

The Retina

The vertebrate retina (from Latin *rēte*, meaning "net") is a light-sensitive layer of tissue, lining the inner surface of the eye. The optics of the eye create an image of the visual world on the retina (through the cornea and lens), which serves much the same function as the film in a camera. Light striking the retina initiates a cascade of chemical and electrical events that ultimately trigger nerve impulses. These are sent to various visual centers of the brain through the fibers of the optic nerve.

The retina is a complex, layered structure with several layers of neurons interconnected by synapses. The only neurons that are directly sensitive to light are the photoreceptor cells. These are mainly of two types:
the rods and cones.

Rods function mainly in **dim light** and provide **black-and-white** vision, while cones support daytime vision and the perception of color. A third, much rarer type of photoreceptor, the photosensitive ganglion cell, is important for reflexive responses to bright daylight. Neural signals from the rods and cones undergo processing by other neurons of the retina. The output takes the form of action potentials in retinal ganglion cells whose axons form the optic nerve.

Structure

The vertebrate retina has ten distinct layers from innermost to outermost, they include:

- 1- Inner limiting membrane
- 2- Nerve fiber layer
- 3- Ganglion cell layer – contains nuclei of ganglion cells, the axons of which become the optic nerve fibers.
- 4- Inner plexiform layer.
- 5- Inner nuclear layer – contains bipolar cells.
- 6- Outer plexiform layer.
- 7- Outer nuclear layer.
- 8- External limiting membrane – layer that separates the inner segment portions of the photoreceptors from their cell nucleus.
- 9- Photoreceptor layer – Rods & Cones.
- 10- Retinal pigment epithelium.

Of these the four main layers, from outside are: Retinal pigment epithelium, Photoreceptor layer, bipolar cells, and finally, the Ganglion cell layer.

The retina consists of a **pigmented retina** and a **sensory retina**. The

sensory retina contains three layers of neurons: photoreceptor, bipolar, and ganglionic. The cell bodies of these neurons form nuclear layers separated by plexiform layers, where the neurons of adjacent layers synapse with each other.

The outer plexiform (plexus like) layer is between the photoreceptor and bipolar cell layers. The inner plexiform layer is between the bipolar and ganglionic cell layers.

The pigmented retina, or pigmented epithelium, consists of a single layer of cells. This layer of cells is filled with melanin pigment and, together with the pigment in the choroid, provides **a black matrix**, which enhances visual acuity by isolating individual photoreceptors and reducing light scattering. Pigmentation is not strictly necessary for vision, however. People with albinism (lack of pigment) can see, although their visual acuity is reduced because of some light scattering.

The layer of the sensory retina nearest the pigmented retina is the layer of rods and cones. The rods and cones are photoreceptor cells, which are sensitive to stimulation from “visible” light. The light-sensitive portion of each photoreceptor cell is adjacent to the pigmented layer.

In adult humans, the entire retina is approximately **72%** of a sphere about **22 mm** in diameter. The sensory retina contains about **7 million cones** and **75 to 150 million rods**.

When the **posterior region** of the retina is examined with an **ophthalmoscope**, several important features can be observed. Near the center of the posterior retina is a small yellow spot approximately **4 mm** in diameter, the **macula lutea**. In the center of the macula lutea is a small pit, the **fovea centralis**.

The fovea and macula make up the region of the retina where light is focused. The fovea is the portion of the retina with the greatest visual acuity, the ability to see fine image **because** the photoreceptor cells are **more tightly packed** in that portion of the retina than anywhere else. Just medial to the macula lutea is a white spot, the **optic disc**, through which blood vessels enter the eye and spread over the surface of the retina. This is also the spot where nerve processes from the sensory retina meet, pass through, and exit the eye as the optic nerve.

The optic disc contains no photoreceptor cells and does not respond to light; therefore it's called the blind spot of the eye .

Rods

Rods are bipolar photoreceptor cells involved in non-color vision and are responsible for vision under conditions of reduced light. The modified, dendritic, light-sensitive part of rod cells is cylindrical, with no taper from base to apex. This rod-shaped photoreceptive part of the rod cell contains about 700 double-layered membranous discs. The discs contain rhodopsin, which consists of the protein opsin covalently bound to a pigment called retinal (derived from vitamin A).

Function of Rhodopsin

The changes that rhodopsin undergoes in response to light.

In the resting (dark) state, the shape of opsin keeps 11-cis-retinal tightly bound to the internal surface of opsin. As light is absorbed by rod cells, opsin changes shape from 11-cis-retinal to all-trans-retinal. These changes activate the attached G protein, called transducin, which closes Na^+ channels, resulting in hyperpolarization of the cell.

This hyperpolarization in the photoreceptor cells is somewhat remarkable, because most neurons respond to stimuli by depolarizing. When photoreceptor cells are not exposed to light and are in a resting, non-activated state, some of the Na^+ channels in their membranes are open, and Na^+ flow into the cell. This influx of Na^+ causes the photoreceptor cells to release the neurotransmitter **glutamate** from their presynaptic terminals. Glutamate binds to receptors on the postsynaptic membranes of the bipolar cells of the retina, causing them to hyperpolarize. Thus, glutamate causes an inhibitory postsynaptic potential (IPSP) in the bipolar cells.

When photoreceptor cells are exposed to light, the Na^+ channels close, fewer Na^+ enter the cell, and the amount of glutamate released from the presynaptic terminals decreases. As a result, the hyperpolarization in the bipolar cells decreases and the cells depolarize sufficiently to release neurotransmitters, which stimulate ganglionic cells to generate action potentials. The number of Na^+ channels that close and the degree to which they close is proportional to the amount of light exposure.

At the final stage of this light-initiated reaction, retinal is completely released from the opsin. This free retinal may then be converted back to vitamin A, from which it was originally derived. The total vitamin A/retinal pool is in equilibrium so that under normal conditions the amount of free retinal is relatively constant. To create more rhodopsin, the altered retinal must be converted back to its original shape, a reaction that requires energy. Once the retinal resumes its original shape, its recombination with opsin is spontaneous, and the newly formed rhodopsin can again respond to light.

Light and dark adaptation

Light and dark adaptation is the adjustment of the eyes to changes in light. Adaptation to light or dark conditions, which occurs when a person comes out of a darkened building into the sunlight or vice versa, is accomplished by changes in the amount of available rhodopsin. **In bright light excess rhodopsin** is broken down so that not as much is available to initiate action potentials, and the eyes become “adapted” to bright light. Conversely, **in a dark room** more rhodopsin is produced, making the retina more light-sensitive.

Light and dark adaptation also involves **pupil reflexes**. The pupil enlarges in dim light to allow more light into the eye and contracts in bright light to allow less light into the eye. In addition, **Rod function** decreases and cone function increases in light conditions, and vice versa during dark conditions. This occurs because rod cells are more sensitive to light than cone cells and because rhodopsin is depleted more rapidly in rods than in cones.

Rhodopsin Cycle

1. Retinal (in an inactive configuration called II-cis-) is attached inside opsin to make rhodopsin.
2. Light causes opsin to change shape, and retinal changes shape from II-cis retinal to all-trans-retinal. This activated rhodopsin also activates the attached G protein (called transducin), which closes Na⁺ channels, resulting in hyperpolarization of the cell.
3. All-trans-retinal detaches from opsin.
- 4- All-trans-retinal is converted to II-cis-retinal, a process that requires energy.
5. II-cis-retinal attaches to opsin, which returns to its original (dark) configuration.

Cons

Color vision and visual acuity are functions of cone cells. Color is a function of the wavelength of light, and each color results from a certain wavelength within the visible spectrum. Even though rods are very sensitive to light, they cannot detect color, and sensory input that ultimately reaches the brain from these cells is interpreted by the brain as shades of gray. Cones require relatively bright light to function. As a result, as the light decreases, so does the color of objects that can be seen until, under conditions of very low

illumination, the objects appear gray. This occurs because as the light decreases, fewer cone cells respond to the dim light.

Cones are bipolar photoreceptor cells with a **conical** light sensitive part that tapers slightly from base to apex. The outer segments of the cone cells, like those of the rods, consist of double-layered discs. The discs are slightly more numerous and more closely stacked in the cones than in the rods. Cone cells contain a **visual pigment, iodopsin**, which consists of retinal combined with a photopigment opsin protein. **Three** major types of color-sensitive opsin exist: **blue, red, and green;**

each closely resembles the opsin proteins of rod cells **but** with somewhat different amino acid sequences. These color photopigments function in much the same manner as rhodopsin, but whereas rhodopsin responds to the entire spectrum of visible light, each iodopsin is sensitive to a much narrower spectrum.

Most people have one red pigment gene and one or more green pigment genes located on each X chromosome.

Although considerable overlap occurs in the wavelength of light to which these pigments are sensitive, each pigment absorbs light of a certain range of wavelengths. As light of a given wavelength, representing a certain color, strikes the retina, all cone cells containing photopigments capable of responding to that wavelength generate action potentials. Because of the overlap among the three types of cones, especially between the green and red pigments, different proportions of cone cells respond to each wavelength, thus allowing color perception over a wide range. Color is interpreted in the visual cortex as combinations of sensory input originating from cone cells. For example, when **orange** light strikes the retina, **99%** of the **red**-sensitive cones respond, **42%** of the **green**-sensitive cones respond, and no blue cones respond. When yellow light strikes the retina, the response is shifted so that a greater number of green sensitive cones respond. The variety of combinations created allows humans to distinguish several million gradations of light and shades of color.

Color vision

Color Blindness

Causes

- Aging
- Macular degeneration
- Glaucoma
- Injury/trauma to the eye
- Certain medications



Definitions

- **Color vision:** Is the capacity of an organism or machine to distinguish objects based on the wavelengths or frequencies of the light they reflect or omit.

- **Color blindness or deficiency:**
A condition in which certain colors cant be distinguished due to absence or deficiency in color receptor cones.

- **Hue** Is the identification of color.
- **Brightness** Is the intensity of color.
- **Saturation** Is the purity of a color.

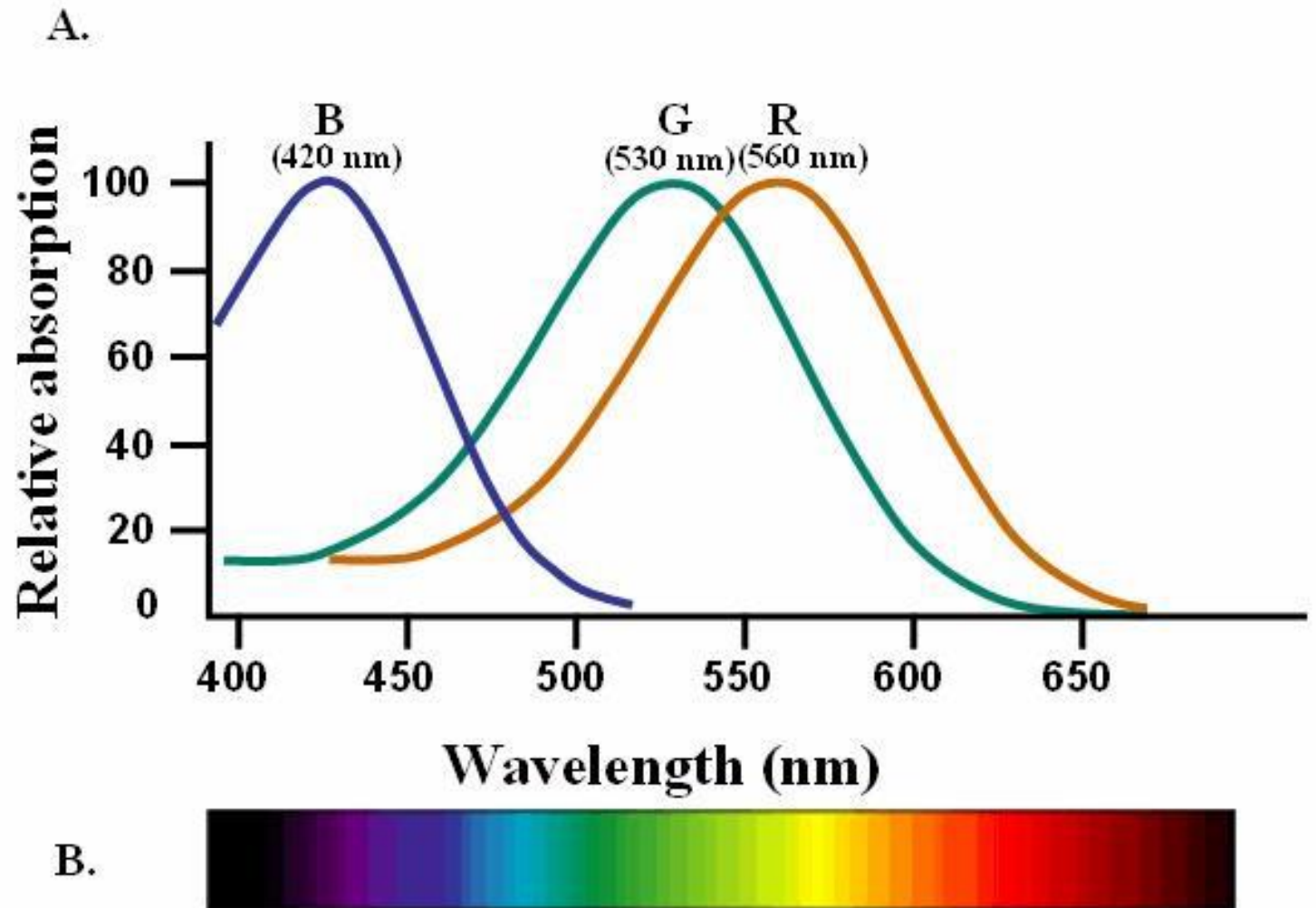
The principle of color vision

Color vision is the function of 3 populations of cones.

Tritan blue at 414-424nm.

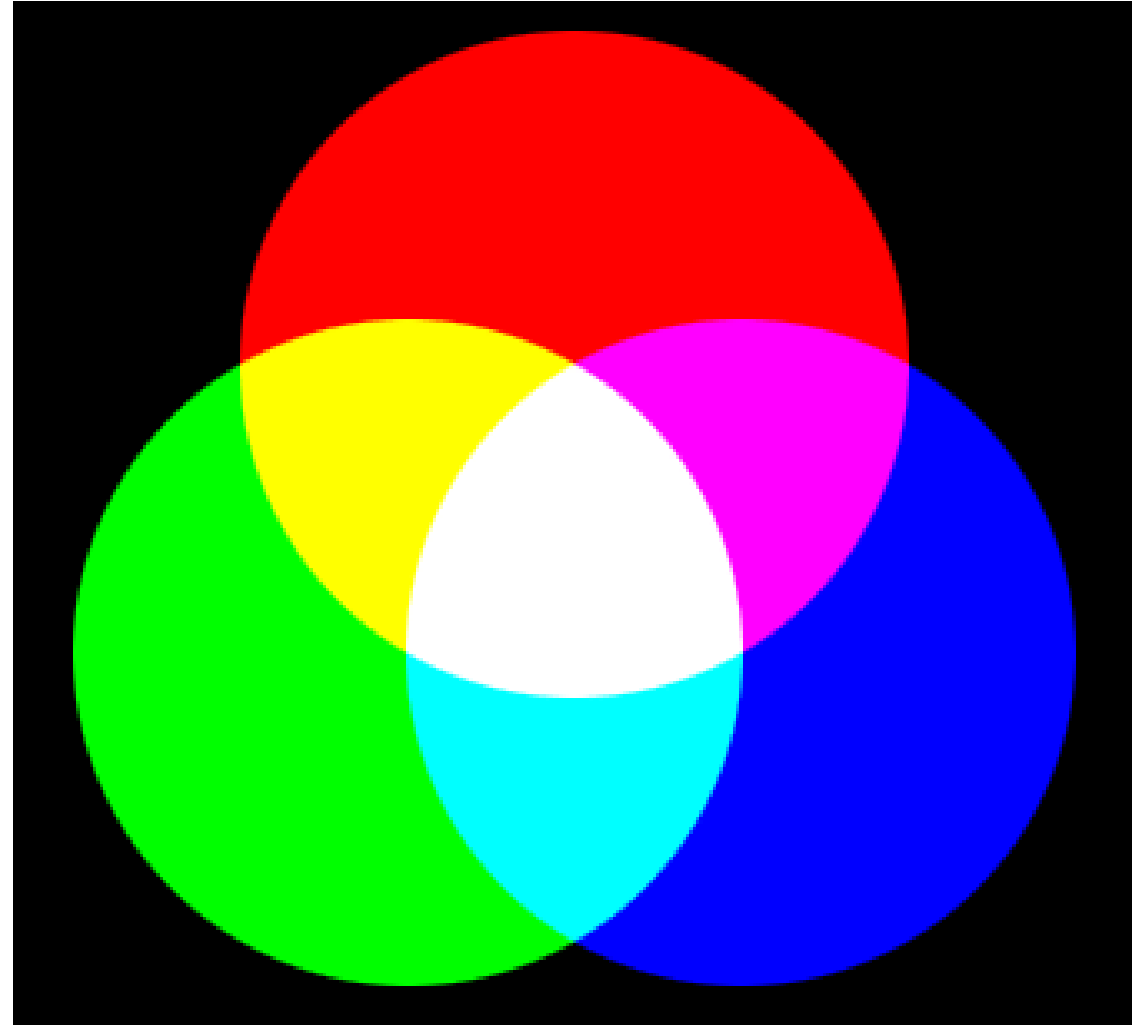
Deutran green 522-539nm.

Protan red 549-570nm.



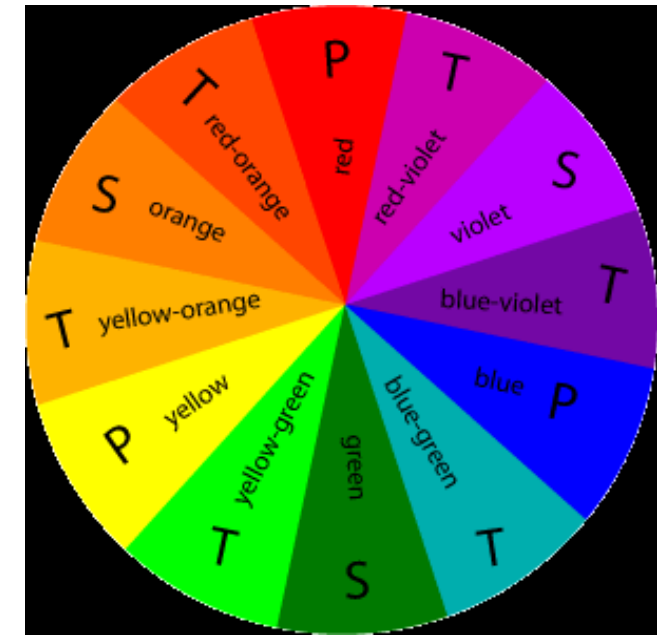
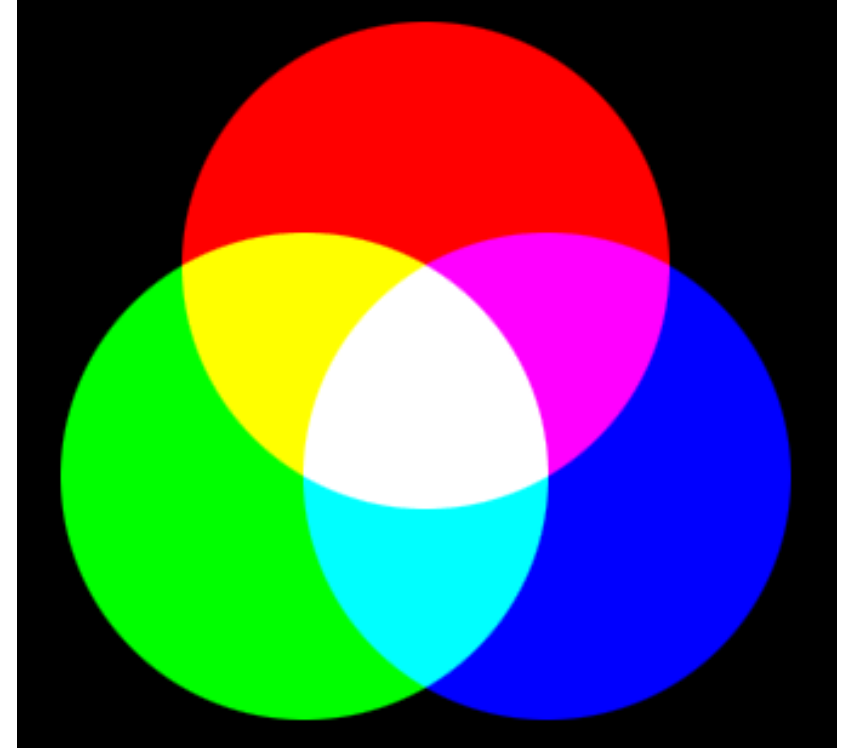
-Short wavelengths causes the green receptor to fire.

- As the wavelength gets longer and closer to 580 nm the red begins to fire, surpassing the green.



Therefore, color vision is the consequence of unequal stimulation of the 3 types of cones. In a specific ratio.

Example : If you stimulate all 3 types of cones about equally the result is white or no color.



Abnormal color vision

- **Color vision abnormality** :is either congenital or acquired.
- **Congenital color vision defect:**
 - Is equal and non progressive.
 - More common in males than females.
 - X linked and almost red green.
- **Acquired color vision defect**
 - Is progressive or regressive.
 - Often involves loss of blue sense.
 - Visual acuity is affected.
 - Common cause is exposure to drugs or toxins as xanthopsia from cardiac glycosides.

Types

At any cone pigment may deficient, Or absent totally.

- **Trichromatism (Normal sight)** Which person can differentiate all colors.
 - All 3 cones although not necessary functioning perfectly.
- **Anomalous trichromatism** can differentiate all colors but on reduced or displaced sensitivities.
 - **Tritanomaly** blue displaced sensitivity .
 - **Deutanomaly** green displaced sensitivity.
 - **Protanomaly** red displaced sensitivity.

- **Dichromatism** absence of one cone
 - **Tritanopia** blue is missing (red ,green are present).
 - **Deutanopia** green is missing (red and blue are present)
 - **Protanopia** red is missing while blue and green are present.
- **Monochromatism** totally unable to dedifferentiate colors of equal brightness.
- **Note:** Patient with **tritanopia** confuse in **yellow** and **blue**, **orange** and **violet**.
 - Most congenital color vision defects are trichromats and use abnormal proportions of 3 colors to match those in spectrum .

How rainbow is seen?

These are the colours of the rainbow and also what they would look like if you were colour blind



This is what a normal person would see as the colours of the rainbow



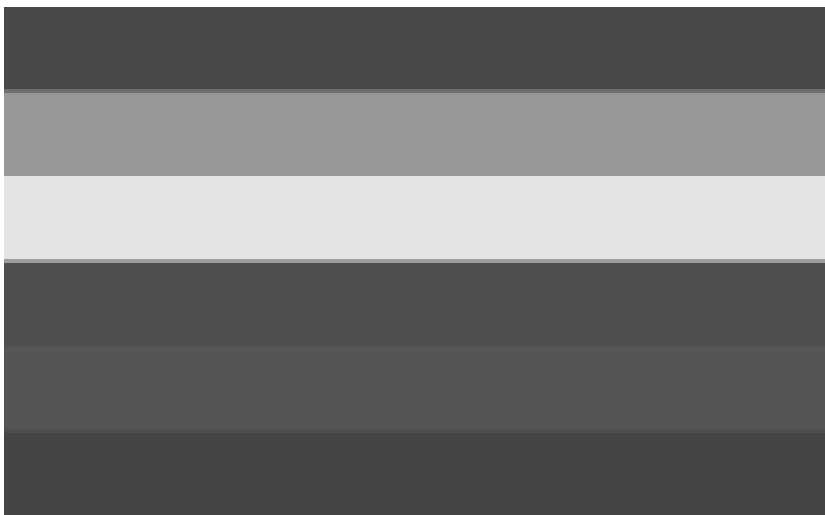
This is the same picture viewed by someone with **protanopia**.



This is the same picture viewed by someone with **deuteranopia**



This is the same picture viewed by someone with
tritanopia



This is the same picture viewed by someone with
Monochromacy which Is full colour blindness

Types of Color Vision tests

- Pseudoisochromatic color confusion charts.

Like Ishihara.

- Hue arrangement task

Like Fransworth Munsell 100 hue test, Farnsworth pand D 15,lanthony desaturated D15.

- Lantern detection tests

Edridge green.

All tests determines congenital and acquired disorders .

Needs more investigations like anomaloscope.

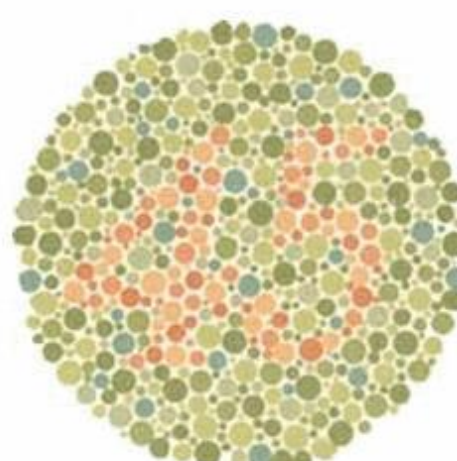
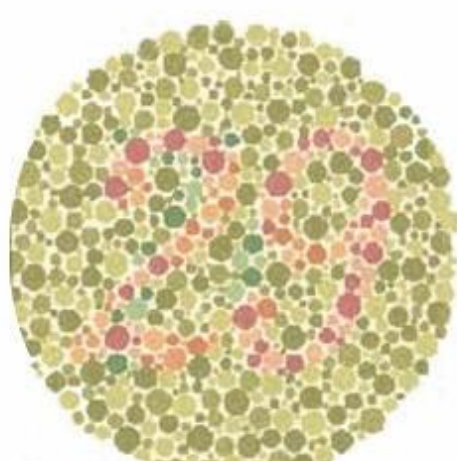
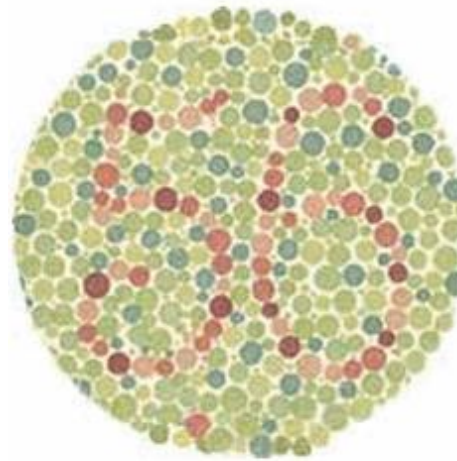
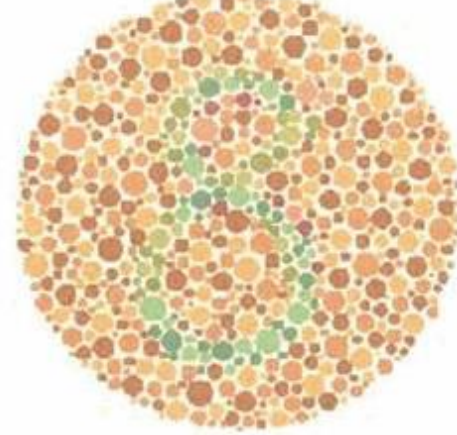
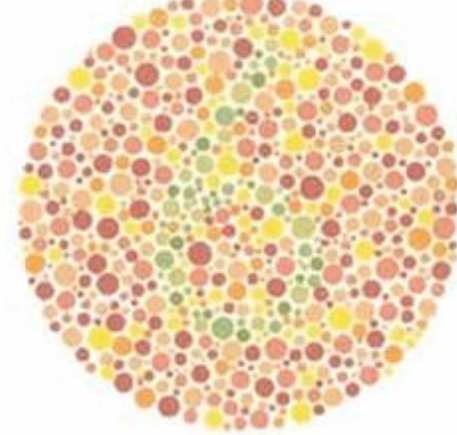
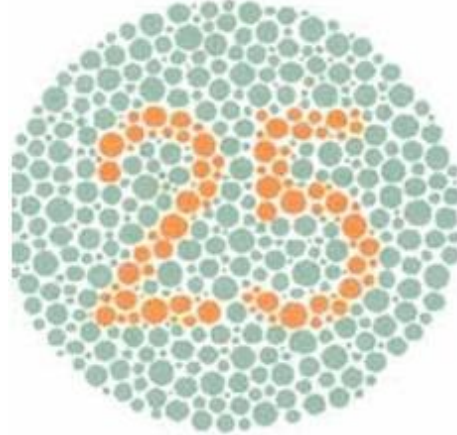
How To Do Tests..!

- Notes for color vision testing
 - Use proper illumination (day light).
 - Explain test for the patient.
 - In screening for congenital diseased test is done binocularly and monocularly for acquired abnormality.
 - Patient should use his or her near correction.
 - Avoid tinted spectacles or contact lenses.

Ishihara Test

-Named by Dr shinobo Ishihara
at Tokyo university.

-Pseudoisochromatic color
plates which are circles in
multiple patterns.



The test consists of 25-30 plate in the complete group.

- The first plate is for demonstration and malingerers.
- Plates from 2-9 are transformation .
- Plates from 10-17 are vanishing.
- Plates from 18-21 are hidden .

- **Procedure:**

1- Test is done at 75 cm at day light at right angle of the visual plane.

2- Allow the patient to see just in 3 sec.

3-If the subject is unable to read numbers on plates 1-17 traces with x endings are used.

- **Recording**

-Write the number of correct attempted as 12/16 correct.

- **Interpretations**

-Normal see them all except hidden digits(18-21).

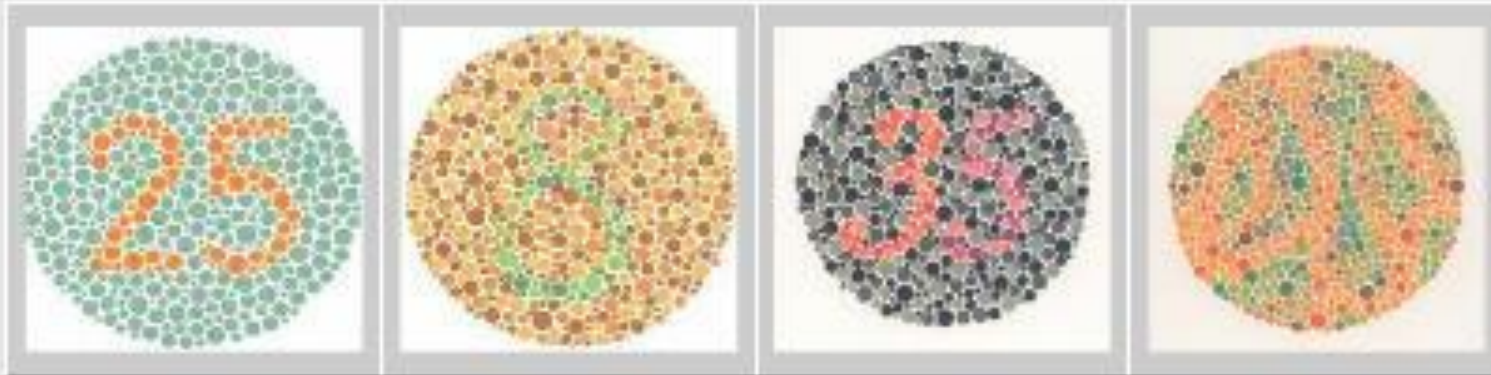
-Red green defect cant see vanishing plates(10-17) but apart of hidden and transforming.

-Plates (22-25) to distinguish deuterans and protans.

-Purple blue plates are not seen by deuterans.

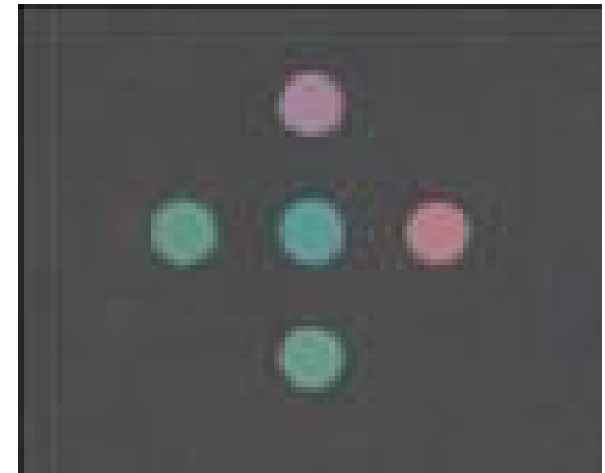
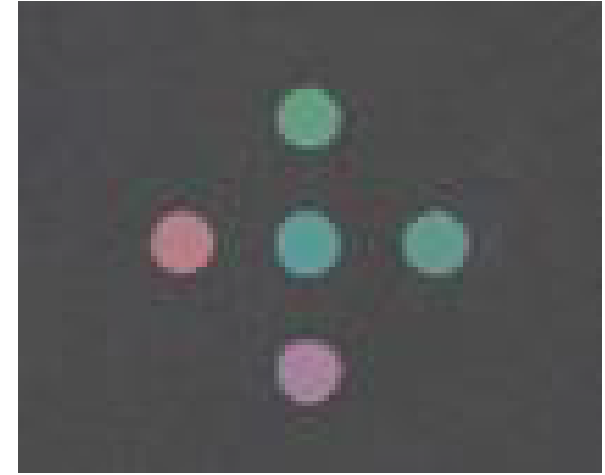
-Red purple are not seen by protans.

Examples of the Ishihara Test.



The City University test TCU

- Procedure
 - Test is done at 35 cm at day light at right angle of the visual plane.
 - It consists of 10 plates each contains four peripheral colored dots with one on the centre.
 - The patient is asked to select the peripheral that most closely matches the central one
 - Results are written as Top(T), Bottom (B),Right (R),Left (L) and score paper is present to analyze defect due to patient response.



Fransworth- Munsell 100 hue test

- Most sensitive test for congenital and acquired but rarely used.
- Consists of 85 hue caps not like named, are in 4 separate racks in each of 2 end caps are fixed while others are loose to be randomized by examiner.
- The box is then closed ,turned upsidedown ,then opened so that markers of the inside caps are visible.

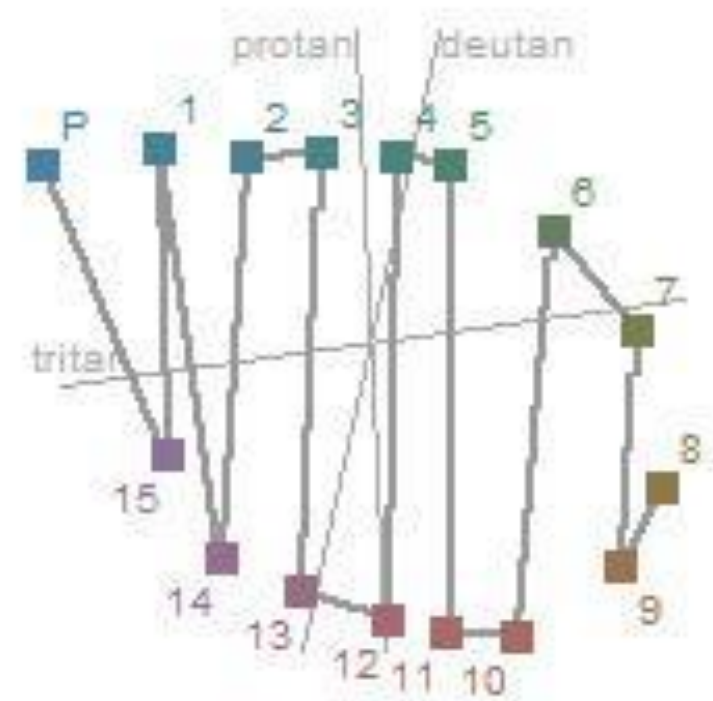


- Recording

- The findings are recorded in cumulative manner on circular chart .
- Each of 3 forms of dichromatism is characterized by failure in specific meridian of the chart.

FransworthD 15

- Test is similar to Farnsworth- Munsell100 hue test but with 15 caps used.
- Test is done at 50 cm on a table.



Protan result

Hardy-Rand- Ritter(HRR)

- Can detect the 3 types of color defects.
- Pseudoisochromatic Subset Book for Red/Green test .
- contains two demonstration plates, four screener plates, and 10 diagnostic series for a total of 16 plates.
 - Each of these plates has its own tab for easy and clean page selection.



Harady –Rand-Ritter

- The test also includes a set of instructions in English and a laminated copy of the score sheet.
- The score sheet can then be copied directly onto the patient's record or copies can be made locally. A pad of score sheets is available separately.



Standard pseudoisochromatic plates part 2

SPP-2

- The test is very similar to Ishihara .

- **Recording**

Place X on the missed plate and write the number of blue yellow , red green defects. If only plate no 4 is missed then repeat the plate.

- **Interpretations**

Ignore mistakes on plate no 3 and 6 because they are difficult and mostly missed.

People over 60 fail the BY part with 2 or more errors. People less than 20 years old fail the BY 2 or more are missed.