.
- After seeing the correct arrangement, we requested from each individual to arrange the 15 chromatic caps according to their similarity, starting from the reference cap.
- The test took a total of approximatively 20 minutes for each patient to be examined comfortably.
- After the test, we collected each patient's clinical sheet, as well as the results sheet



Figure 28: Color Vision test procedure, (A): Saturated Bar D-15, (B): Desaturated Bar D-15

6. Statistical analysis

The statistical analysis was performed using Microsoft Excel.

7. Materials

The study was applied using two tests: The Farnsworth Dichotomous Test for Color Blindness (Farnsworth Panel D-15) and the Lanthony Desaturated Panel D-15 Test (Desaturated Panel D-15), in addition to Ishihara's pseudo – isochromatic plates (on some cases). For identifying acquired color vision deficiencies types according to their etiology and detecting underlying pathologies at a pre-diagnostic stage.

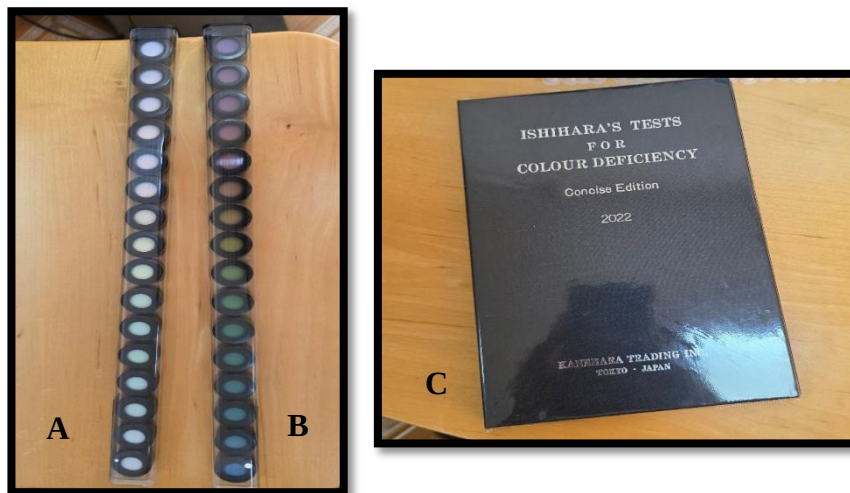


Figure 29: The used tests during the study, (A): Desaturated D-15, (B): Saturated D-15, (C): Ishihara test

Thus, a slit lamp used by Pr. DERDOUR to examine patients thoroughly. In addition to, a visual acuity scale to evaluate patients' acuity.

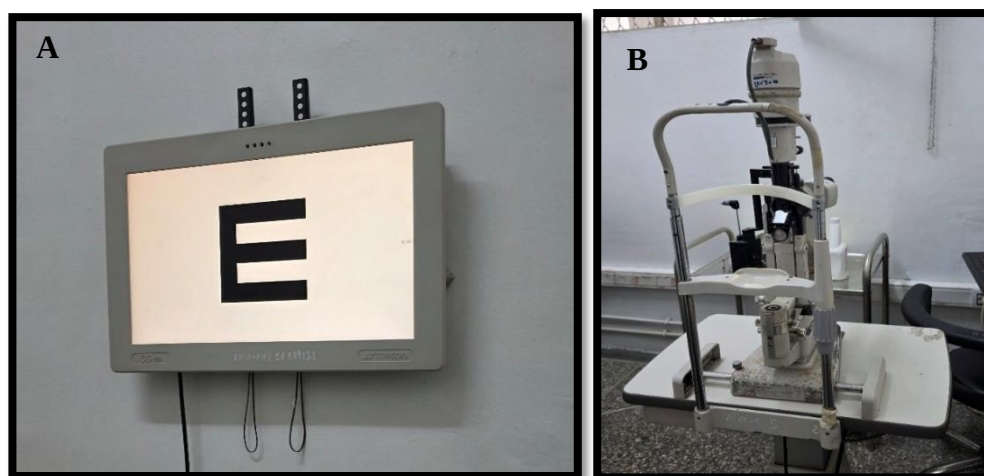


Figure 30: The Materials used during the consultation, (A): visual acuity scale, (B): slit lamp

8. Results

8.1. General data about the sample

Forty patients with various ocular conditions, systemic disease, and drug-related causes were included in the study. There was an equal distribution of 20 (50%) males and 20 (50%) females.

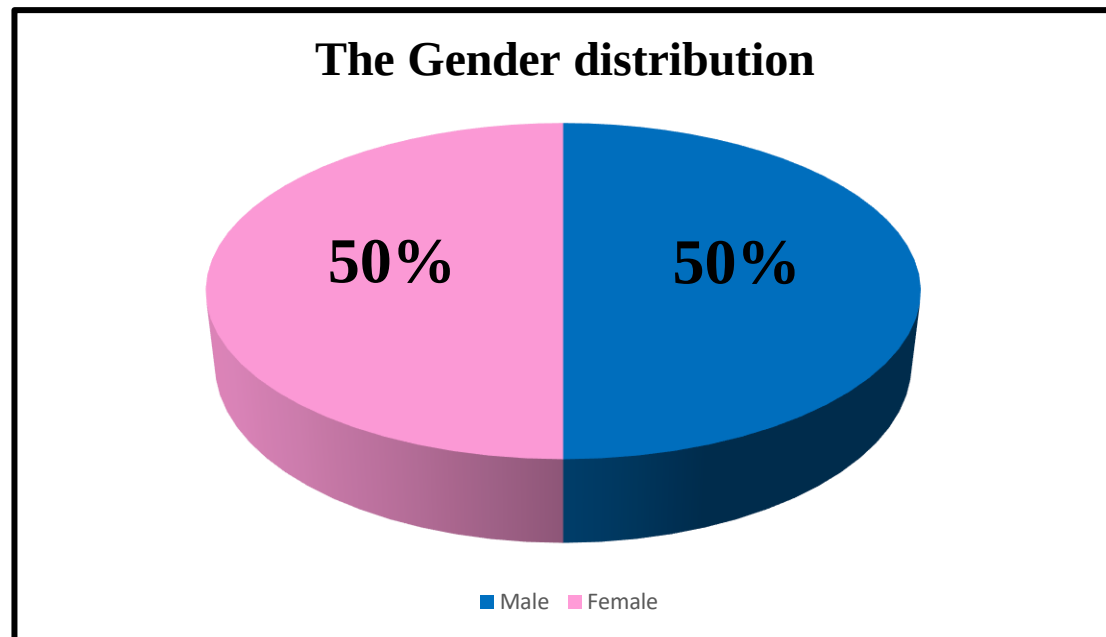


Chart 1: Gender distribution.

8.2. Specific data

A confirmed /suspected ocular disease, systemic condition or drug-related causes, was considered important for inclusion in this study.

Among the 40 evaluated patients, 38 patients (95%) were diagnosed with ocular diseases, three patients (7.5 %) with systemic diseases and one patients (2.5 %) with drug-related causes.

Most of the patients with the systemic diseases are associated with the ocular diseases. Thus, several patients presented with two or three ocular diseases in parallel. The following pie charts represent the distribution of the patients' conditions per patient and eye.

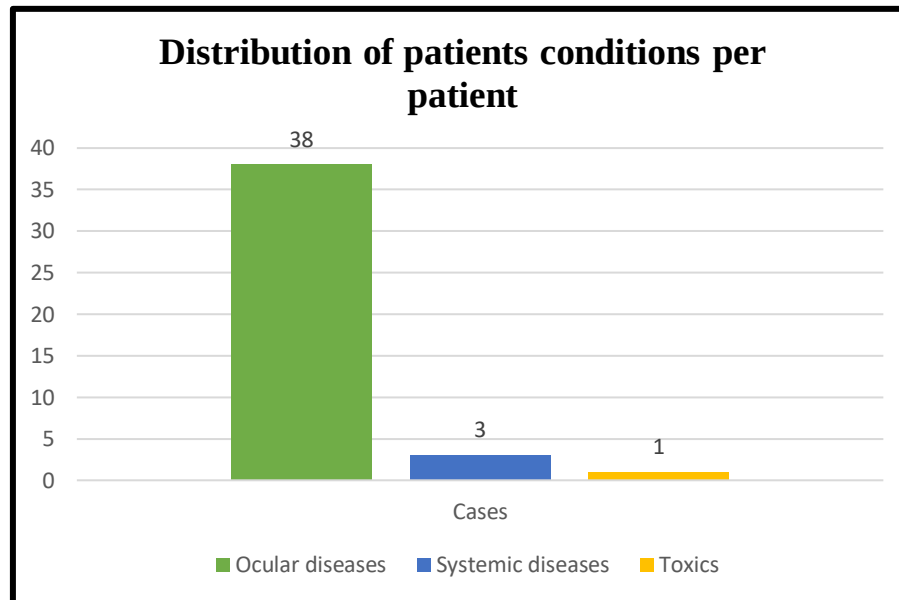


Chart 2: Distribution of patients conditions per patients

8.3. Patient pathology profile

A number of 22 different pathologies were identified among the 40 evaluated patients; several of these conditions were repeated across multiple individuals with some patients presenting one or two diseases at once.

Thus, glaucoma was the most frequent pathology with nine cases (17%), followed by six cases of diabetic retinopathy and neuropathy (12%), cataract with five cases (10%). On the other hand, systematic diseases and other ocular conditions occurred less frequently. Glaucoma and diabetic retinopathy are bilateral, on the contrary the rest of ocular pathologies such as neuropathy and cataract are unilateral. The bar chart below present the distribution and frequency of pathologies per eye.

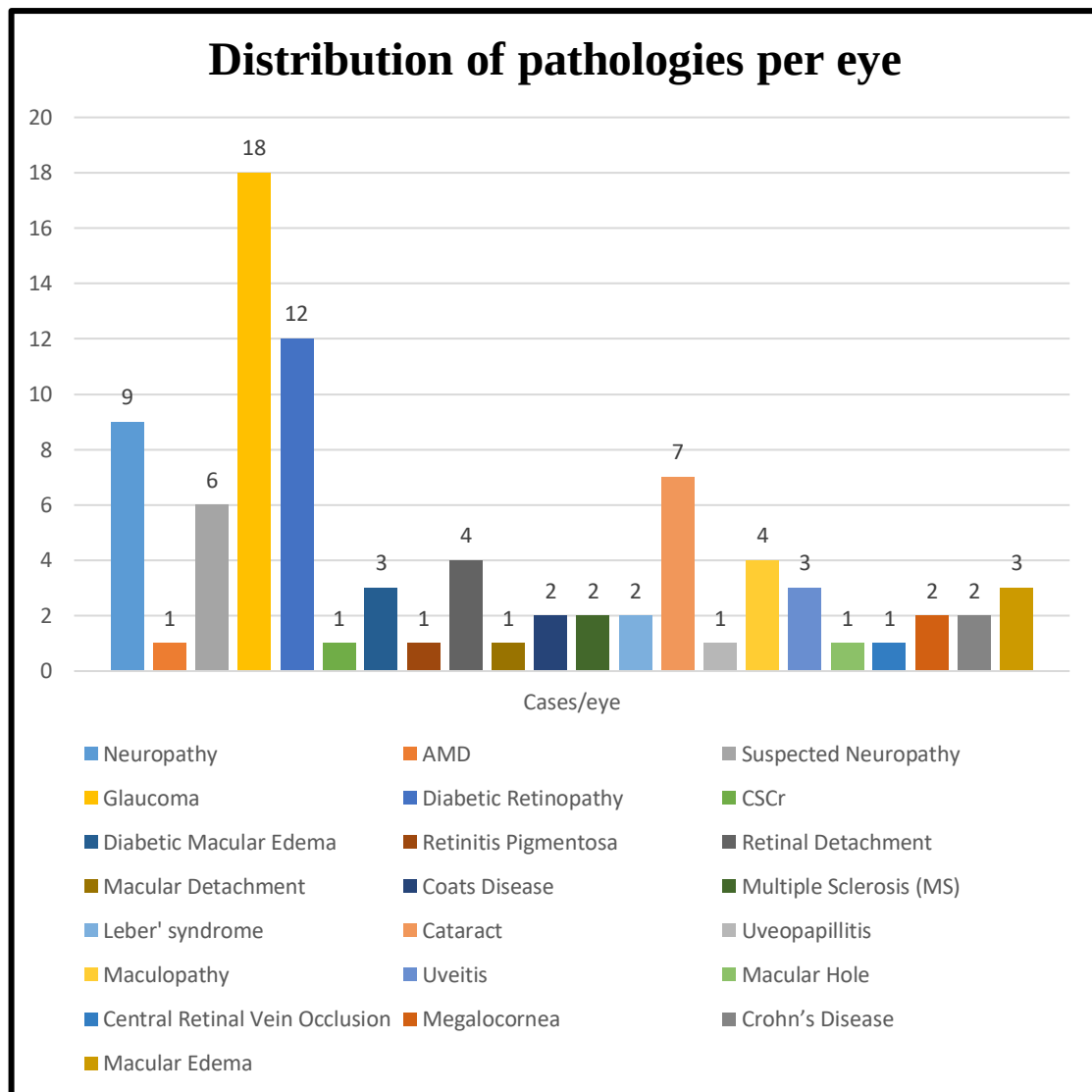


Chart 3: Distribution of pathologies per eye

8.4. Patient Analysis by Visual Color Deficiency, and Eye Condition

Out of 80 examined eye, six eyes (8%) were blind and could not be tested. Among the 74 remaining eyes (92.5), nine of them (11%) showed no pathology, 12 eye (15%) presented without CVD and 53 (66%) cases with confirmed acquired CVD. The severity of these deficits varied according to the severity of the pathology.

This analysis shows that most of the patients with pathologies were found with color vision deficiencies.

The following stacked bar chart and pie chart shows the distribution of patients according to color vision deficiencies CVD status and ocular conditions.

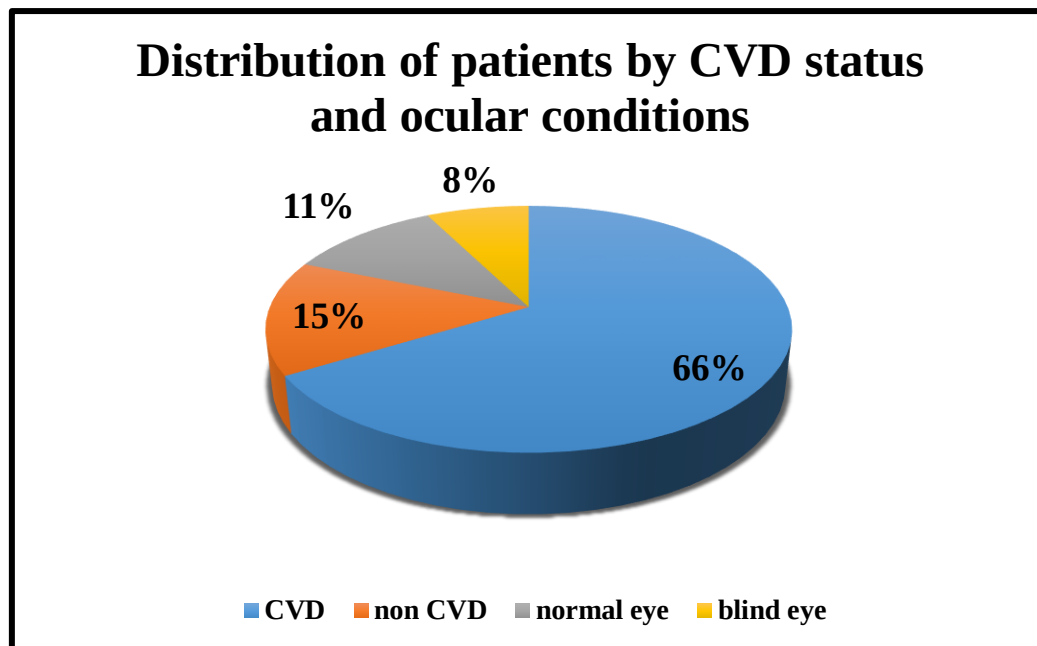


Chart 4: Distribution of patients by CVD status and ocular conditions

8.5. Etiological Distribution of Color Vision Deficiency

From the results of the distribution of the diseases origins and the corresponding color-vision testing, we obtained 64 cases with color vision defects (CVD) out of 77 cases of ocular diseases, 4 cases with CVD out of 6 cases of systemic diseases and 2 cases of color vision deficiencies out of 2 cases with drug-related causes.

The following stacked bar chart illustrate the distribution of color-vision deficiency, cases by etiological category per eye:

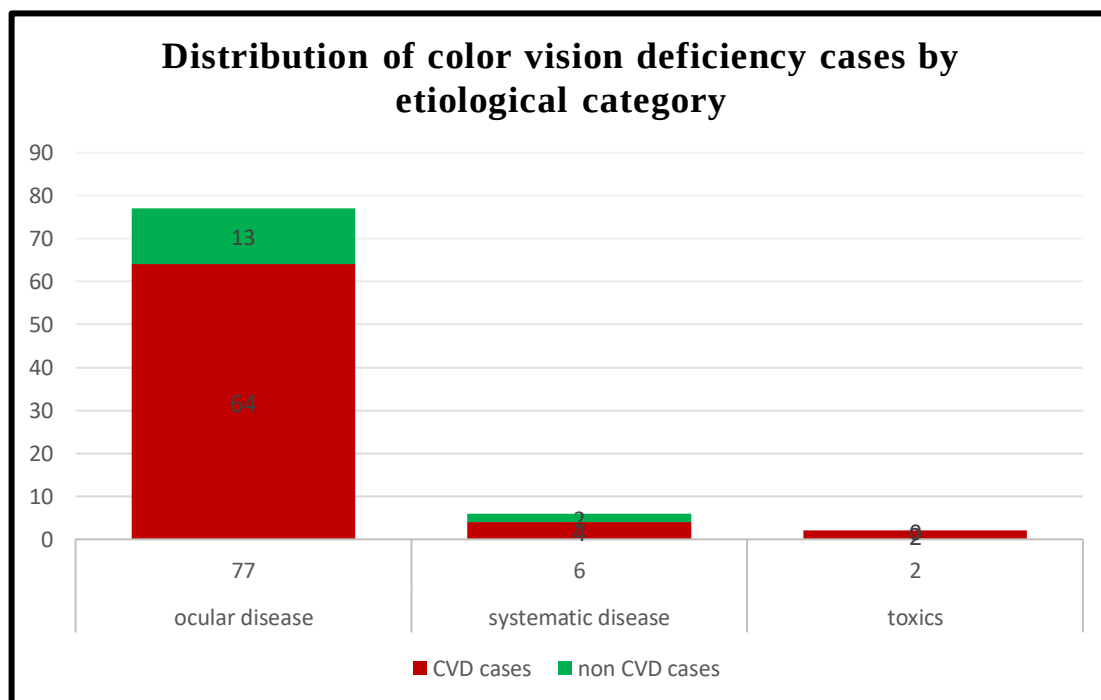


Chart 5: Distribution of color vision deficiency cases by etiological category

8.6. Distribution of CVD Patients by Pathology

After testing patients with confirmed pathologies using the Farnsworth saturated and Desaturated Panel D-15, we found:

- 16 out of 18 glaucoma cases presented a CVD, all nine patients with neuropathy showed CVD.
- 8 out of 12 cases with diabetic retinopathy exhibited color vision deficits.
- No patients with macular detachment and Crohn disease demonstrated CVD.

These findings indicate that while not all patients with pathologies develop color vision deficiency, the majority do. The chart bar below present distribution of CVD cases by associated pathologies per eye.

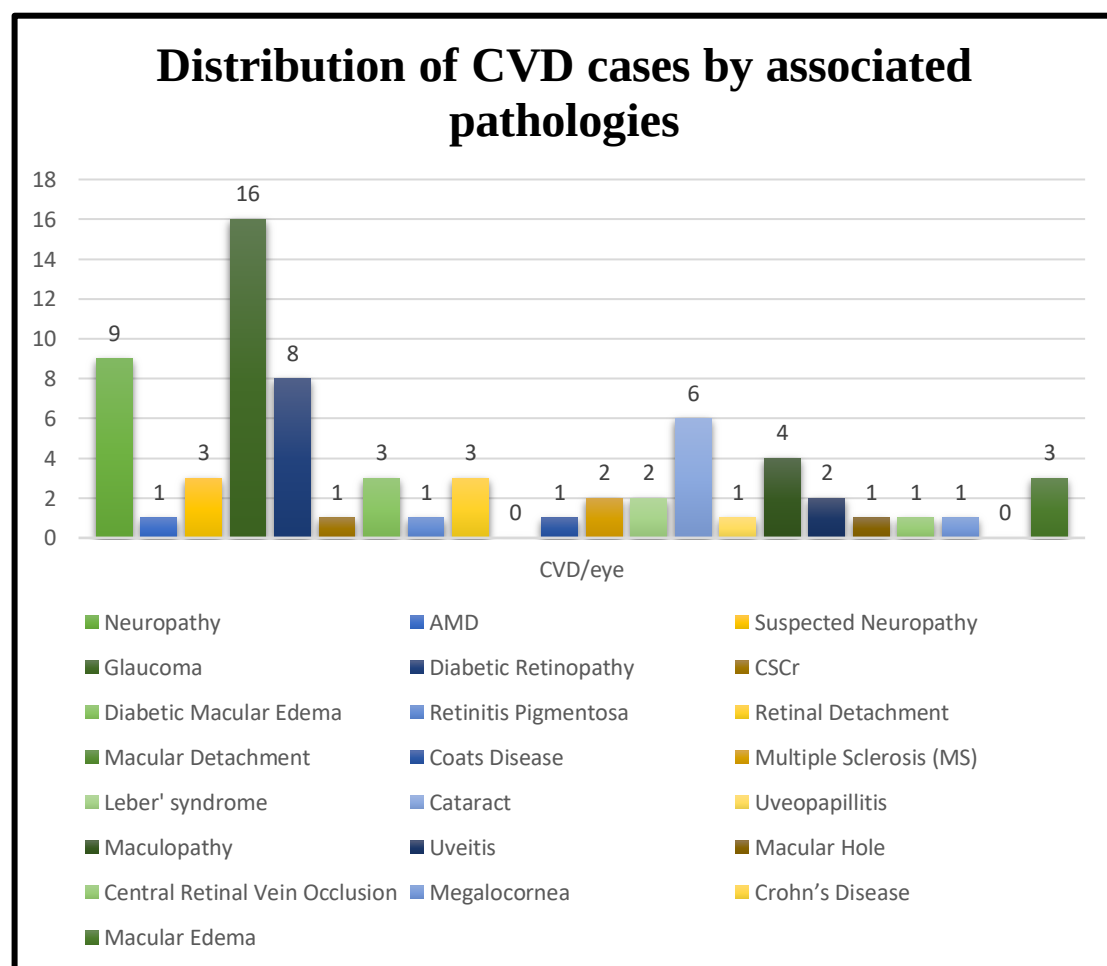


Chart 6: Distribution of CVD cases by associated pathologies

8.7. Comparative distribution of CVD by D15 test type

Using the two Lanthony D – 15 tests (Desaturated /Saturated) on 40 patients, we collected a total of 53 eyes with color vision deficiencies. Both tests were applied successively; however, seven cases (21%) showed color vision anomalies only with the

Desaturated D – 15 test, which is practically detect and identify the weak and mild color vision deficits, whilst, their answers on the Saturated test were correct and normal.

On the contrary, 27 (79%) cases showed color vision deficiencies in both tests. These results highlighted the use and importance of both Desaturated and Saturated in detection and diagnostic of the deficits in the individuals, and not relying on one protocol. The following pie chart shows the cases identified by Desaturated and Saturated D-15 tests.

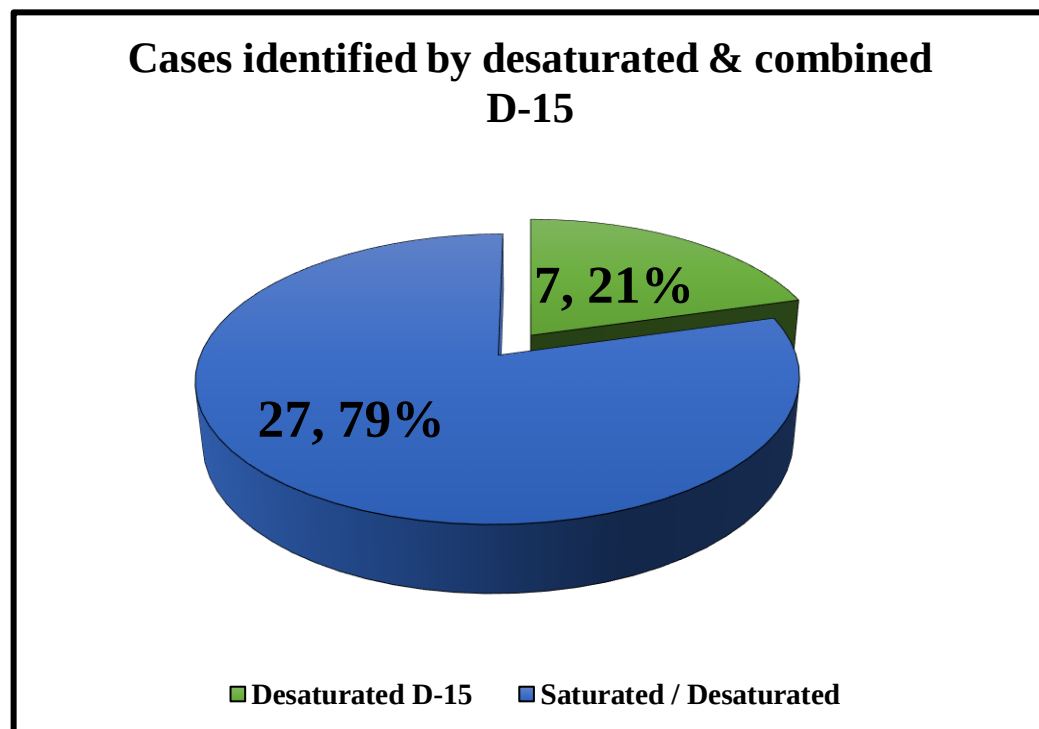


Chart 7: Cases identified by Desaturated & combined D-15

8.8. Classification of Color Vision Deficiency Cases by Type

After analyzing the results, we observed a clear predominance of deficits along the blue–yellow axis, with 36 cases (68%). In contrast, red–green deficiencies were relatively low, accounting for only two cases (4%). Meanwhile, 15 patients (28%) presented combined red–green and blue–yellow deficits.

These findings shows that acquired color vision deficiencies in our study are more frequently associated with the blue–yellow axis.

The pie chart below illustrate an analysis of color vision deficiencies types among patients, demonstrating the Red – Green and the Blue – Yellow axis.

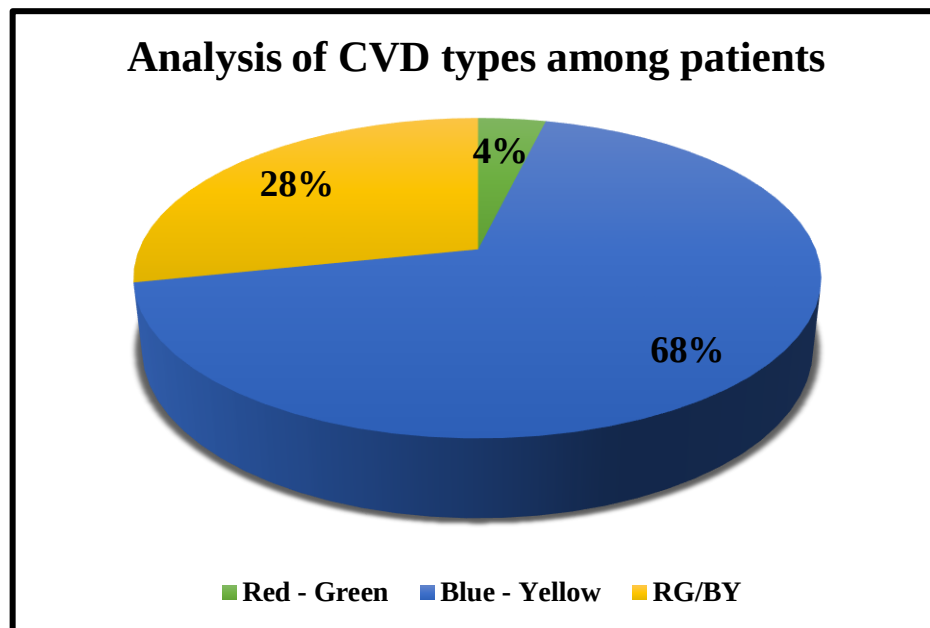


Chart 8: Analysis of CVD types among patients

9. Discussion

9.1. Interpretation of the results

- **General sample**

The study included 40 patients with an equal gender distribution (50 % males, 50 % females), aged 18 to 77 years. This balanced distribution reduces gender bias in the interpretation of acquired color vision deficiency (CVD), which is particularly relevant since congenital color vision deficiencies (CVD) predominantly affects males. In addition to, the wide age range, which reflects the diversity of ocular and systemic conditions, encountered in clinical practice.

These results revealed that acquired color vision deficiency has an equal gender distribution, in contrast to congenital color vision, which affects mainly males. This finding is coherent with the observation of Tait and Carroll. 2010, who acknowledge that acquired CVD have an equal incidence between males and females [17].

- **Predominance of ocular etiologies**

The results showed that the ocular diseases were the most widespread one causing acquired color vision deficiencies, with obtaining a 92 % of cases, in comparison to the 6 % of systemic diseases and 2 % of drug-related causes.

These findings were consistent with the records of Tait and Carroll, who admitted that the most common acquired CVD result from different diseases, which can influence the eye in a number of ways [17].

- **Pathologies' frequency**

Among the 40 patients, it was observed that some pathologies were frequent, such as glaucoma with 9 cases, followed by 6 cases of diabetic retinopathy, and 5 cases each with neuropathy and cataract, on the other hand there were less cases with macular pathologies such as AMD, macular hole .. Etc. with also less systemic diseases as an example multiple sclerosis disease. Which also shows the frequency of these pathologies.

Results were coherent with current studies of Quigley and Broman, (2006), reflecting glaucoma as one of the most leading causes of visual impairment worldwide with also the diabetic retinopathy according to Department of Ophthalmology (2023) showing a predominance in color clinical series [30],[31].

By analyzing this small sample, it permits us to estimate the frequency of certain pathologies in Algeria.

- **Etiological and Clinical Distribution of Color Vision Deficiency**

8 % of the sample were found blind and could not be tested. With 53 eyes (66%) were established cases with confirmed acquired color vision deficiencies, which confirm that ocular and systemic pathologies can cause a loss in the color vision of the individual.

Certain pathologies (15 %) showed no color vision deficiencies, which may suggest early stages of the diseases with no color vision is affected.

With most ocular cases turned to be diagnostic with CVD, it established the fact that most acquired CVD result mostly from ocular pathologies as it recognized with Tait and Carroll (2010) and with Hart (1987) [17], [32].

Systemically, 4 out of 6 cases presented with confirmed CVD, reflecting the well-documented capacity of systemic diseases to affect the visual pathway and produce secondary color impairment (Simunovic, 2016) [33].

The 2 out of 2 drug-related cases both presented with confirmed CVD, further supporting the role of toxic and pharmacological agents in producing acquired

chromatic dysfunction even in the absence of subjective complaint (Ageed et al., 2024) [34].

- **Importance of the testing protocol**

The results demonstrate the value and importance of combining the Desaturated D-15 with the Saturated D-15. The Desaturated D-15 identified only seven patients (21%) while their performance on the Saturated D-15 was correct and normal. Which indicate that the Desaturated D-15 is more sensitive and accurate to detect mild and weak color vision defects, which can be sometimes go undetected.

Moreover, this data has direct practical involvement: relying only on the standard Saturated D-15 risks missing early-stage deficiencies such as conditions like early glaucoma or mild diabetic retinopathy and pre-diagnostic of pathologies. The use of both tests in combination should therefore be considered a best practice standard in clinical color vision assessment.

Thus, Lanthony (1978) introduced the Desaturated D-15 precisely for this purpose, noting that its reduced chromatic saturation demands finer discrimination and therefore detects mild and subclinical deficits invisible to the standard version [35].

This dual-sensitivity design has direct clinical implications: relying solely on the Saturated D-15 risks systematically missing early-stage pathology. This study's results therefore reinforce the recommendation by Dain (2004) and Simunovic (2016) that combined saturated and desaturated D-15 testing should constitute the standard protocol in clinical colour vision assessment, particularly for conditions known to produce mild early-stage CVD such as glaucoma, diabetic retinopathy, and subclinical toxic exposure [33], [36].

- **Predominance of blue-yellow axis deficiencies**

After the analysis of the results, a clear predominance of the Blue-Yellow axis were found with 68 % of all color vision deficiencies. Unlike congenital CVD, which predominantly affects the red–green axis, acquired CVD most commonly manifests along the blue–yellow axis, due to damaging the short-wavelength-sensitive (S-cone) pathways in conditions such as glaucoma, diabetic retinopathy, and cataract.

The comparatively low proportion of red–green deficiencies (4%) further confirms the acquired nature of the deficits observed, as does the presence of combined axis deficiencies in 28% of cases, which likely reflects more advanced or multi-pathology disease states.

Thus, it was proven by Verriest (1963) that the predominance of Blue-Yellow deficits in acquired pathologies, in contrary with the Red-Green predominance of the congenital CVD. in addition, François and Verriest (1961) also stated that the most retinal and media conditions most likely affect short-wavelength-sensitive (S-cone) pathways [37], [38].

With The low rate of red–green deficiency (4%) confirming the predominantly of the acquired and ocular origin of the deficits, as red–green errors are more characteristic of optic nerve pathology (Hart, 1987) or specific genetic conditions such as LHON (Ventura et al., 2007).

The 28% combined-axis involvement is explained as advanced disease, multi-pathology condition, or combined retinal and optic nerve involvement, consistent with findings reported by Simunovic (2016) in patients with complex or advanced stages pathology [32], [33], [39].

- **Prevalence of pathologies affecting CVD**

- **Glaucoma**

Glaucoma was predominantly bilateral and the most frequent pathology in this study, with nine patients (18 eyes), and resulting to 16 out of 18 eyes with color vision deficiencies, and one was a blind eye with another with no CVD.

Thus, glaucoma corresponds to a number of handicaps within the optic nerve presented by a loss of retinal ganglion cells with a loss of some or most of the fibers of the optic nerve, leading to visual field loss. The disease preferentially damages the short-wavelength sensitive pathways, which explains the characteristic Blue-Yellow axis deficiency observed across all glaucomatous patients in this study [17].

Patient 08, a stable glaucoma patient of 12 years, presented simple errors within the Blue-Yellow axis in the Desaturated D-15, and tolerable errors in the Saturated D-15, proposing a mild but confirmed CVD detectable across both tests despite clinical stability of glaucoma. **Patient 31** a glaucomatous with high myopia and an onset of

cataract, presented multiple B-Y axis errors bilaterally in the Desaturated D-15 with fewer but still B-Y mistakes in the Saturated D-15. Confirming Blue-Yellow defects in both eyes.

Patient 07 reported that the Saturated D-15 was easier than the Desaturated D-15, while is clinically coherent, as the Desaturated test is more precise and reveals mild deficits that the Saturated test may partially mask. For patient 17, the glaucoma was associated with megalocornea a condition that may lead to glaucoma on later stages. The results showed only one eye with B-Y CVD in the Desaturated test, while the Saturated test were nearly correct bilaterally, displaying that an asymmetric mild deficit was detected in one eye by the Desaturated test.

Patient 18, presented with both Red – Green axis and Blue – Yellow axis deficits, in the Desaturated test and Saturated one bilaterally, shows a severe color vision deficiencies on both eyes with combined axis involvement.

The finding of blue–yellow axis CVD across all glaucomatous patients (16 out of 18 eyes) is strongly consistent with the literature. Sample, Weinreb & Boynton (1986) and Adams et al. (1987) established that glaucoma selectively damages the short-wavelength-sensitive (S-cone) pathways [40], [41], [32].

The asymmetric CVD in patient 17 (detected only in the Desaturated test) further illustrates the value of the more sensitive protocol for detecting subclinical unilateral deficits, as described by Vingrys and King-Smith (1988) [42].

The combined R-G and B-Y involvement in patient 18 reflects the progression of chromatic loss to a multi-axis pattern in advanced disease, consistent with Hart et al. (1987) [32].

Diabetic retinopathy

Diabetic retinopathy was identified in six patients (12 eyes), 8 out of 12 eyes were found with color vision deficiencies, and it was bilateral in the majority of cases.

Diabetic retinopathy results from a chronic systemic disease, which is diabetes. It results to a severe changes in the retinal blood vessels affecting patients' vision and leading to photoreceptor dysfunction, producing acquired CVD predominantly along the Blue – Yellow axis [17].

Patient 03 exhibited a clear asymmetry between the two eyes: one eye was affected, showing multiple errors on both the Desaturated and Saturated D-15 tests, which confirmed a blue–yellow color vision deficiency, while the other eye showed no color vision impairment. This finding highlights the asymmetric manifestation of diabetic retinopathy and its associated CVD.

Patient 11 presented bilateral B-Y deficit errors in both the Desaturated and the Saturated D-15, with equal difficulty in both tests and severe photophobia, indicating a moderate to severe confirmed CVD.

Patient 32 presented with tolerable mistakes of the Blue – Yellow axis in the Desaturated D-15 and near normal results in the Saturated test, proposing an early or subclinical stage where retinal damage has not yet produced a confirmed color vision deficit under standard testing conditions.

Patient 34, diabetic retinopathy and macular edema bilaterally manifested with combined Red – Green and Blue – Yellow axis errors in both eyes across both tests. This demonstrate the progression phase of color vision loss in relation to the severity of diabetic retinal damage. Which, also support the use of combined D-15 protocol for monitoring disease progression in diabetic patients.

It was consistent with studies proven by Cho et al. (2000), that the S-cones are damaged by diabetic retinopathy resulting to Blue – Yellow color blindness [43].

It was also admitted that color vision deficits was observed in early diabetic retinopathy, including patients clinically normal, which confirm the fact that the dual of D-15 can pre-diagnostic the disease [30], [43].

According to Wisam Chasib Alhamdani et al. (2023), for acquired color vision defects, it is not easy to classify type or color vision defects which explains the combined Red – Green and Blue – Yellow involvement in patient 34 (bilateral diabetic retinopathy with macular edema) [30].

Optic neuropathy

Neuropathy was found in five patients with a total of 9 affected eyes, with representing the pathology with the highest CVD conversion rate in the study i.e. all confirmed neuropathy eyes manifested color vision deficiency.

Patient 22, presented with congenital optic atrophy, visual acuity of 1/10, and the errors was with both axis Red – Green and Blue – Yellow in the Desaturated and Saturated tests, which reflect a severe and complex bilateral acquired color vision deficiency consistent with long-standing nature of the condition.

Patient 33, diagnosed with optic neuropathy since 2013, presented with severe results, absolute absence of color perception in the Desaturated D-15 test bilaterally which appeared completely white to the patient, on the other hand, the Saturated test was presented with errors and mistakes with the Red – Green and Blue – Yellow axis. Thus, it shows a total color disruption in the Desaturated and a severe confirmed acquired CVD in the Saturated range.

Patient 39, exhibited with retrobulbar neuropathy in the right eye only, showed errors with both axis involved R-G and B-Y in the Desaturated and saturated tests, with the left eye remaining entirely normal.

These findings established that color vision testing could detect underlying neuropathy at a pre-diagnostic stage, and that the dual D-15 protocol is essential for fully characterizing the color deficits in optic nerve pathologies.

The 100% CVD rate in confirmed neuropathy cases (the highest of any pathological group) is consistent with the well-documented role of color vision as a sensitive early marker of optic nerve dysfunction. Thus, optic nerve diseases can influence color vision earlier and more profoundly than other ocular conditions with the same degree of visual acuity loss, as established by Grover et al. (2010), who showed that patients with optic neuropathy had significantly worse color vision than those with macular or media pathology at equivalent acuity levels [44].

The bilateral total chromatic imperceptions in patient 33's Desaturated test (bar appearing entirely white) represents the most severe CVD profile in the neuropathy group and is consistent with reports of complete achromatic responses in longstanding or advanced compressive optic neuropathies (Hart, 1987) [32].

The multi-axis R-G and B-Y involvement in patient 39 (retrobulbar neuropathy) is consistent with the finding that acute retrobulbar optic neuritis, may lead to combined Red – Green and Blue – Yellow deficits although is rare but it may occur, hart et al. (1987) [32].

 Suspected neuropathy

Suspected optic neuropathy was identified in four (six eyes) patients presented with variable CVD outcomes that reflect the subclinical and early nature of the condition at the time of testing.

Suspected optic neuropathy refers to cases where clinical signs suggest optic nerve involvement such as visual field complaints or altered visual acuity, without meeting the full diagnostic criteria for confirmed neuropathy. In such cases, color vision testing is particularly valuable, as color discrimination loss may represent the earliest detectable functional sign of optic nerve damage, preceding structural changes visible on fundus examination.

Patient 30 presented the clearest CVD finding among suspected cases: the right eye was normal, while the left eye (the clinically suspicious eye) showed multiple Blue - Yellow errors in both the Desaturated and the saturated, confirming a unilateral blue–yellow CVD consistent with early unilateral optic nerve involvement.

Patient 35 similarly showed a normal right eye throughout, while the left eye presented multi-axis errors (R-G and B-Y) in the Desaturated and saturated tests, reflecting a both axis CVD in the suspected eye.

On the contrary, **patients 28 and 29** presented milder findings. Patient 28 showed B-Y errors in the Desaturated of both eyes, but only one error in the saturated of the right eye and a correct result in the left eye, leading to a CVD classification in the right eye only and a non-CVD in the left.

Patient 29 presented only tolerable errors in the Desaturated bilaterally, with correct and near-correct results in the saturated, resulting in a non-CVD classification, yet the Desaturated still flagged subclinical color anomalies that the saturated test alone would have completely missed.

These findings demonstrate that in suspected neuropathy cases, the Desaturated is indispensable for detecting early and mild color changes, and support the role of color vision testing as a pre-diagnostic tool capable of identifying optic nerve dysfunction before it becomes clinically confirmed.

The variable CVD outcomes among suspected neuropathy cases are consistent with the subclinical and early nature of the condition at the time of testing. The role of color

vision testing as a pre-diagnostic indicator of optic nerve involvement is well-established: dyschromatopsia is frequently the first measurable functional sign of optic nerve damage, preceding detectable visual field changes (Hart, 1987) [32].

The finding that the Desaturated D-15 detected subclinical chromatic anomalies in patients 28 and 29 that the Saturated D-15 would have completely missed is consistent with the established sensitivity advantage of the Desaturated version for early-stage deficits (Lanthony, 1978; Vingrys & King-Smith, 1988) [35], [42].

These findings reinforce the value of systematic dual D-15 color vision screening in patients presenting with unexplained visual complaints without fully confirmed neuropathy.

Cataract

Cataract was observed in five patients presenting unilaterally in most cases, with CVD confirmed in the majority of affected eyes.

Cataract causes progressive yellowing and opacification of the crystalline lens, selectively absorbing short-wavelength (blue) light and producing a characteristic blue–yellow deficiency that is a media-related, rather than neural, deficit [17].

Patient 19 illustrates this most clearly: the non-operated right eye showed complete color imperceptions in the Desaturated (the bar appeared entirely white), and B-Y parallel errors persisted in the saturated, with severe photophobia. The recently operated left eye, on the contrary, showed fewer errors in the Desaturated and reduced B-Y errors in the saturated, demonstrating that surgical lens extraction partially restores color discrimination even in the early post-operative period. This directly demonstrates the progression phases of color vision recovery in relation to surgical treatment of cataract.

Patient 06, who recently underwent cataract surgery in the right eye, showed only a single tolerated error in the Desaturated and totally correct results in the saturated, confirming near-complete color vision recovery post-operatively.

Thus, these findings prove that cataract-related CVD is largely reversible, and using both tests for color vision detecting provides an intent measure of functional color recovery following surgical intervention.

In addition, the blue–yellow CVD errors observed in cataract cases, and its partial reversibility following lens extraction, is entirely compatible with the literature. Thus, the progressive yellowing and opacification of the crystalline lens absorbs short-wavelength (blue) light, producing a special Blue - Yellow acquired dyschromatopsia that represents a media-based, rather than neural, deficit (Ao et al. 2019) [45].

Phacoemulsification showed favourable results to effectively restore color perception in the short-wavelength bands (470–580 nm), with statistically significant improvements found within the early post-operative period (Ao et al. 2019) [45].

Diabetic macular edema

Diabetic macular edema was diagnosed in two patients, with CVD present in all affected eyes.

Diabetic macular edema is the cause of visual loss in diabetic patients, described by color vision deficits predominantly along the blue–yellow axis, as B-Y color vision has been well known to be impaired in diabetic patients, with color vision error significantly related with foveal thickness and significantly greater in patients with confirmed macular edema [46].

Patient 01 presented with diabetic macular edema alongside edematous optic neuropathy showed very disturbed color perception in the Desaturated with B-Y axis parallel errors, while the saturated was comparatively more correct, suggesting residual color function still detectable at the saturated threshold.

Patient 24 showed clear blue-yellow parallel errors of the left eye, the affected one across both tests, while the right eye presented only a single tolerable error in the Desaturated and a normal result in the saturated, confirming the asymmetric and localized nature of diabetic macular edema-related CVD.

Diabetic macular edema involves visual loss in diabetic patients with a central color vision deficit predominantly along the blue–yellow axis, consistent with the established pattern of acquired color vision deficiencies in macular conditions (Verriest, 1963) [37].

 Macular pathologies (AMD, Macular hole, Macular detachment, Myopic maculopathy, Macular edema)

Several macular pathologies were encountered in this study, AMD (patient 05), macular hole (patient 13), macular detachment (patient 16), and myopic maculopathy (patient 02), macular edema (patient 20) with each producing distinct CVD profiles.

Patient 05, (AMD and blind right eye), showed Red – Green and Blue – Yellow errors in both the Desaturated and saturated of the left eye, with nearly identical results across both tests. The presence of both axis, in acquired CVD that is more commonly involves the Blue – Yellow axis, it may reflect the specific pattern of cone' damage in advanced AMD.

Patient 02, with bilateral myopic maculopathy, revealed multiple Blue – Yellow errors in the Desaturated test with clearer and still B – Y mistakes in the saturated one too. With the left eye being more pronounce, representing the asymmetric severity of bilateral macular involvement.

Patient 13 (recent macular hole) presented tolerable B-Y errors in the Desaturated of the right eye after three attempts, with nearly identical errors in the saturated, and a normal left eye throughout. The need for three attempts reflects the visual disturbance and instability associated with an active macular hole, rather than a severe color deficit.

Patient 16 (macular detachment with macular malformation) is the most clinically instructive case in this group: at the first examination, B-Y errors were present in the Desaturated of the right eye, while the saturated was entirely correct and the left eye was normal throughout. At the follow-up control one month later, errors in the Desaturated were reduced, confirming a progressive improvement of color vision following partial resolution of the macular detachment. This demonstrates the role of color vision testing in monitoring the recovery of macular conditions over time.

Patient 20 (glaucoma with a macular edema in the right eye) presented Blue - Yellow errors in both tests for the right eye, while the left eye showed complete color imperceptions in the Desaturated but B-Y and R-G errors in the saturated B.F, reflecting the compounding effect of glaucoma and macular edema on color vision.

The range of macular CVD patterns observed across AMD, macular hole, macular detachment, myopic maculopathy, and macular edema is consistent with the

predominantly blue – yellow of macular acquired CVD documented in the literature. Hart (1987) admitted that macular disorders typically produce blue–yellow axis deficiency due to the central concentration of S-cones [32].

The combined R-G and B-Y involvement in patient 05 (advanced AMD with a blind fellow eye) reflects the pattern reported by Simunovic (2016), in which advanced macular disease can extend color loss beyond the blue - yellow axis as broader cone populations are affected [33].

The progressive improvement in patient 16 (macular detachment) with errors reducing in the Desaturated at one-month follow-up demonstrates the role of color vision testing as a monitoring tool for macular recovery, consistent with the reversibility of color vision if the main cause was treated as it provided by Department of Ophthalmology, Emergency County Hospital, Cluj-Napoca, Romania, (2020). [16].

Central serous chorioretinopathy (CSC)

Central serous chorioretinopathy was found in 01 patient, a 35-year-old presented with a normal eye in addition to a CVD in the left eye.

CSC is a result of a serous detachment of the neurosensory retina from the retinal pigment epithelium (RPE) at the level of the macular, disturbing photoreceptor function and producing a central color vision deficit [47].

Patient 04 displayed a clear Blue – Yellow errors in the Desaturated, with the same error pattern reproduced in the saturated, indicating a consistent and confirmed blue–yellow deficit detected equally by both tests. Clinically, the patient reported severely disturbed vision, difficult to distinguish colors in monocular conditions, black-and-white vision during daytime and severe photophobia, which reflect the profound impact of foveal serous detachment on color processing. The right eye remained entirely normal, confirming the strictly unilateral and macular origin of the CVD.

The results of the patient are coherent with the findings of Simunovic (2016), confirming that central serous chorioretinopathy may lead to a combined Red – Green and Blue – Yellow deficits [33].

 Retinal detachment

Retinal detachment was identified in three patients, with variable CVD outcomes depending on the extent and location of the detachment.

Patient 15 (operated bilaterally for retinal detachment), presented B-Y errors in the Desaturated of the right eye, with fewer but still B-Y errors in the saturated. The left eye showed combined R-G and B-Y errors in the Desaturated, with the same combined pattern persisting in the saturated, reflecting a more severe and multi-axis CVD in the left eye consistent with greater retinal involvement on that side.

Patient 37 (glaucoma with retinal detachment in the right eye and a blind left eye), showed mild B-Y CVD in the right eye across both tests, illustrating the compounding effect of two concurrent pathologies on color vision.

Patient 06 (post-operative retinal detachment surgery) presented near-normal color vision with only one tolerated error in the Desaturated and entirely correct results in the saturated, confirming good functional color recovery following successful surgical reattachment.

Simunovic (2016) demonstrated that macular involvement in retinal detachment produces significant Blue – Yellow color vision deficits. With, the near-normal CVD in patient 06 post-operatively confirms that successful surgical reattachment of the retina can restore color discrimination [33].

On the other hand, studies of costa et al. (2024) proved that retinal detachment might not only cause Blue – Yellow CVD, it can also include the Red – Green deficiencies which is what is present in patient 15 [48].

 Uveopapillitis and uveitis

Uveopapillitis was identified in 01 patient, and uveitis as part of a multi-pathology presentation in 2 patients, with variable CVD outcomes.

Patient 09 (blind right eye, uveopapillitis in the left eye) presented R-G and B-Y errors in the Desaturated, while the saturated, though easier overall, still showed errors parallel to both axis, reflecting a severe and complex multi-axis CVD driven by combined retinal and optic nerve inflammatory involvement. The severe photophobia and the urge to close the eyes further illustrate the significant functional burden of this condition.

Patient 38 (presenting with diabetic retinopathy, maculopathy, and uveitis simultaneously) showed complete color imperceptions bilaterally in the Desaturated, while the saturated revealed R-G and B-Y errors in the right eye and B-Y errors in the left eye, confirming a severe blue–yellow CVD with a mild red–green component. This case illustrates how the accumulation of multiple concurrent pathologies produces a particularly severe and complex color deficit.

On the contrary, **patient 21** (blind right eye with uveitis and post-cataract surgery in the left eye) presented only a single tolerable error in the Desaturated and entirely correct results in the saturated, resulting in a non-CVD classification. This improvement reflects good functional recovery following treatment, and demonstrates that uveitis-related CVD can be reversible when the inflammatory process is controlled.

The involvement of the retina and optic nerve caused by the Uveopapillitis and uveitis produce blue–yellow axis CVD and extending to the red–green axis when the optic nerve is affected which is consistent with the studies of Simunovic, (2016) [33].

Central retinal occlusion

Central retinal occlusion was identified in one patient, a 64-year-old with unilateral CVD in the left eye.

Central retinal occlusion causes acute ischemic (reduction of blood flow to a part of the body) damage to the retinal layers, disrupting photoreceptor and inner retinal function in the affected eye.

Patient 14 presented a moderately difficult arrangement in the Desaturated with blue–yellow errors, while the saturated was entirely correct and easy, with no errors representing one of the cases where CVD was detected by the Desaturated. The right eye remained normal throughout. This finding confirms that retinal occlusion can produce a mild blue–yellow deficit detectable only with the more sensitive Desaturated test, and that the saturated alone would have classified this patient as normal, underlining the clinical value of the dual-test protocol.

These findings are consistent with Hart 1987, that any disruption to the retina either degeneration or inflammatory, or even damage may cause abnormalities with color vision [32].

In addition, Lanthony 1978 established that the Desaturated version detects mild deficits missed by the standard test [35].

Retinitis pigmentosa and coats disease

Retinitis pigmentosa was identified in patient 26, presenting alongside Coats disease, with a blind right eye and confirmed CVD in the left eye.

Retinitis pigmentosa is a hereditary photoreceptor dystrophy characterized by progressive rod then cone degeneration [17].

In **patient 26** (post-surgical with partial improvement) the left eye showed Red-Green and Blue-Yellow errors in the Desaturated, with clearly Blue-Yellow errors in the saturated, confirming a blue–yellow dominant CVD with a residual red–green component in the Desaturated range, simplifying to a predominant tritan deficit in the saturated range. The coexistence of Coats disease (a retinal vascular exudative condition) further compounds the retinal damage and may contribute to the multi-axis nature of the color deficit observed in the Desaturated test.

These results is coherent with the findings of Simunovic (2016), who demonstrated that retinitis pigmentosa lead to Blue – Yellow deficits but it may also combine the Blue – Yellow with the Red – Green deficiencies, with also the presence of the retinal damage caused by Coats disease, which results to color vision deficits [33].

Leber hereditary optic neuropathy (LHON)

Leber hereditary optic neuropathy was identified in patient 23, a 32-year-old presenting with bilateral CVD and severely reduced visual acuity of 1/10.

It is originally a genetic monocular condition, which rapidly becomes bilateral. Typically occurs between 15 and 35 years of age, beginning with a sudden decrease in visual acuity in one eye, followed by the other. Color vision is disturbed. This pathology is, in fact, a hereditary atrophy of the optic fibers [49].

Patient 23 presented predominantly red–green axis errors in both eyes across both tests, Desaturated and saturated. The presence of red–green CVD is particularly significant in this case, as acquired deficiencies more commonly involve the blue–yellow axis; the R-G dominant pattern in LHON optic nerve dysfunction. The patient reported

worsening visual acuity over the preceding two years, demonstrating the progressive nature of the color deficit in parallel with disease evolution.

Hart et al. (1987) proved that Leber' syndrome in its early stage cause Red – Green color vision deficiencies, which align with the finding in the Leber patient above, and that it may rarely lead to the Blue – Yellow deficits [32].

Multiple sclerosis

Multiple sclerosis was identified as a systemic condition in **patient 12**, associated with congenital optic neuropathy. The combined effect of demyelination and congenital nerve damage produced complete achromatopsia in the Desaturated (total absence of color perception, with the bar appearing entirely white) and multi-axis errors in the saturated, in addition to nyctalopia (night blindness). This illustrates the devastating impact of dual neurological insults on color vision, and represents the most severe CVD profile observed in this study.

Moreover, Multiple sclerosis is well-documented as a cause of acquired dyschromatopsia through demyelination of the optic nerve, with colour vision loss documented as both an early sign and a persistent deficit following optic neuritis episodes (Hart, 1987; Martínez-Lapiscina et al, 2014) [32], [50].

The combination with congenital optic neuropathy would be expected to compound the baseline colour discrimination capacity and produce a more severe total chromatic impairment than either condition alone. The nyctalopia reported by this patient is consistent with the involvement of rod-mediated pathways secondary to the underlying optic nerve damage, as documented by Simunovic (2016) in cases of severe combined optic nerve dysfunction [33].

Crohn' disease

Crohn's disease was identified in **patient 25**, a 24-year-old with concurrent high myopia, presenting no confirmed CVD in either eye. The desaturated showed only a few tolerable B-Y errors in the right eye and a correct result in the left eye, while the saturated showed only tolerable errors in the right eye and was entirely correct in the left eye. The patient reported a central dark spot in the right eye at the time of examination, and a follow-up test was planned after 15 days. The absence of confirmed

CVD may reflect the indirect and limited ocular involvement of Crohn's disease at this stage, as its ocular complications (when present).

This confirm that the reason for acquired CVD are the pathologies that alter and touch the eye which may lead to a dysfunction [32].

Drug related causes

A drug-related cause was identified in **patient 40**, a 28-year-old with a 7-year history of drug consumption, presenting bilateral CVD.

Drugs can cause alterations in visual processing including color discrimination. Patient 40 presented bilateral Blue-Yellow errors in both the Desaturated and the saturated tests, confirming a moderate and consistent blue–yellow CVD, more pronounced in the right eye, across both levels of color saturation. Importantly, the patient reported no subjective discomfort and no awareness of any color vision problem, demonstrating that drug-related CVD can be entirely subclinical and would go completely undetected without systematic objective testing. This finding supports the use of the dual D-15 protocol as a screening tool in patients with toxic or pharmacological risk factors, particularly in cases where the patient does not spontaneously report visual complaints.

This finding is consistent with Ageed et al.'s (2024) observation that regular drug users do not normally report visual dysfunction despite objectively measurable retinal impairment with confirming the influence of drugs on the color visual system of the individual [34].

9.2. Limitations

- The non-sufficient sample size (40 patients) limits the statistical power and generalize of findings.
- The cross-sectional design prevents conclusions about disease progression or recovery of color vision over time.
- The exclusion of blind eyes (6 eyes) may have introduced a selection bias toward less severe cases.
- The study was performed in one establishment, which may not be representative of the vast population.
- Unequal distribution of pathological categories, with some conditions represented by only one or two patients.

9.3. Study' perspectives

- Larger studies to confirm and generalize the findings of this study.
- Follows up studies to track CVD progression and regression in relation to disease evolution and treatment response.
- Investigation of the pre-diagnostic role of color vision testing in suspected optic neuropathy cases.
- Integration of more sensitive color vision tests alongside the dual D-15 protocol for finer diagnostic of color deficits.

V. Conclusion

The findings of this research confirm that acquired color vision deficiency is a frequent and clinically significant manifestation of ocular, systemic, and drug-related pathologies, with a predominance of blue–yellow axis deficiency coherent with the established literature on acquired CVD. The protocol of using both the Desaturated and saturated D-15 showed a clinical value by detecting 21% of CVD cases only by the Desaturated test, which confirm the standard saturated D-15, is insufficient for detecting and the diagnostic of mild and weak color impairment. Moreover, Across all pathological categories, the nature, axis, and severity of color vision deficiency were found to consider the underlying mechanism and stage of the disease, providing diagnostically and prognostically relevant information beyond what standard visual acuity measurement alone can offer.

In addition, the reversibility of CVD observed in cataract and inflammatory cases further highlights the role of color vision testing as a useful outcome measure in the monitoring of treatment response.

Furthermore, these findings increased the values of color vision assessment as a sensitive, non-invasive, and clinically informative tool in the comprehensive evaluation of visual function.

Conclusion

This dissertation was concluded with success, starting from the theoretical section, which included an overview of every corner of color blindness from the anatomy and mechanism of color vision with its classification reaching the diagnostic and therapeutic approaches currently available. On the other hand, the practical section proved and confirmed that acquired color vision deficiency is a common and frequently clinical manifestation of ocular, systemic, and drug-related pathologies, with an obvious dominance of Blue – Yellow axis deficits and consistent with the established literature.

In addition to the protocol of the dual D-15 test (Desaturated and Saturated D-15), that showed a clinical value confirming the standard Saturated D-15 alone is insufficient for the detection of weak and mild color impairment. Thus, the reversibility of CVD observed in some cases highlights the role of color vision testing as an essential measuring tool of functional recovery following treatment.

In the end, these findings support the important role and value of color vision assessment as a non-invasive and clinically informative tool to assist the health professional to undergo patients' ocular health.

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Annex

Annex

Clinical sheet

1. Patient Information

- ID :
- Age :
- Sex : ☐ Female ☐ Male
- Date of consultation : ... / ... / ...

2. Medical History

- Médicaux :
- Oculaires :
- Médicaments :

3. Farnsworth D-15 Test

- **Bright bar (desaturated):**
 - Result:
.....
.....
 - Interpretation:
.....
.....
- **Dark bar (saturated):**
 - Result:
.....
.....
 - Interpretation:
.....
.....

4. Other Complementary tests

Ishihara's test.....
.....

5. Diagnostic

- ☐ Normal color vision
- ☐ Congenital CVD
- ☐ Acquired CVD

6. Conclusion

.....

Lanthony Desaturated D-15 Evaluation Form

Name _____ Age _____ Date _____ File No _____

Department _____ Tester _____

