Monocular Sensory Processes of Vision:

Color Vision

Acquired Color Vision Defects
Acquired Color Vision Defects

A. Acquired vs. genetic color vision deficiencies

B. Kollner’s Rule

C. Examples of acquired color vision defects

D. S-cone perimetry for diagnosis of glaucoma
Acquired Color Vision Deficiencies

- Color vision deficiencies can be acquired from:
  1. diseases or conditions of the retina, optic nerve, or more posterior visual pathways in the brain.
  2. exposure to toxins and certain drugs.

- Unlike congenital (inherited) color vision defects, conditions that cause acquired defects also often affect visual acuity, are asymmetric from eye to eye, and may change as the disease changes.
Acquired Color Vision Deficiencies

- Acquired color vision defects are referred to as dyschromatopsia – an inability to see or discriminate colors.
- Acquired color vision defects are probably more common than genetic defects, but the incidence is unknown.
- Most acquired color vision defects occur as a result of an ocular or neural disorder affecting the retina or visual pathway.
- The characteristics of acquired defects are not as well defined as genetic defects (the D-15 or 100-Hue are the preferred tests).
- Acquired color vision defects rarely occur in the absence of other signs of pathology.
- Most people who acquire a color vision deficiency retain some ability to perceive all colors and they may not be aware of perceptual alterations.
Acquired Color Vision Deficiencies

- Common causes of acquired color vision defects
  1. Chronic illnesses - Alzheimer’s disease, diabetes mellitus, glaucoma, leukemia, liver disease, chronic alcoholism, macular degeneration, multiple sclerosis, Parkinson’s disease, sickle cell anemia.
  2. Accidents or strokes – damage to the retina or affecting particular areas of the brain/eye.
  3. Medications - such as antibiotics, barbiturates, anti-tuberculosis drugs, high blood pressure medications and several medications to treat nervous disorders.
  4. Industrial or environmental chemicals - carbon monoxide, carbon disulphide and some containing lead can also cause color blindness.
  5. Age – in people over 60 years of age, physical changes can occur which might affect a person’s capacity to see colors.
One of the best known examples of an acquired color vision defect is a tritanomaly caused by a yellowing of the crystalline lens with age.

The figure shows the results of the F-M 100-Hue test before (upper) and after (lower) cataract surgery.

After surgery, patients will often comment on the increased brightness and an increase blue color.
Acquired vs. Genetic Color Vision Deficiencies

1. Acquired color vision defects occur in men and women with the same frequency, while genetic defects occur primarily in men.
2. Patients with hereditary defects rarely misname colors, but the misnaming of colors is characteristic of acquired defects.
3. Hereditary color vision defects are almost always symmetrical between the eyes, acquired defects usually affect one eye more than the other. Patients should be tested monocularly.
4. Most genetic defects are red-green defects, acquired defects may be either red-green or blue-yellow defects, but blue-yellow defects are more common.

Kollner’s rule - Changes in the ocular media and diseases of the retina cause blue-yellow defects. Diseases of the optic nerve and visual pathway cause red-green color vision defects.
# Kollner’s Rule

## Table 1. Acquired color vision defects in diseases of the retina

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Type of color vision defect</th>
<th>Preferred methods of investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>diffuse degenerative</td>
<td>tritan-like</td>
<td>Farnsworth-Munsell tests</td>
</tr>
<tr>
<td>processes</td>
<td></td>
<td>determination of hue discrimination</td>
</tr>
<tr>
<td>intoxication</td>
<td></td>
<td>determination of saturation discrimination</td>
</tr>
<tr>
<td>inflammation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>detachment</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Exceptions*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Type of color vision defect</th>
<th>Preferred methods of investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>central serous retinopathy</td>
<td>protan-like and tritan-like</td>
<td>Nagel anomaloscope, Farnsworth-Munsell tests</td>
</tr>
<tr>
<td>juvenile macular degeneration</td>
<td>protan-like</td>
<td>Nagel anomaloscope, determination of the luminosity curve</td>
</tr>
</tbody>
</table>
# Kollner's Rule

## Table 4. Acquired color vision defects in diseases of the optic nerve

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Type of color vision defect</th>
<th>Preferred methods of investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>retrobulbar neuritis</td>
<td></td>
<td>pseudo-isochromatic plates</td>
</tr>
<tr>
<td>tobacco-alcohol amblyopia</td>
<td></td>
<td>Nagel anomaloscope</td>
</tr>
<tr>
<td>Leber’s optic atrophy</td>
<td>deutan-like</td>
<td>determination of hue discrimination</td>
</tr>
<tr>
<td>neoplasm</td>
<td></td>
<td>determination of the neutral zone</td>
</tr>
<tr>
<td>trauma</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Exceptions

<table>
<thead>
<tr>
<th>Description</th>
<th>Type of color vision defect</th>
<th>Preferred methods of investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>glaucoma</td>
<td>tritan-like</td>
<td>Farnsworth-Munsell tests</td>
</tr>
<tr>
<td></td>
<td></td>
<td>determination of hue discrimination</td>
</tr>
<tr>
<td>infantile optic atrophy with dominant inheritance</td>
<td>tritan-like</td>
<td>Panel D-15 test</td>
</tr>
<tr>
<td></td>
<td></td>
<td>determination of hue discrimination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>determination of the neutral zone</td>
</tr>
<tr>
<td>rare type of the above degenerative disease</td>
<td>protan-like</td>
<td>Nagel anomaloscope</td>
</tr>
</tbody>
</table>
5. Hereditary color vision defects are constant throughout life, acquired defects change with progression or treatment of the underlying condition.

6. Hereditary color vision defects are not usually associated with other pathology, acquired defects are associated with other signs or symptoms of the underlying pathology (reduced visual acuity, visual field defects).

7. Tests of acquired color vision defects may be useful in early diagnosis or assessment of stage of disease.
Examples of Acquired Color Vision Defects

- Examples of spectral sensitivity functions for single flash (solid line) to assess parvocellular pathway function, or 25 Hz flicker (dashed line) to assess magnocellular pathway function, for normal and acquired color vision defects.

- Patients with optic atrophy or retrobulbar neuritis may have specific defects in the parvocellular neural pathway.

Defects Caused by Retinal and Optic Nerve Disease

• The effects of retinal diseases may be different, depending on their primary locus of sensitivity loss.
• RP affects outer retina, OAG affects inner retina, IDDM affects whole retina.
• In this study, IDDM caused the most selective loss for the S-cone system (S-cone vulnerability).
Red Cap Tests for Color Vision Defects in Optic Neuritis

- Color sensitivity is often altered in optic nerve disease.
- A simple test of red saturation is the “red cap” test - red-colored objects may appear washed-out or faded and may be described as appearing orange or pink.
- In addition, the patient should be asked to describe the percentage saturation of the bottle cap.
Selective Vulnerability of S-cones in Glaucoma

- Selective color vision defects in early glaucoma could have considerable benefit for the initial diagnosis and institution of treatment.

- The example of increment-threshold spectral sensitivity shows a comparison of an eye with (filled symbols) and without (open symbols) experimental glaucoma.

- The eye with glaucoma shows a loss of sensitivity only for the S-cone mechanism.
A visual field plot with examples of spectral response for control and treated eyes.

Even in areas of deep visual field defects -
- A 460 nm stimulus isolates the SW-mechanism.
- A 620 nm stimulus isolates the R/G-mechanism.
Early Diagnosis and Progression of Glaucoma

Perimetry based on thresholds of S-cones (SWAP - Short-Wavelength Automated Perimetry) is more sensitive to visual field defects caused by glaucoma than the standard procedure based on white light luminance increment thresholds.

SWAP became a standard clinical procedure for glaucoma diagnosis, but recently has become less utilized because of test-retest variability.
Color Vision Defects Caused by Age-Related Macular Degeneration

- The pigmented epithelium and receptors in central visual field are lost, causing acuity and color vision loss.
- Early cases seem to indicate a selective defect in the S-cone mechanism, but with more advanced cases, all of the color vision mechanisms are affected equally.
Color Vision in AIDS Patients without HIV Retinopathy

• Signs of visual dysfunction prior to HIV retinopathy would be helpful in determining the state of progression.

• Rayleigh matches are normal for aids patients (upper graph), but the matching range may be extended towards the red end of the range (lower graph).

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Monocular Sensory Processes of Vision:

Color Vision

A. Introduction - Normal color vision
B. Colorimetry
C. Retinal fundamentals
D. Neural mechanisms
E. Genetic color vision anomalies
F. Clinical color vision tests
G. Acquired color vision defects
H. Color Perception
  1. Chromatic discrimination (hue and saturation) for normal and defective color vision
  2. Color mixture and appearance
  3. Color contrast, constancy, and adaptation
  4. Color specification and colorimetry (CIE)
  5. Spectral sensitivity of normal and defective color vision
  6. Mechanisms of color deficiencies
  7. Inherited anomalies of color vision
     a. Classification
     b. Inheritance patterns
     c. Color vision tests (e.g., pseudoisochromatic tests, arrangement tests, anomaloscope)
  8. Acquired anomalies of color vision
     a. Classification
     b. Etiology
     c. Color vision tests
  9. Conditions for color vision testing
  10. Societal implications of color vision anomalies
      a. School
      b. Vocational requirements
      c. Patient interest
  11. Patient management strategies
      a. Counseling
      b. Special aids