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CHILL MEDICOS

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SUBSCRIBE



PHOTOCHEMISTRY OF VISION

- PHOTSENSITIVE pigments present in RODS & CONES CONCERNED WITH
↓
CHEMICAL BASIS OF VISUAL PROCESS
↓
PHOTOCHEMICAL CHANGES IN VISUAL RECEPTOR CELLS
↓
CALLED WALD VISUAL CYCLE.
- CHEMICAL REACTIONS INVOLVED IN pigMENTS LEAD TO
↓
DEVELOPMENT OF ELECTRICAL ACTIVITY IN RETINA
↓
GENERATION OF IMPULSES (ACTION POTENTIAL)
↓
TRANSMITTED THROUGH OPTIC NERVE.

★ RHODOPSIN →

- VISUAL PURPLE IS pigment (photosensitive) OF ROD CELLS.
- PRESENT IN MEMBRANOUS DISKS LOCATED IN OUTER SEGMENT OF ROD CELLS.
- RHODOPSIN IS A CONJUGATED PROTEIN.
- MADE UP OF PROTEIN CALLED **opsin** AND **CHROMOPHORE**.

↳ PRESENT IN RHODOPSIN CALLED **scotopsin**.

↳ CHEMICAL SUBSTANCE DEVELOPS COLOR IN CELL.

↳ PRESENT IN RHODOPSIN/ROD CELLS IS CALLED **RETINAL**

↓
ALDEHYDE OF vit. A & RETINOL.

RETINAL DERIVED FROM
CAROTINOID SUBSTANCE (β-CAROTENE)
PRESENT IN CARROTS.

★ RETINAL PRESENT IN FORM OF 11-CIS RETINAL CALLED **RETININE 1** (in human eye)

↓
ONLY THIS FORM COMBINES WITH SCOTOPSIN TO SYNTHESISE **RHODOPSIN**

★ WALD VISUAL CYCLE →

↓
IT IS PHOTOCHEMICAL CHANGES IN RHODOPSIN.

- RETINA IS ISOLATED & EXAMINED IN DARK ↓

DARK → RODS APPEAR RED

DURING LIGHT → RODS BECOME YELLOW.

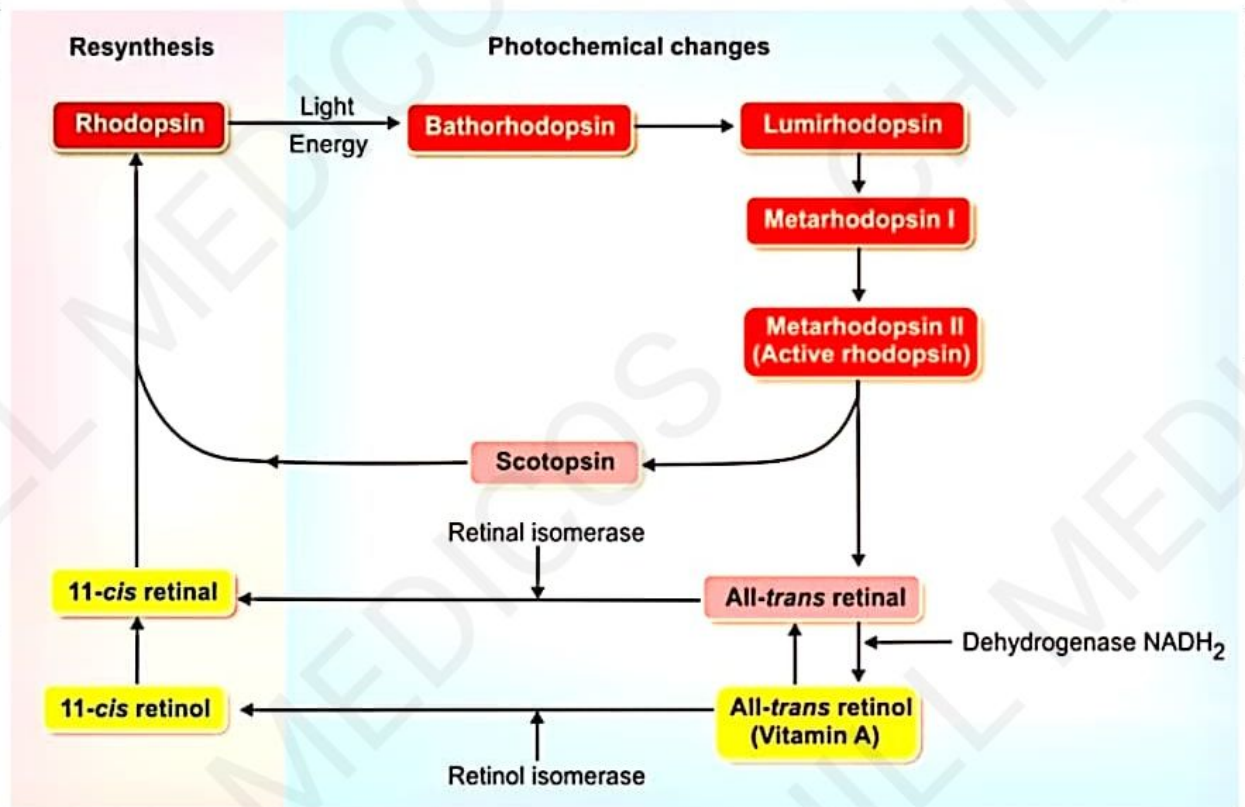


FIGURE 166.2: Photochemical changes and resynthesis of rhodopsin (Wald visual cycle).
 NADH_2 = Reduced nicotinamide adenine dinucleotide.

★ SYNTHESIS OF RHODOPSIN FROM 11-CIS RETINAL (RETININE) FASTER THAN FROM 11-CIS RETINOL (vit. A).

★ PHOTOTRANSDUCTION →

↳ VISUAL OR PHOTOTRANSDUCTION IS PROCESS BY WHICH LIGHT ENERGY CONVERTED INTO RECEPTOR POTENTIAL IN VISUAL RECEPTORS.

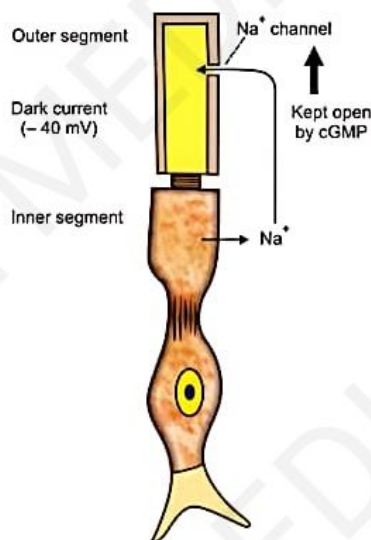


FIGURE 166.3: Maintenance of dark current (resting potential) in outer segment of rod cell

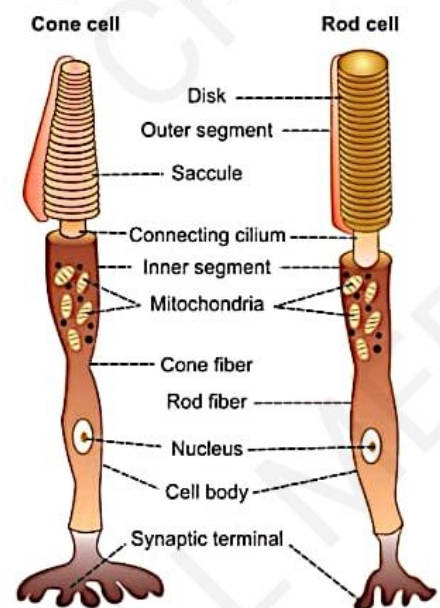


FIGURE 166.1: Structure of visual receptors

- RMP IN SENSORY RECEPTOR CELLS IS B/W (-70mv to -90mv).
- DURING DARKNESS → IN VISUAL RECEPTORS NEGATIVITY IS REDUCED.
 - ↓
 - RMP IS ABOUT -40mv. [BECZ OF INFLUX OF Na^+]
 - ↓
 - IN DARK, Na^+ IONS PUMPED OUT OF INNER SEGMENTS OF ROD CELL TO ECF.
 - ↓
 - Na^+ IONS LEAK BACK INTO ROD CELLS THROUGH OUTER SEGMENT
 - ↓
 - REDUCE ELECTRONEGATIVITY INSIDE ROD CELL.
 - ↓
 - POTENTIAL CONSTANT & ALSO CALLED DARK CURRENT.
- * INFUX OF Na^+ IONS BECZ OF CYCLIC GUANOSINE MONOPHOSPHATE (cGMP).
 - ↓
 - ALWAYS KEEPS Na^+ CHANNEL OPENED.

* PHOTOTRANSDUCTION CASCADE OF RECEPTOR POTENTIAL →

↳ WHEN OTHER SENSORY RECEPTORS EXCITED, ELECTRICAL RESPONSE IN FORM OF DEPOLARIZATION
 BUT IN VISUAL RECEPTORS, RESPONSE IS IN FORM OF HYPERPOLARIZATION.

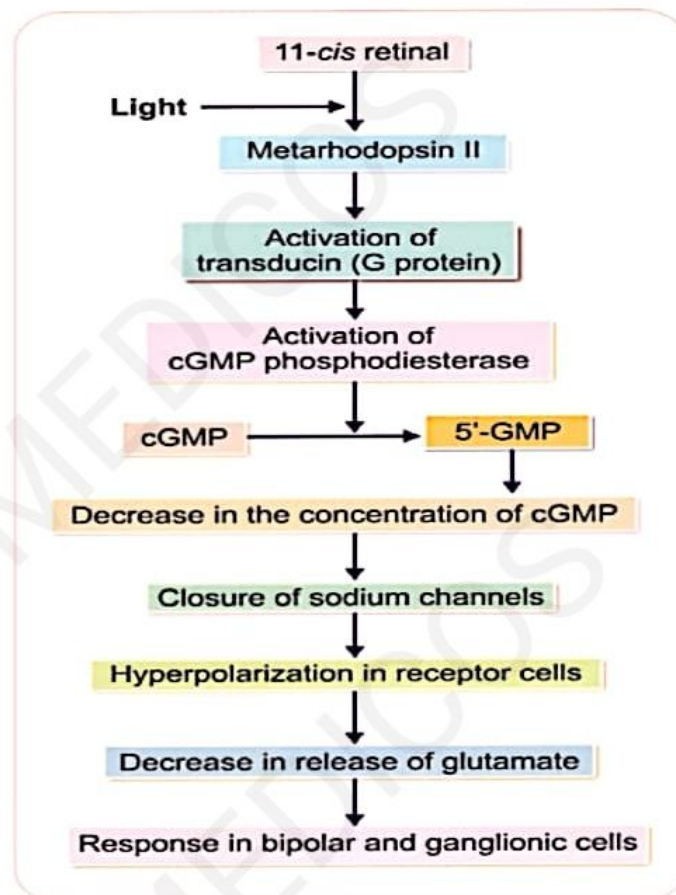


FIGURE 166.4: Phototransduction cascade.
 cGMP = Cyclic guanosine monophosphate.

DARK ADAPTATION

★ DEFINITION → DARK ADAPTATION IS PROCESS BY WHICH PERSON ABLE TO SEE OBJECTS IN DIM LIGHT.
↳ MAX. DURATION IS ABOUT 20 min.

★ CAUSES → 1) ↑ SENSITIVITY OF RODS RESULT OF RESYNTHESIS OF RHODOPSIN
↓
↳ IN DIM LIGHT, IT REQUIRES SOME TIME FOR REGENERATION OF CERTAIN AMOUNT OF RHODOPSIN
↳ DARK ADAPTATION OCCURS IN CONES ALSO.
2) DILATION OF PUPIL →
↓
ALLOWS MORE & MORE LIGHT TO ENTER EYE.

★ RADIOLOGISTS, AIRCRAFT PILOTS & OTHERS (NEED MAX. VISUAL SENSITIVITY IN DIM LIGHT).
↓
WEAR RED GLASS BECOZ
↓
RED LIGHT OF SPECTRUM STIMULATES RODS SLIGHTLY WHILE CONES ALLOWED TO FUNCTION WELL.
↓
RED GOGGLES SEE WELL IN BRIGHT-LIGHTED AREA & ALSO IN DIM-LIGHTED AREA.

★ DARK-ADAPTATION CURVE → • CURVE THAT DEMONSTRATES THE RELATIONSHIP B/W THRESHOLD OF LIGHT STIMULUS & TIME IN DARK.

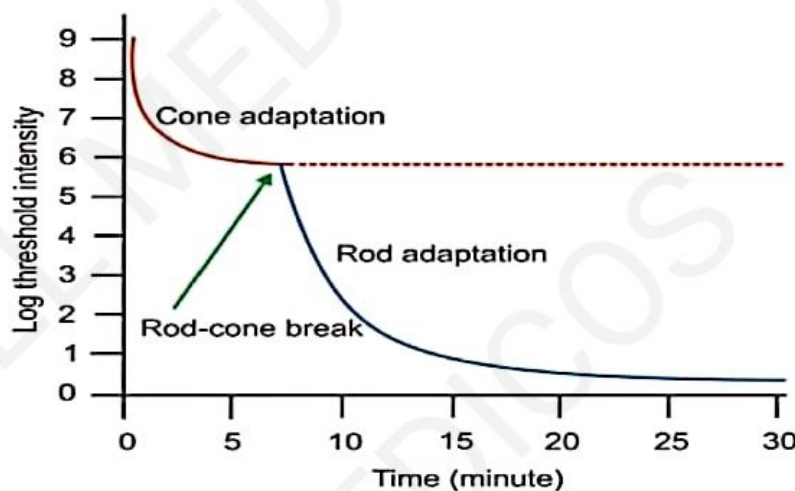
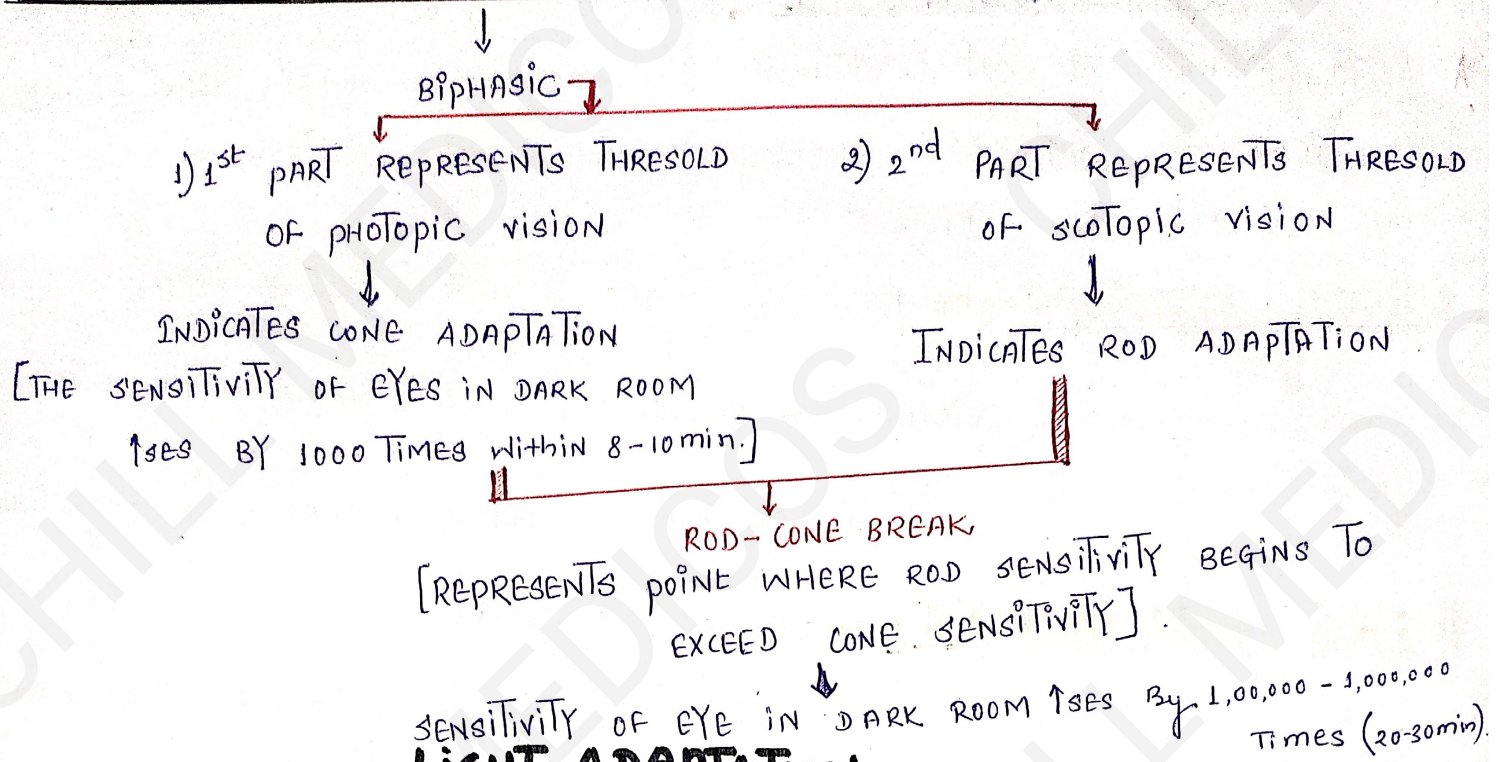


FIGURE 166.5: Dark adaptation curve. During cone adaptation, threshold decreases by 3 logs and sensitivity of the eyes increases 1,000 times. During rod adaptation, threshold decreases by 6 logs and sensitivity of the eyes increases by 1,000,000.

★ PARTS OF DARK ADAPTATION CURVE



LIGHT ADAPTATION

• ROD ADAPTATION → GRADUAL ↓SE IN THRESHOLD & COMPLETE IN 20-30 MINUTE

★ DEFINITION → PROCESS IN WHICH EYES GET ADAPTED TO ↑SED ILLUMINATION.

↓

IT IS MERE DISAPPEARANCE OF DARK ADAPTATION

↓

MAX. PERIOD IS ABOUT 5 MINUTES.

★ CAUSES → 1) REDUCED SENSITIVITY OF RODS

↓

DUE TO BREAKDOWN OF RHODOPSIN.

2) CONSTRICTION OF PUPIL

↓

REDUCES QUANTITY OF LIGHT RAYS ENTERING EYE.

■ ACUITY OF VISION

■ DEFINITION

Acuity of vision is the ability of eye to determine the precise shape and details of the object. It is also called **visual acuity**. Acuity of vision is also defined as the ability to recognize the separateness of two objects placed together. Cones of retina are responsible for acuity of vision. Visual acuity is highly exhibited in fovea centralis, which contains only cones. It is greatly reduced during the refractory errors.

■ TEST FOR VISUAL ACUITY

Acuity of vision is tested for distant vision as well as near vision. If there is any difficulty in seeing the distant object or the near object, the defect is known

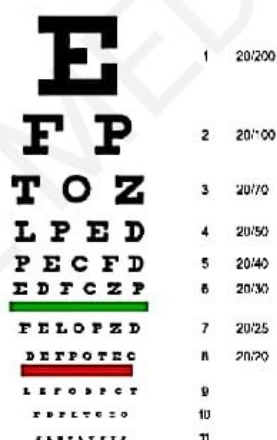
as **error of refraction**. Refractive errors are described separately in Chapter 171.

Distant Vision

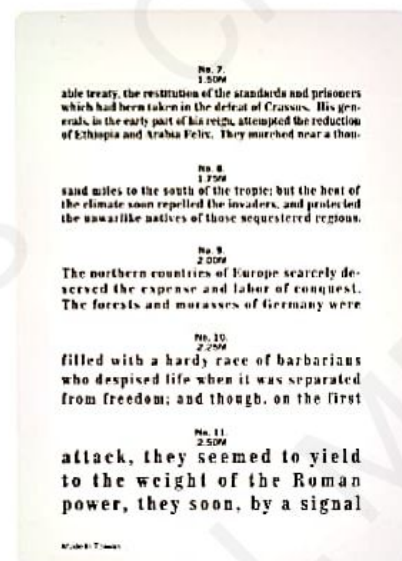
Snellen chart is used to test the acuity of vision for distant vision in the diagnosis of refractive errors of the eye.

Near Vision

Jaeger chart is used to test the visual acuity for near vision.



Snellen Chart



Jaeger Chart

VISUAL PATHWAY

★ VISUAL PATHWAY OR OPTIC PATHWAY IS NERVOUS PATHWAY THAT TRANSMITS IMPULSES FROM RETINAL VISUAL CENTER IN CEREBRAL CORTEX.

■ VISUAL RECEPTORS → RODS & CONES IN RETINA FORM VISUAL RECEPTOR
↓
FIBERS FROM VISUAL RECEPTORS SYNAPSE WITH DENDRITES
↓
OF BIPOLAR CELLS OF OUTER NUCLEAR LAYER OF RETINA.

FIRST ORDER NEURONS → PRIMARY NEURONS ARE BIPOLAR CELLS
↓
AXON FROM BIPOLAR C. SYNAPSE WITH DENDRITE OF GANGLIONIC CELLS.

SECOND ORDER NEURONS → SECONDARY NEURONS ARE GANGLIONIC CELLS
↓
AXONS OF GANGLIONIC CELLS FORM OPTIC NERVE
↓
OPTIC NERVE TERMINATES IN LATERAL GENICULATE BODY.

★ THIRD ORDER NEURONS → IN LATERAL GENICULATE BODY.
↓
FIBERS FROM HERE, REACH VISUAL CORTEX.

COURSE OF VISUAL PATHWAY →

↓
CONSISTS OF SIX COMPONENTS

- a) OPTIC NERVE
- b) OPTIC CHIASMA
- c) OPTIC TRACT
- d) LATERAL GENICULATE BODY
- e) OPTIC RADIATION
- f) VISUAL CORTEX.

1. OPTIC NERVE →
• FORMED BY AXONS OF GANGLIONIC CELLS.
• LEAVES EYE THROUGH OPTIC DISK.

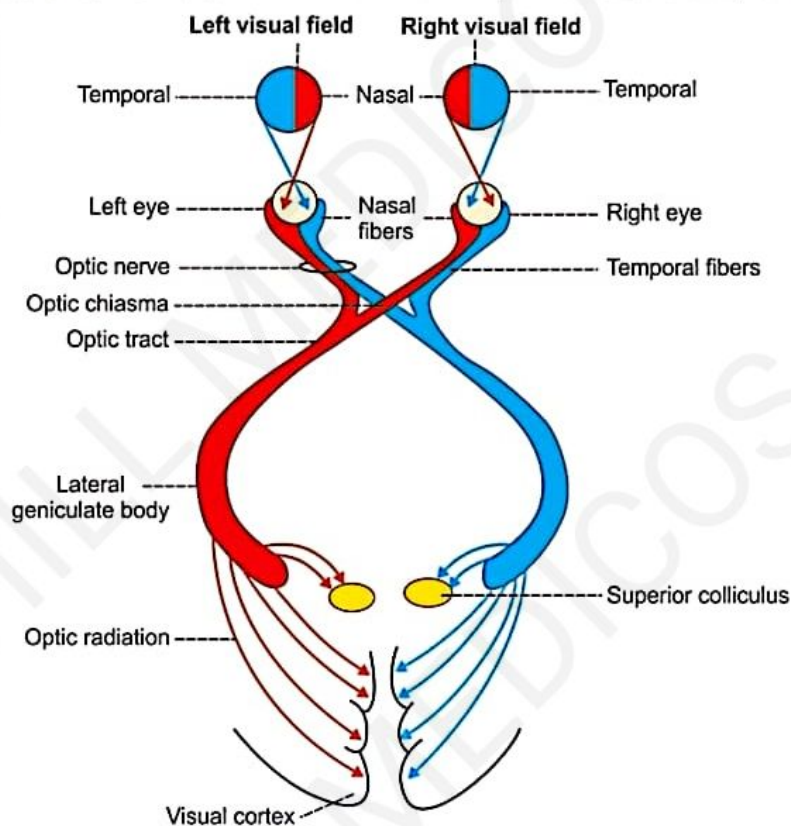


FIGURE 168.1: Visual pathway

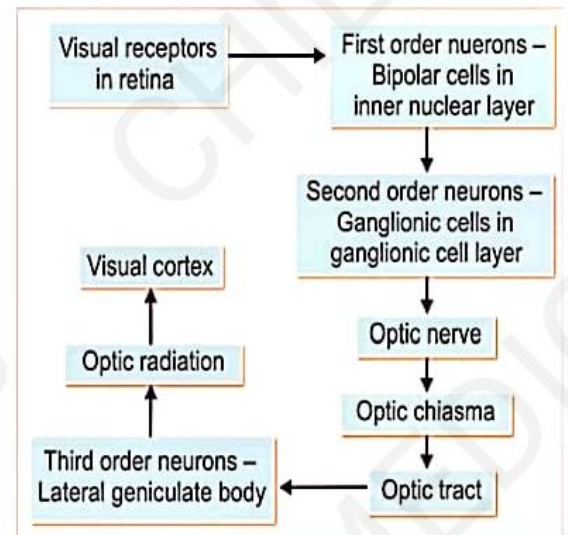


FIGURE 168.2: Representation of visual pathway

2. Optic Chiasma → • MEDIAL FIBERS OF EACH OPTIC NERVE CROSS MIDLINE

↓
JOIN THE UNCROSSED LATERAL FIBERS OF OPPOSITE SIDE

↓
TO FORM THE OPTIC TRACT

3. Optic Tract → • FORMED BY UNCROSSED FIBERS OF OPTIC NERVE ON SAME SIDE

↓
CROSSED FIBERS OF OPTIC NERVE FROM OPPOSITE SIDE.

★ ALL FIBERS OF OPTIC TRACT RUN BACKWARD, OUTWARD, TOWARDS CEREBRAL PEDUNCLE.

↓
FIBERS TURN AROUND PEDUNCLE TO REACH LATERAL GENICULATE BODY IN THALAMUS

★ FIBERS FROM FOVEA DO NOT ENTER SUPERIOR COLICULUS.

★ LEFT OPTIC TRACT [IMPULSES FROM TEMPORAL OF LEFT RETINA + NASAL OF RT RETINA]

↳ RESPONSIBLE FOR VISION IN NASAL 1/2 OF LEFT VISUAL FIELD

AND TEMPORAL 1/2 OF RIGHT VISUAL FIELD.

↳ and VICE - VERSA.

4. LATERAL GENICULATE BODY →

- FIBERS OF OPTIC TRACT TERMINATE IN LATERAL GENICULATE BODY
↓
FORMS THE SUBCORTICAL CENTER FOR VISUAL SENSATION.

↓
THE GENICULOCALCARINE TRACT OR OPTIC RADIATION ARISES.

↓
TRACT IS LAST RELAY OF VISUAL PATHWAY.

- SOME OF FIBERS FROM OPTIC TRACT TERMINATE IN ONE OF FOLLOWING CENTERS:-
 - 1) SUPERIOR COLICULUS [CONCERNED WITH REFLEX MOVEMENTS OF EYEBALLS & HEADS]
 - 2) PRETECTAL NUCLEUS [CONCERNED WITH LIGHT REFLEXES]
 - 3) SUPRAOPTIC NUCLEUS OF HYPOTHALAMUS [CONCERNED WITH RETINAL CONTROL OF PITUITARY IN ANIMAL]

5. OPTIC RADIATION →

• FIBERS FROM LATERAL GENICULATE BODY
PASS THROUGH INTERNAL CAPSULE

↓
FORM OPTIC RADIATION

↓
ENDS IN VISUAL CORTIX.

6. VISUAL CORTIX → PRIMARY CORTICAL CENTER FOR VISION CALLED VISUAL CORTIX.

↓
LOCATED ON MEDIAL SURFACE OF OCCIPITAL LOBE.

• AREAS OF VISUAL CORTIX →

- 1) PRIMARY VISUAL AREA (Area 17) → PERCEPTION OF VISUAL IMPULSE
- 2) SECONDARY VISUAL AREA / VISUAL ASSOCIATION AREA (Area 18) → INTERPRETATION OF VISUAL IMPULSE
- 3) OCCIPITAL EYE FIELDS (Area 19) → MOVEMENTS OF EYES.

... EFFECTS OF LESION OF VISUAL PATHWAY ...

- INJURY TO ANY PART OF OPTIC PATHWAY CAUSES VISUAL DEFECT & NATURE OF DEFECT DEPENDS UPON LOCATION & EXTENT OF INJURY.
- LOSS OF VISION IN ONE VISUAL FIELD

↓
CALLED ANOPIA.

- LOSS OF VISION IN ONE HALF OF VISUAL FIELD CALLED HEMIANOPIA.

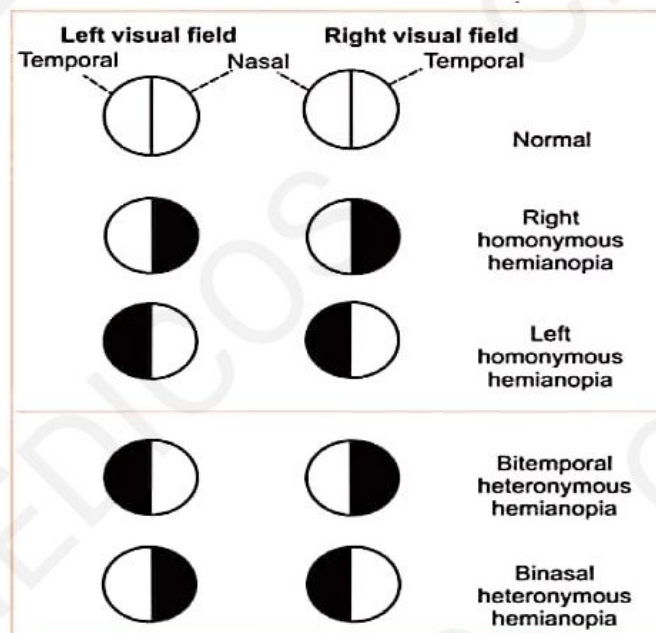
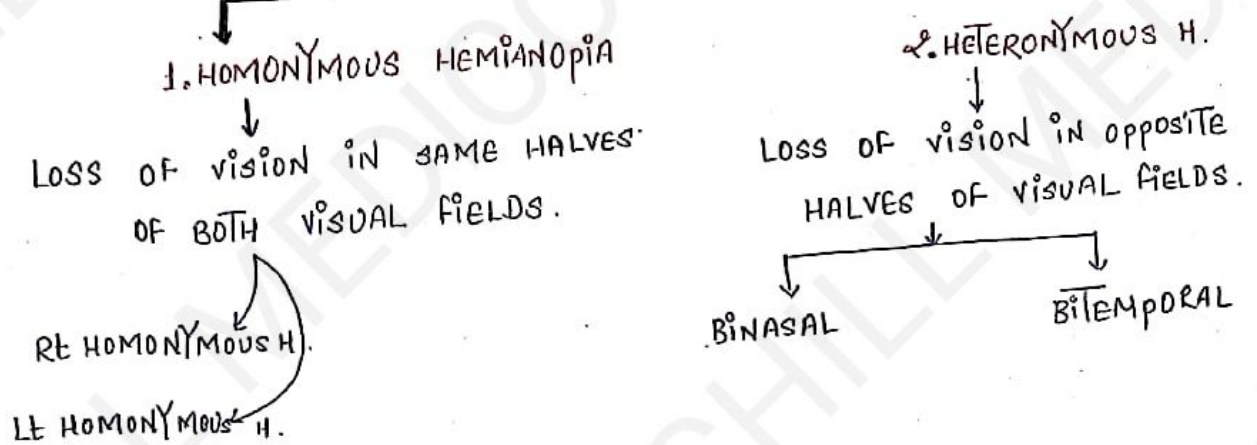


FIGURE 168.3: Types of hemianopia

1. EFFECTS OF LESION OF OPTIC NERVE →

- LESION IN ONE OPTIC NERVE CAUSE TOTAL BLINDNESS.
↓
OR ANOPIA IN CORRESPONDING VISUAL FIELD.
- LESION OCCURS DUE TO ↑ ICD INTRACRANIAL PRESSURE.

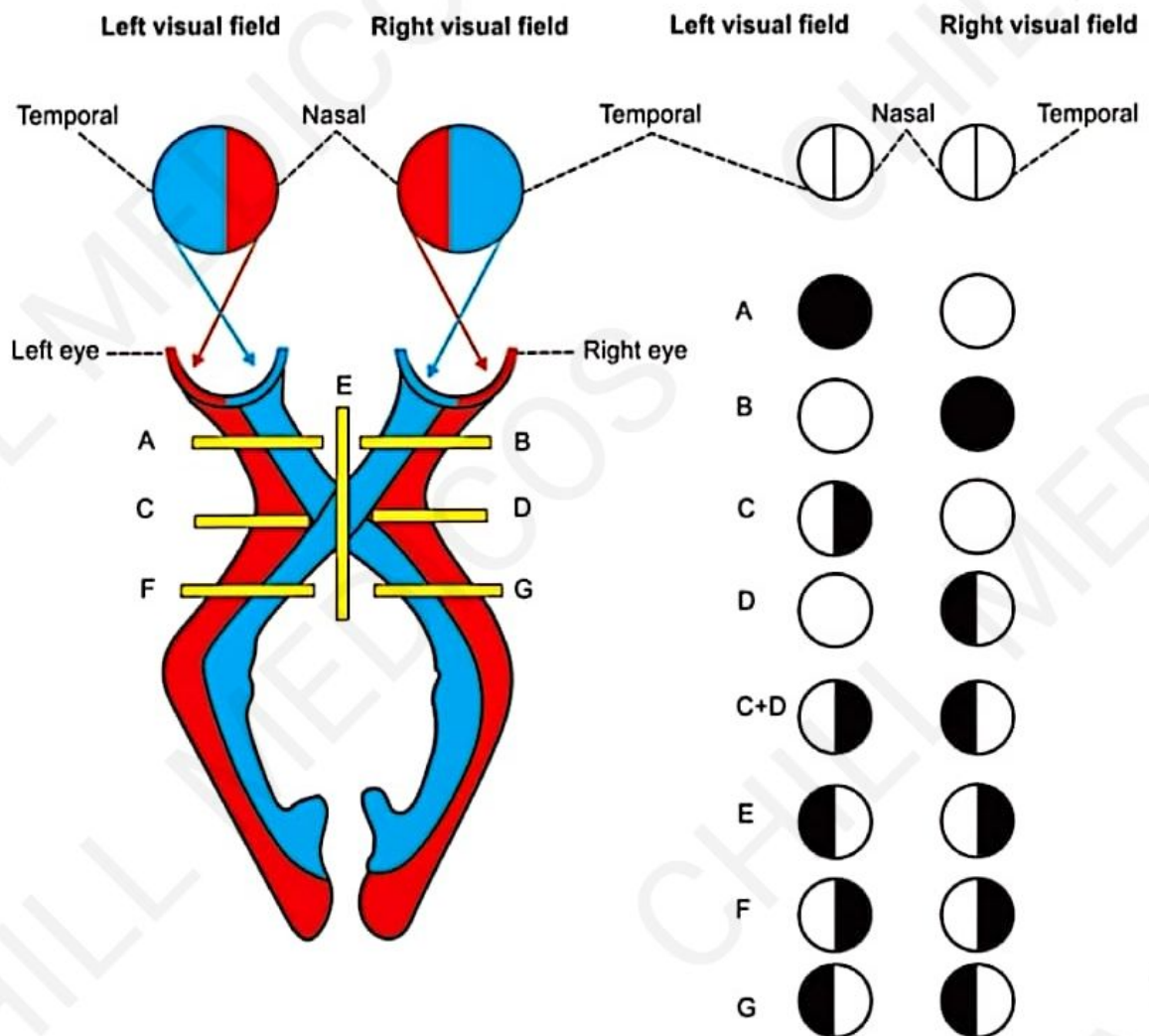


FIGURE 168.4: Effects of lesions of optic pathway. Dark shade in circles indicates blindness.

- A. Lesion of left optic nerve: Total blindness of left eye
- B. Lesion of right optic nerve: Total blindness of right eye
- C. Lesion of lateral fibers in left side of optic chiasma: Left nasal hemianopia
- D. Lesion of lateral fibers in right side of optic chiasma: Right nasal hemianopia
- C + D. Lesion of lateral fibers in both sides of optic chiasma: Binasal hemianopia
- E. Lesion of medial fibers in optic chiasma: Bitemporal hemianopia
- F. Lesion of left optic radiation: Right homonymous hemianopia
- G. Lesion of right optic radiation: Left homonymous hemianopia.

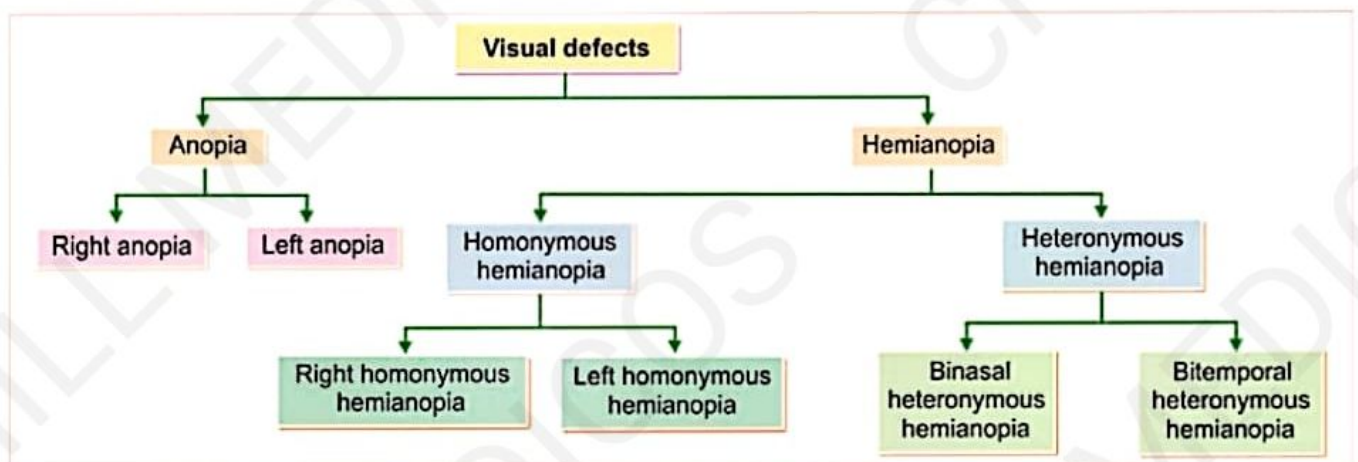


FIGURE 168.5: Visual defects

2. EFFECTS OF LESION OF OPTIC CHIASMA →

1) LEFT OR RIGHT NASAL HEMIANOPIA →

PRESSURE ON ^{LATERAL} UNCROSSED FIBERS BY ANEURYSMAL DILATATION

↓
OF CAROTID ARTERY CAUSES

↓
BLINDNESS IN TEMPORAL PART OF RETINA OF SAME SIDE.

↓
HEMIANOPIA DEVELOPED.

2) BINASAL HEMIANOPIA → If LATERAL FIBERS OF BOTH SIDES AFFECTED

↓
VISION IS LOST IN NASAL $\frac{1}{2}$ OF BOTH VA

↓
DUE TO DILATED 3rd VENTRICLE / DILATATION OF CAROTID ARTERY ON BOTH SIDES.

3) BITEMPORAL HEMIANOPIA → COMPRESSION OF NASAL FIBERS [CROSSED FIBERS BY PITUITARY TUMOR

3. EFFECTS OF LESION OF —

1) OPTIC TRACT

2) LATERAL GENICULATE BODY

3) OPTIC RADIATION

- CAUSES HOMONYMOUS HEMIANOPIA
 - ↳ 1) RT HOMONYMOUS H. (LT - SIDED LESION)
 - ↳ 2) LT HOMONYMOUS H. (RT - SIDED LESION)

4. EFFECTS OF LESION OF VISUAL CORTX →

- CAUSES INFERIOR OR SUPERIOR HOMONYMOUS HEMIANOPIA.

★ MACULAR SPARING → IN ALL CONDITIONS ABOVE, TOTAL BLINDNESS DOES NOT OCCUR BECOZ

↓
MACULAR VISION IS NOT LOST IN COND^N OF HEMIANOPIA.
↓
MACULAR SPARING.

- ★ BECOZ OF →
- 1) FIBERS FROM MACULA PROJECT INTO VISUAL CORTX OF BOTH SIDES.
 - 2) FIBERS FROM MACULAR REGION PROJECTED INTO BOTH ANT. and post. PARTS OF EACH VISUAL CORTX.

★ ONLY BILATERAL LESION OF VISUAL CORTX CAUSE TOTAL BLINDNESS.

*** ACCOMMODATION REFLEX ***

• ACCOMMODATION IS A REFLEX ACTION. WHEN A PERSON LOOKS AT A NEAR OBJECT AFTER SEEING A FAR OBJECT.

↓
THREE ADJUSTMENTS ARE MADE IN EYEBALLS.
→ CONVERGENCE OF EYEBALLS DUE TO CONTRACTION OF MEDIAL RECTI
→ CONSTRICTION OF PUPIL DUE TO CONTRACTION OF CONSTRICTOR PUPILLAE OF IRIS.
→ ↑ IN ANTERIOR CURVATURE OF LENS DUE TO CONTRACTION OF CILIARY MUSCLE.

• ACCOMMODATION REFLEX INVOLVES BOTH SKELETAL MUSCLE (medial recti) and SMOOTH MUSCLE (CILIARY M. & SPHINCTER PUPILLAE).

* PATHWAY FOR ACCOMMODATION REFLEX →

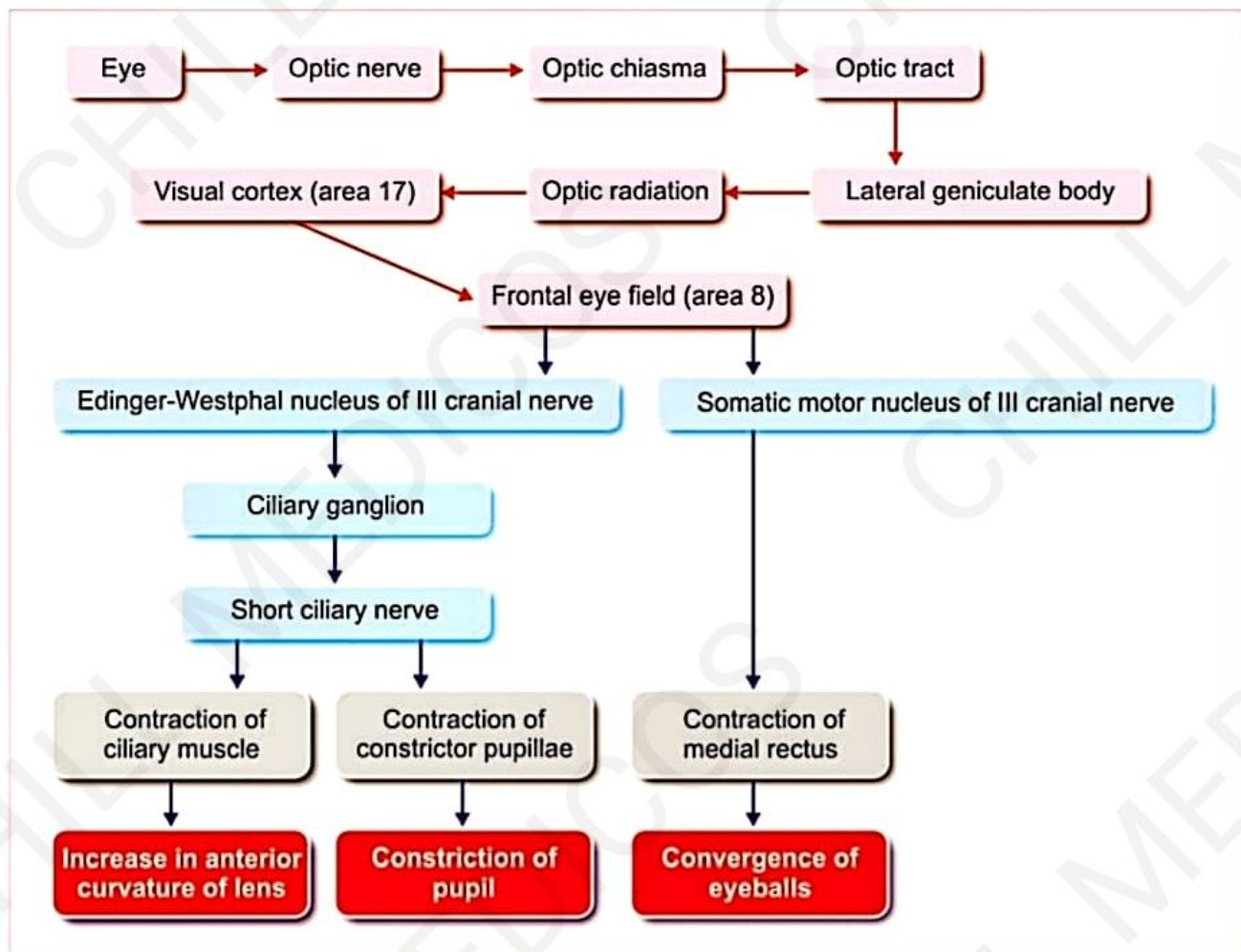


FIGURE 169.4: Pathway for accommodation reflex

★ AFFERENT PATHWAY →

- VISUAL IMPULSES FROM RETINA PASS THROUGH OPTIC NERVE, OPTIC CHIASMA, OPTIC TRACT, LATERAL GENICULATE BODY & OPTIC RADIATION TO VISUAL CORTEX (area 17) OF OCCIPITAL LOBE.
- ASSOCIATION FIBERS CARRY IMPULSES TO FRONTAL LOBE.

★ CENTER → • LIES IN FRONTAL EYE FIELD (area 8) THAT IS SITUATED IN FRONTAL LOBE OF CEREBRAL CORTEX.

★ EFFERENT PATHWAY →

1) EFFERENT FIBERS TO CILIARY MUSCLE / SPHINCTER PUPILLAE →

FROM AREA 8, CORTICONUCLEAR FIBERS PASS VIA INTERNAL CAPSULE



TO EDINGER - WESTPHAL NUCLEUS OF 3RD CRANIAL NERVE.



PREGANGLIONIC FIBERS PASS THROUGH 3RD CRANIAL NERVE TO CILIARY GANGLION.



POSTGANGLIONIC FIBERS FROM CILIARY GANGLION PASS VIA SHORT CILIARY NERVES & SUPPLY CILIARY MUSCLE & CONSTRUCTOR PUPILLAE.

2) EFFERENT FIBERS TO MEDIAL RECTUS →

↳ FIBERS FROM FRONTAL EYE FIELD TERMINATE IN SOMATIC MOTOR NUCLEUS OF OCCULOMOTOR NERVE.



FIBERS FROM MOTOR NUCLEUS SUPPLY MEDIAL RECTUS.

ERRORS OF REFRACTION

- ★ 4 Types → 1) AMETROPIA → MYOPIA / SLIGHT OR SHORT SIGHTENESS
HYPERMETROPIA / LONG SIGHTENESS
- 2) ANISOMETROPIA
3) ASTIGMATISM
4) PRESBYOPIA

★ AMETROPIA →

- EYE WITH NORMAL REFRACTIVE POWER CALLED EMMETROPIC EYE

↓
CONDITION CALLED EMMETROPIA

- ANY DEVIATION IN THE REFRACTIVE POWER FROM NORMAL CONDITION.

↓
CALLED AMETROPIA & EYE CALLED AMETROPIC EYE.

- ↓
- 1) MYOPIA 2) HYPERMETROPIA

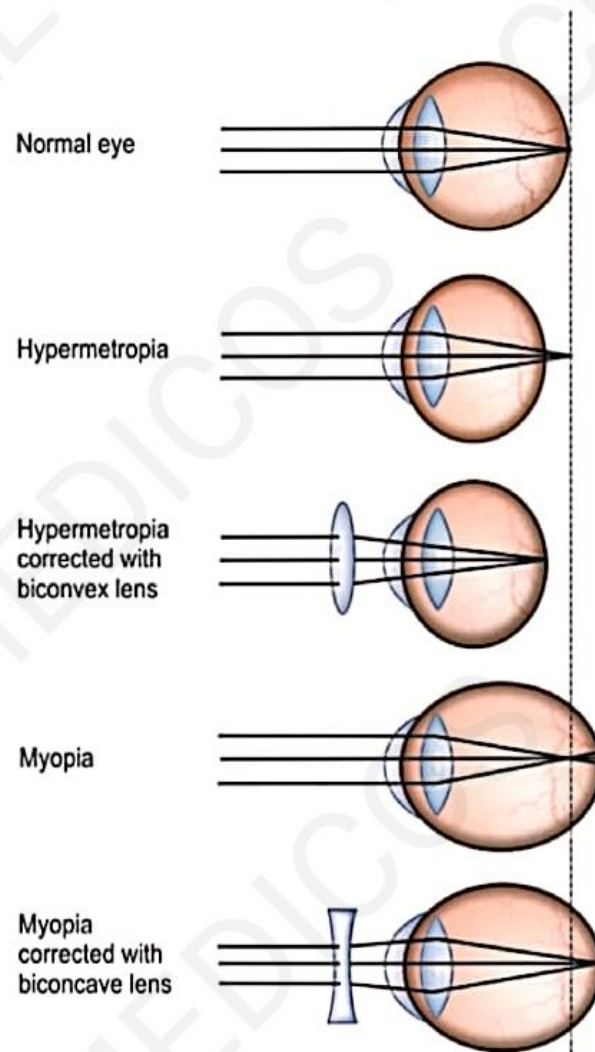


FIGURE 171.1: Errors of refraction

1. MYOPIA OR SHORT SIGHTEDNESS →

↓
EYE DEFECT CHARACTERIZED BY INABILITY TO SEE THE DISTANT OBJECT.

↓
CALLED SHORT SIGHTEDNESS BECOZ PERSON CAN SEE NEAR OBJECTS CLEARLY
BUT NOT THE DISTANT OBJECTS.

• NEAR VISION → NORMAL

FAR POINT → NOT INFINITE [at a DEFINITIVE DISTANCE].

CAUSE → 1) REFRACTIVE POWER OF LENS IS NORMAL
2) BUT ANTEROPOSTERIOR DIAMETER OF EYEBALL IS ABNORMALLY LONG.
↓
IMAGE IS TO FOCUS A LITTLE IN FRONT OF RETINA.
3) BLURRED IMAGE FORMED UPON RETINA.

CORRECTION → • THE LIGHT RAY ENTERING THE EYE MUST BE DIVERGENT
↓
NOT PARALLEL.
USING A BICONCAVE LENS.

2. HYPERMETROPIA OR LONG SIGHTEDNESS →

↓
• THE EYE DEFECT CHARACTERIZED BY INABILITY TO SEE NEAR OBJECT.

• CALLED LONG SIGHTEDNESS BECAUSE

↓
THE PERSON CAN SEE DISTANT OBJECTS CLEARLY BUT NOT NEAR OBJECTS.

• ALSO CALLED HYPEROPIA.

• DISTANT VISION → NORMAL

NEAR VISION → AFFECTED.

* CAUSE → 1) DUE TO ↓SED ANTEROPOSTERIOR DIAMETER OF EYEBALL.
2) THE REFRACTIVE POWER OF LENS IS NORMAL.

↓
LIGHT RAYS ARE NOT CONVERGED ENOUGH TO FORM
A CLEAR IMAGE ON RETINA.

↓
LIGHT RAYS ARE TO FOCUS BEHIND RETINA.

3) CAUSES BLURRED IMAGE OF NEAR OBJECTS.

* CORRECTION → * USING BICONVEX LENS

↓
LIGHT RAYS CONVERGED BEFORE ENTERING EYE.

★ ANISOMETROPIA →

↓
THE CONDITION IN WHICH 2 EYES HAVE UNEQUAL REFRACTIVE POWER.
CORRECTED BY → SEPARATE LENS (BICONCAVE & BICONVEX) FOR EACH EYE.

★ ASTIGMATISM →

★ THE CONDITION IN WHICH LIGHT RAYS ARE NOT BROUGHT TO A SHARP POINT UPON RETINA.

° WHEN IT IS MODERATE, CALLED PHYSIOLOGICAL ASTIGMATISM.

★ CAUSE → ★ IF CURVATURE IS DIFFERENT IN DIFFERENT MERIDIANS

↓
VERTICAL, HORIZONTAL & OBLIQUE

↓
REFRACTIVE POWER IS DIFFERENT IN DIFFERENT MERIDIANS.

↓
IRREGULARITY OF CURVATURE OF LENS CAUSES ASTIGMATISM.

★ TYPES → 2 TYPES

↓
1) REGULAR ASTIGMATISM

• REFRACTIVE POWER IS UNEQUAL IN DIFFERENT MERIDIANS BECAUSE

↓
ALTERATION OF CURVATURE IN ONE MERIDIAN.

• UNIFORM IN ALL POINTS THROUGHOUT AFFECTED MERIDIAN.

↓
2) IRREGULAR ASTIGMATISM

• THE REFRACTIVE POWER IS UNEQUAL NOT ONLY IN DIFFERENT MERIDIANS

↓
BUT ALSO UNEQUAL IN DIFFERENT POINTS OF SAME MERIDIAN.

★ CORRECTION → USING CYLINDRICAL GLASS LENS HAVING THE CONVEXITY IN MERIDIANS, CORRESPONDING TO THAT OF LENS OF EYE HAVING A LESSER CURVATURE.

★ PRESBYOPIA →

↓
CONDITION CHARACTERIZED BY PROGRESSIVE DIMINISHED ABILITY OF EYES TO FOCUS ON NEAR OBJECTS WITH AGE.

↓
DUE TO GRADUAL REDUCTION IN AMPLITUDE OF ACCOMODATION.

★ DISTANT VISION → UNAFFECTED
NEAR VISION → AFFECTED.

• THE ANTERIOR CURVATURE OF LENS DOES NOT INCREASE DURING NEAR VISION.

↓
SO, LIGHT RAYS FROM NEAR OBJECTS NOT BROUGHT TO FOCUS ON RETINA.

* CAUSE → 1) ↓sed ELASTICITY OF LENS BEC^g OF

↓
PHYSICAL CHANGES IN LENS & ITS CAPSULE DURING OLD AGE.

↓
SO, ANTERIOR CURVATURE NOT INCREASED DURING NEAR VISION.

2) ↓sed CONVERGENCE OF EYEBALLS

↓
DUE TO CONCOMITANT WEAKNESS OF OCULAR MUSCLE IN OLD AGE.

* CORRECTION → USING BICONVEX LENS.

TABLE 171.1: Errors of refraction

Type of error	Cause	Correction
Myopia	Increase in anteroposterior diameter of the eyeball	Biconcave lens
Hypermetropia	Decrease in anteroposterior diameter of the eyeball	Biconvex lens
Anisometropia	Difference in refractive power of both eyes	Separate lens (biconcave or biconvex) for each eye as required
Astigmatism	Refractory power of lens is different in different meridians	Cylindrical lens
Regular astigmatism	Refractory power of lens is unequal in different meridians but uniform in one single meridian	
Irregular astigmatism	Refractory power of lens is unequal in different meridians as well as in different points in same meridian	
Presbyopia	Loss of elasticity in lens and weakness of ocular muscles due to old age	Biconvex lens

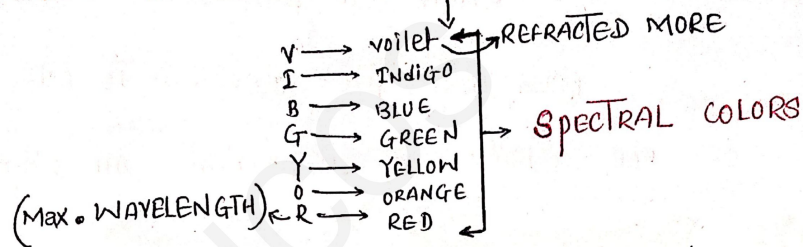
COLOR VISION

★ INTRODUCTION →

- DISCRIMINATION AND APPRECIATION OF COLORS DEPEND UPON ABILITY OF RECEPTORS IN RETINA.
- HUMAN EYE RECOGNIZE ABOUT 150 DIFFERENT COLORS

★ VISIBLE SPECTRUM →

- SERIES OF COLORED LIGHT PRODUCED BY PRISM



- ★ LIGHT RAYS LONGER THAN RED CALLED INFRARED RAYS.
- SHORTER THAN VIOLET CALLED ULTRAVIOLET RAYS.

PURKINJE PHENOMENON →

SHIFT OF BRIGHTEST PART OF SPECTRUM, WHEN INTENSITY OF ILLUMINATION IS CHANGED.

↓
DUE TO THE MAXIMAL STIMULATION OF CONES BY YELLOW & MAXIMAL STIMULATION OF RODS BY GREEN.

↳ IF COLORS AT HIGH ILLUMINATION → BRIGHTEST PART IS YELLOW.

↓
SHIFTED TO LEFT.

IF LIGHT INTENSITY IS ↓ USED / REDUCED → COLOR OF SPECTRUM IS FADES
BRIGHTEST PART IS GREEN.

↓
SHIFTED TO RIGHT.

PRIMARY COLORS →

↳ WHICH WHEN COMBINED TOGETHER PRODUCE WHITE

↳ THIS ARE RED, GREEN & BLUE (IN EQUAL PROPORTION GIVE WHITE).

COMPLEMENTARY COLORS →

↳ THE PAIR OF 2 COLORS, WHICH PRODUCE WHITE [MIXED / COMBINED IN PROPER PROPORTION]

↳ EX. - RED & GREENISH BLUE
ORANGE AND CYAN BLUE
YELLOW & INDIGO BLUE
VIOLET & GREENISH YELLOW
PURPLE & GREEN.

★ THEORIES OF COLOR VISION →

↓
To explain mechanism of perception of color by eyes.

1. THOMAS YOUNG TRICHROMATIC THEORY →

↓
According to, retina has 3 types of cones.

↓
Each one possesses its own photosensitive substance

↓
Each cone gives response to one of 1° color (Red, Green, blue).

↓
For sensation of white light, all 3 types stimulated equally.

2. HELMHOLTZ TRICHROMATIC THEORY →

↓
→ Substituted the sensitive filaments of optic nerve for cones.

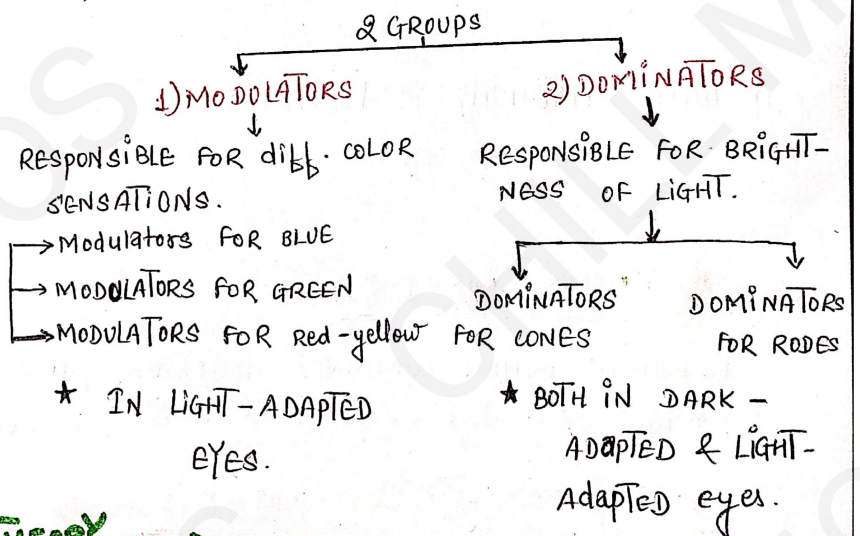
↓
Give response selectively to one / other of 3 primary colors.

→ Also called YOUNG - HELMHOLTZ THEORY.

3. GRANIT DOMINATOR - MODULATOR THEORY →

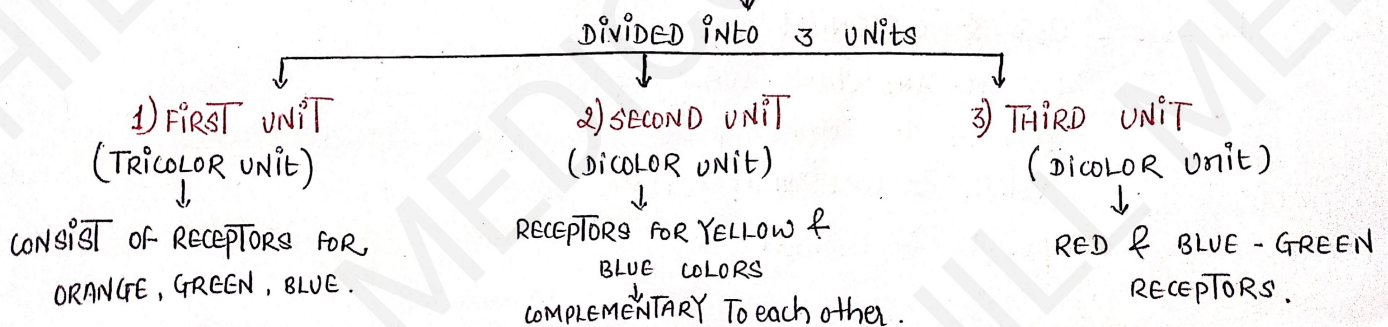
↓
→ The ganglionic cells of retina are stimulated by whole of visual spectrum.

→ On basis of sensitivity curves, Granit classified ganglionic cells into



4. HARTRIDGE POLYCHROMATIC THEORY →

↓
According to this, human retina has seven types of receptors.



5. HERING'S THEORY OF OPPOSITE COLORS →

RETINA HAS 3 PHOTOCHEMICAL SUBSTANCES.

EACH SUBSTANCE PRODUCES THE SENSATION OF A PARTICULAR COLOR BY ITS BREAKDOWN OR RESYNTHESIS.

- | 1) FIRST SUBSTANCE
(WHITE - BLACK SUBSTANCE) | 2) SECOND SUBSTANCE
(YELLOW-BLUE SUBSTANCE) | 3) THIRD SUBSTANCE
(RED - GREEN SUBSTANCE) |
|---|--|---|
| 1) BREAKDOWN CAUSES SENSATION OF WHITE. | 1) BREAKDOWN CAUSES SENSATION OF YELLOW. | 1) BREAKDOWN CAUSES SENSATION OF RED. |
| 2) RESYNTHESIS CAUSES SENSATION OF BLACK. | 2) RESYNTHESIS CAUSES SENSATION OF GREEN-BLUE. | 2) RESYNTHESIS CAUSES SENSATION OF GREEN. |

★ COLOR SENSITIVE AREAS IN RETINA →

→ PERIPHERAL PART OF RETINA IS DEVOID OF CONES

↓
INSENSITIVE TO COLOR & GIVES SENSATION OF WHITE, BLACK & GREY ONLY.

→ CENTRAL PORTION OF RETINA, FOVEA CENTRALIS

↓
HAS MORE CONES (MORE SENSITIVE TO COLOR)

→ RETINAL AREA SENSITIVE TO BLUE IS LARGEST & GREEN IS SMALLEST.

★ CONTRAST EFFECTS →

1) SIMULTANEOUS CONTRAST
[THE EFFECT THAT INTENSIFIES THE CONTRAST (DIFFERENCE) b/w 2 COLORS]
↓
PLACED AGAINST EACH OTHER.

2) SUCCESSIVE CONTRAST
[THE EFFECT OF PREVIOUSLY VIEWED COLOR FIELD ON APPEARANCE OF CURRENTLY VIEWED COLOR FIELD.]
↓
THERE IS AN INC IN SENSITIVITY TO THE COMPLEMENTARY COLOR.

★ AFTERIMAGE →

★ THE PHENOMENON IN WHICH RETENTION OF IMAGE OCCURS EVEN AFTER CESSATION OF LIGHT STIMULUS.

1. +VE AFTER IMAGE → AN AFTERIMAGE PERSISTING AFTER CLOSURE OF EYES OR TURNING TOWARDS A DARK BACKGROUND
2. -VE AFTER IMAGE → AN AFTERIMAGE THAT PERSISTS WHILE TURNING TOWARDS A BRIGHT BACKGROUND.

COLOR BLINDNESS

- ↳ THE FAILURE TO APPRECIATE ONE / MORE COLORS.
- ↳ COMMON IN 8% MALES & 0.4% OF FEMALES [COLOR BLINDNESS IS AN INHERITED SEX-LINKED RECESSIVE CHARACTER].
- ↳ 'COLOR BLIND' DOES NOT MEAN THAT OBJECTS ARE SEEN ONLY IN BLACK & WHITE.
- ↳ MOST APPROPRIATE TERM FOR COLOR BLINDNESS - DEFICIENCY OF COLOR VISION.

★ CAUSES →

- 1. TRAUMA** → 1) INJURY TO EYE (DUE TO ACCIDENTS OR STROKES).
- 2. CHRONIC BLINDNESS DISEASES** →
 - 1) GLAUCOMA
 - 2) DEGENERATION OF MACULA OF EYE
 - 3) RETINITIS
 - 4) SICKLE CELL ANEMIA
 - 5) LEUKEMIA, DIABETES
 - 6) PARKINSON DISEASE
 - 7) ALZHEIMER DISEASE
 - 8) MULTIPLE SCLEROSIS
- 3. DRUGS** →
 - 1) ANTIBIOTICS
 - 2) ANTIHYPERTENSIVE DRUGS
 - 3) ANTITUBERCULOSIS DRUGS
 - 4) BARBITURATES
- 4. TOXINS** →
 - 1) FERTILIZERS
 - 2) CARBON MONOXIDE
 - 3) CARBON DISULFIDE
 - 4) CHEMICALS WITH HIGH LEAD CONTENT.
- 5. ALCOHOLISM** → 1) CHRONIC ALCOHOLISM
- 6. AGING** → AFTER 60 YRS OF AGE

★ CLASSIFICATION →

BASED ON YOUNG - HELMHOLTZ TRICHROMATIC THEORY, CLASSIFIED INTO 3 TYPES →

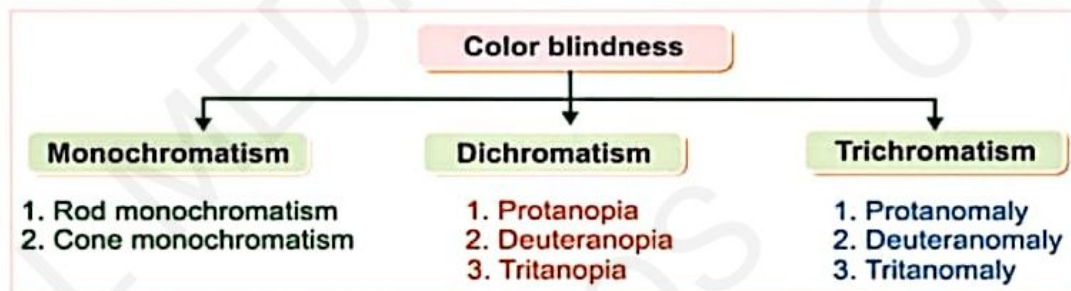


FIGURE 170.1: Color blindness

1. MONOCHROMATISM →

- ↳ CONDITION CHARACTERIZED BY TOTAL INABILITY TO PERCEIVE COLOR.
- ↳ ALSO CALLED TOTAL COLOR BLINDNESS / ACHROMATOPSIA.
- ↳ PERSON WITH MONOCHROMATISM → MONOCHROMATS.

↳ RETINA OF MONOCHROMATS IS TOTALLY INSENSITIVE TO COLOR.

↓
THEIR VISION IS SIMILAR TO BLACK & WHITE PHOTOGRAPHY.

1) ROD MONOCHROMATISM

↳ CONDITION IN WHICH CONES ARE FUNCTIONLESS & VISION DEPENDS ON RODS.

↳ TOTALLY COLOR BLIND & ARE NOT BLIND DURING DAYLIGHT.

↳ VISUAL ACUITY IS LOWERED & FOVEAL VISION IS ABSENT

↓
RESULTS IN CENTRAL SCOTOMA

FORMATION OF BIG BLIND SPOT IN FOVEA CENTRALIS DUE TO NON-FUNCTIONING OF CONES

2) CONE MONOCHROMATISM

↳ THE CONDITION IN WHICH VISION DEPENDS UPON ONE SINGLE TYPE OF CONE.

↳ CENTRAL SCOTOMA DOES NOT OCCUR.

2. DICHROMATISM →

↳ THE COLOR BLINDNESS IN WHICH SUBJECT CAN APPRECIATE ONLY 2 COLORS.

↳ PERSONS CALLED DICHROMATS.

↳ MATCH ENTIRE SPECTRUM OF COLORS BY ONLY 2 PRIMARY COLORS.

↓
RECEPTORS FOR 3RD COLOR DEFECTIVE.

3 GROUPS ↓

1) PROTANOPIA

CAUSED BY DEFECT IN RECEPTOR OF 1ST PRIMARY COLOR. (RED)

↳ PERSONS (PROTANOPES)

↳ USE GREEN & BLUE TO MATCH COLORS.

2) DEUTERANOPIA

CAUSED BY DEFECT IN RECEPTOR OF 2ND PRIMARY COLOR. (GREEN)

↳ PERSON (DEUTERANOPES)

↳ USE BLUE & RED COLORS.

3) TRITANOPIA

CAUSED BY DEFECT IN RECEPTOR OF 3RD PRIMARY COLOR (BLUE)

↳ PERSON (TRITANOPES)

↳ USE RED & GREEN COLORS

3. TRICHROMATISM →

↳ COLOR BLINDNESS IN WHICH INTENSITY OF ONE OF PRIMARY COLORS CAN NOT BE APPRECIATED CORRECTLY THROUGH AFFECTED PERSONS ARE ABLE TO PERCEIVE ALL 3 COLORS.

↳ PERSONS [TRICHROMATES].

3 Types

1) PROTANOMALY

↓
IN WHICH THE PERCEPTION FOR RED IS WEAK.

2) DEUTERANOMALY

↓
IN WHICH PERCEPTION FOR GREEN IS WEAK.

3) TRITANOMALY

↓
TRICHROMATISM WITH WEAK PERCEPTION OF BLUE.

★ TESTS FOR COLOR BLINDNESS →

- 3 METHODS →
- 1) USING Ishihara color charts
 - 2) USING Holmgren colored wool
 - 3) USING Edridge - GREEN LANTERN.

ORGAN OF CORTI

* THE RECEPTOR ORGAN FOR HEARING.

↓
IT IS **NEUROEPITHELIAL** STRUCTURE IN COCHLEA.

* SITUATION AND EXTENT →

↓
RESTS UPON THE LIP OF OSSEOUS SPIRAL LAMINA & BASILAR MEMB.

• EXTENDS THROUGHOUT THE COCHLEAR DUCT EXCEPT FOR A SHORT DISTANCE OF EITHER END.

* ROOF OF ORGAN OF CORTI IS FORMED BY GELATINOUS TECTORIAL MEMB.

STRUCTURE → • MADE UP OF SENSORY ELEMENTS CALLED **HAIR CELLS** & VARIOUS SUPPORTING CELLS.

↓
CELLS ARE ARRANGED IN ORDER FROM CENTER TOWARD PERIPHERY.

- | | |
|---------------------------|--|
| 1. BORDER CELLS | 6. OUTER PHALANGEAL CELLS |
| 2. INNER HAIR CELLS | 7. OUTER HAIR CELLS |
| 3. INNER PHALANGEAL CELLS | 8. CELLS OF HENSEN |
| 4. INNER PILLAR CELLS | 9. CELLS OF CLAUDIUS |
| 5. OUTER PILLAR CELLS | 10. TECTORIAL MEMBRANE
LAMINA RETICULAR |

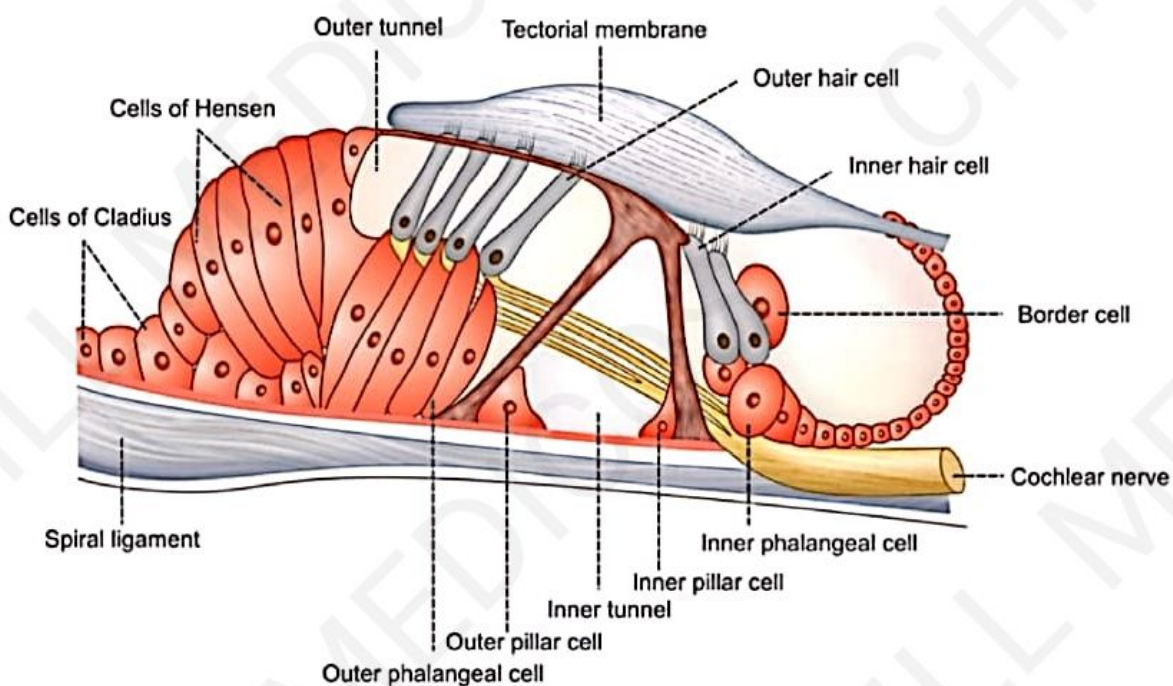


FIGURE 172.4: Organ of Corti

1. BORDER CELLS →

- ★ THE SLENDER COLUMNAR CELLS, ARRANGED IN A SINGLE LAYER ON THE TYMPANIC LIP ALONG THE INNER SIDE OF INNER HAIR CELLS.
- ↓
- SURFACES OF BORDER CELLS HAVE CUTICLE.

2. INNER HAIR CELLS →

- ★ ARE FLASK-SHAPED CELLS & ARE BROADER THAN OUTER HAIR CELLS.
- ★ THIS ARE ARRANGED IN SINGLE LAYER / ROW & OCCUPY ONLY UPPER PART OF EPITHELIAL LAYER.
- ★ ROUNDED BASE OF EACH CELL RESTS UPON ADJACENT SUPPORTING CELLS
- ↓
- CALLED INNER PHALANGEAL CELL.
- ★ SURFACE OF INNER HAIR CELL BEARS A CUTICULAR PLATE & STEREOCILIA.
- ★ LARGER STEREOCILIA CALLED KINOCILIUM.
- ★ INNER HAIR CELLS AND OUTER HAIR CELLS TOGETHER FORM RECEPTORS CELLS.
- ↓
- BOTH HAVE AFFERENT & EFFERENT NERVE FIBERS.

3. INNER PHALANGEAL CELLS →

- ↓
- ↳ SUPPORTING CELLS OF INNER HAIR CELLS & ARRANGED IN A ROW ALONG INNER SURFACE OF INNER PILLAR CELLS.
- ↳ BASES REST ON BASILAR MEMBRANE.
- ↳ CUTICULAR PLATE OF CELLS LOOK LIKE FINGER BONES, PHALANGES.

4.5. INNER & OUTER PILLAR CELLS → RODS OF CORTI.

- ↳ EACH PILLAR CELL HAS A BROADER BASE, AN ELONGATED BODY OR PILLAR AND A HEAD AT TIP OF PILLAR.
- ↳ BASES OF INNER PILLAR CELLS → CLOSE TO LIP OF OSSEOUS SPIRAL LAMINA (TYMPANIC LIP).
- ↳ BASES OF OUTER PILLAR CELLS → CLOSE TO BASILAR MEMBRANE.
- ↳ PILLARS OF CELLS FORM SERIES OF ARCHES
- ↓
- ENCLOSE A TRIANGULAR TUNNEL CALLED
- ↓

INNER TUNNEL / TUNNEL OF CORTI

6. OUTER PHALANGEAL CELLS →

- ↓
- ↳ OR THE CELLS OF DEITERS ARE SUPPORTING CELLS OF OUTER HAIR CELLS.
- ↳ THIS ARE TALL COLUMNAR CELL.
- ↳ SENDS PHALANGEAL PROCESSES UPWARD B/W HAIR CELLS, TO FORM PART OF LAMINA RETICULARIS.
- ↳ B/W INNER MOST OUTER PHALANGEAL CELLS & OUTER pillar cells
- ↓
- FLUID SPACE CALLED SPACE OF NUEL.

7. OUTER HAIR CELLS →

- ↓
- ↳ COLUMNAR CELLS OCCUPYING SUPERFICIAL PART OF EPITHELIUM OF ORGAN OF CORTI.
- ↳ BASES SUPPORTED BY OUTER PHALANGEAL CELLS.
- ↳ STRUCTURE SIMILAR TO INNER HAIR CELLS.

8. CELLS OF HENSEN →

- ↓
- ↳ TALL COLUMNAR CELLS FORMING OUTER BORDER CELLS OF ORGAN OF CORTI.
- ↳ CELLS ARRANGED IN SEVERAL ROWS ON BASILAR MEMBRANE.
- ↓
- LATERAL TO OUTER PHALANGEAL CELLS.
- ↳ SPACE B/W OUTER PHALANGEAL CELLS & CELLS OF HENSEN CALLED
- ↓
- OUTER TUNNEL.

9. CELLS OF CLAUDIUS →

- ↓
- ↳ CUBOIDAL IN NATURE & LINE LOWER SURFACE OF EXTERNAL SPIRAL SULCUS.
- ↳ GROUP OF CELLS PRESENT B/W CELLS OF CLAUDIUS & BASILAR MEMBRANE
- ↓
- called BOETTCHER CELLS.

10. TECTORIAL MEMBRANE/LAMINA RETICULARIS →

- ↓
- EXTENDS FROM VESTIBULAR LIP TO LEVEL OF CELLS OF HENSEN [FORMS ROOF OF ORGAN OF CORTI].
- ↳ PROCESSES OF HAIR CELLS STIMULATED BY MOVEMENTS OF TECTORIAL MEMBRANE.
- ↓
- VIBRATIONS IN ENDOLYMPH.
- ↳ CUTICULAR CELLS/PLATES OF ALL SUPPORTING CELLS FORM A RETICULAR MEMB.
- ↓
- called AS LAMINA RETICULARIS.

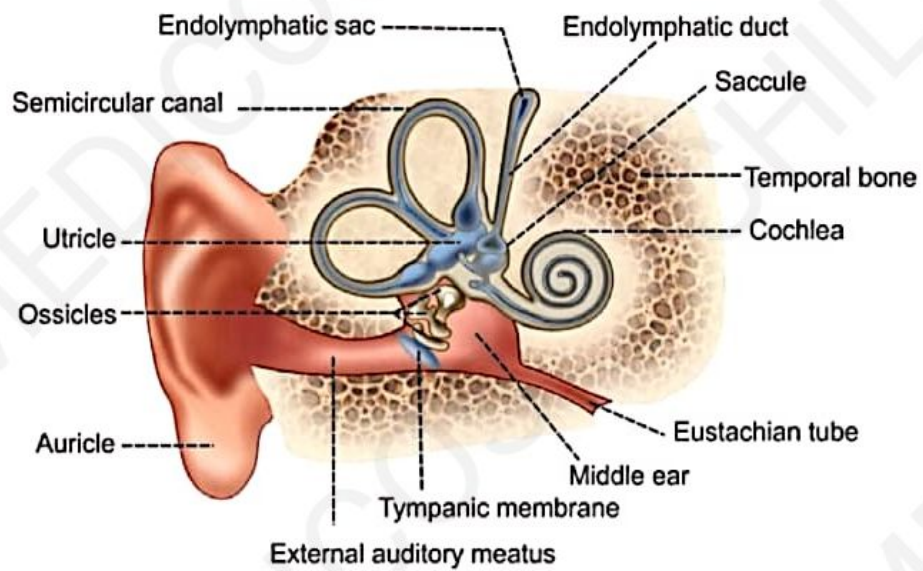
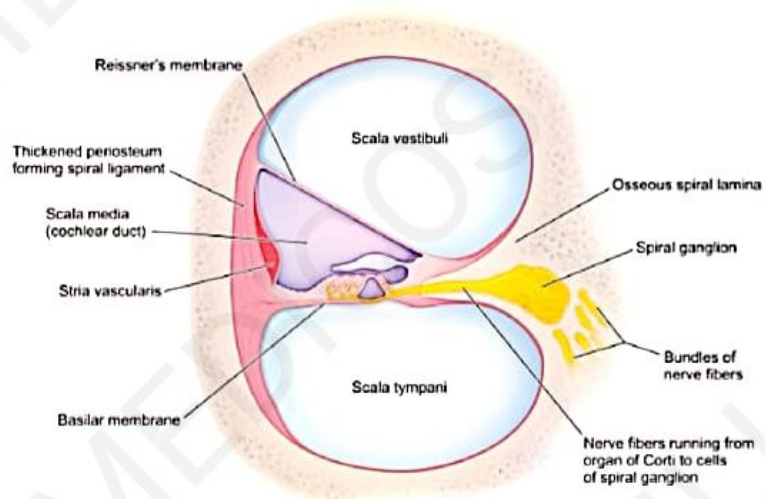
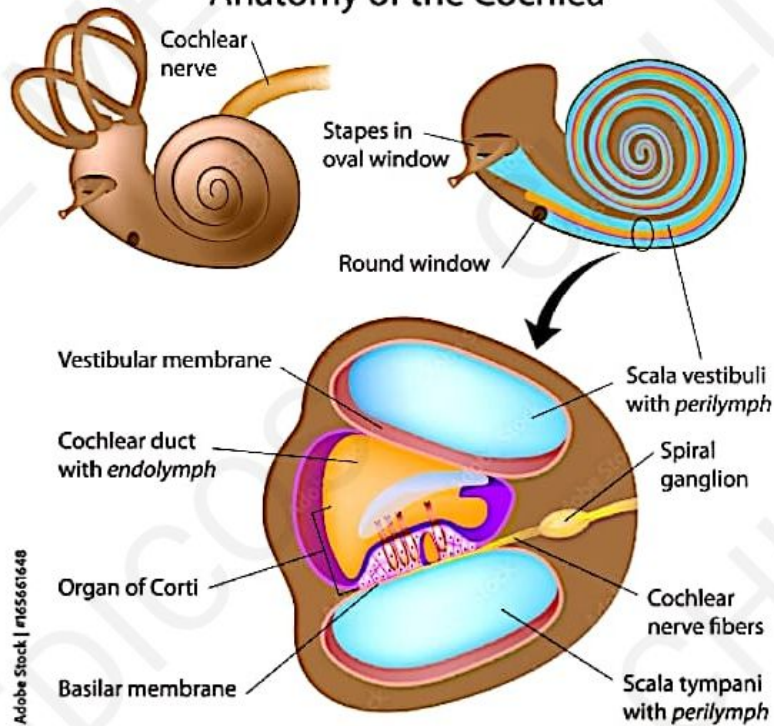


FIGURE 172.1: Diagram showing the structure of ear

Anatomy of the Cochlea



MECHANISM OF HEARING

★ INTRODUCTION →

- SOUND WAVES TRAVEL THROUGH EXTERNAL AUDITORY MEATUS AND PRODUCE VIBRATIONS IN TYMPANIC MEMBRANE → MIDDLE EAR (BONES) → RESULTS IN VIBRATIONS IN FLUIDS OF COCHLEA.



STIMULATE HAIR CELLS AND IN RETURN ACTION POTENTIAL IS GENERATED.

↳ AUDITORY IMPULSES (ACTION POTENTIAL) REACH THE CORTEX, & PERCEPTION OF SOUND OCCURS.

↳ SOUND WAVES → ACTION POTENTIAL [SOUND TRANSDUCTION].

★ ROLE OF EXTERNAL EAR →

↳ DIRECTS THE SOUND WAVES TOWARDS TYMPANIC MEMBRANE.

↳ WAX ACCUMULATION PREVENTS CONDUCTION OF SOUND.

★ ROLE OF MIDDLE EAR →

1) TYMPANIC MEMBRANE → DUE TO PRESSURE CHANGES PRODUCED BY SOUND WAVES, TYMPANIC MEMBRANE VIBRATES i.e. IT MOVES IN AND OUT OF MIDDLE EAR.

↳ TYMPANIC MEMBRANE ACTS AS A **RESONATOR**.

2) AUDITORY OSSICLES → VIBRATIONS SET UP IN TYMPANIC MEMBRANE ARE TRANSMITTED THROUGH THE MALLEUS → INCUS → STAPES AND CAUSE VIBRATIONS IN COCHLEA.

★ IMPEDANCE MATCHING → IT IS THE PROCESS BY WHICH TYMPANIC MEMBRANE AND AUDITORY OSSICLES CONVERT THE SOUND ENERGY INTO MECHANICAL VIBRATION WITH MINIMUM LOSS OF ENERGY BY MATCHING THE IMPEDANCE OFFERED BY FLUID.

↳ IMPEDANCE MATCHING IS THE MOST IMPORTANT FUNCTION OF MIDDLE EAR. WITHOUT IT, CONDUCTIVE DEAFNESS OCCURS.

★ TYPES OF CONDUCTION →

1) OSSICULAR CONDUCTION → via NORMAL ossicles.

2) AIR CONDUCTION → It occurs in MIDDLE EAR WHEN THERE is A DEFECT in EAR OSSICLES.

[Like OTOSCLEROSIS is DISEASE ASSOCIATED WITH FIXATION OF STAPES TO OVAL WINDOW].

3) BONE CONDUCTION → It is THE CONDUCTION OF SOUND WAVES THROUGH MIDDLE EAR BY BONES.

• WE HEAR OUR VOICES BY BONE CONDUCTION BY OUR SKULL.

3) ROLE OF EUSTACHIAN TUBE →

↳ It is NOT CONCERNED WITH HEARING BUT RESPONSIBLE FOR EQUALISING THE PRESSURE ON EITHER SIDE OF TYMPANIC MEMBRANE.

★ ROLE OF INNER EAR →

■ TRAVELING WAVE → MOVEMENT OF FOOTPLATE OF STAPES AGAINST OVAL WINDOW.
↓
CAUSES MOVEMENT OF PERILYMPH IN SCALA VESTIBULI.
↓
IMMEDIATELY HITS THE VESTIBULAR MEMB. NEAR OVAL WINDOW.
↓
CAUSES MOVEMENT OF FLUID IN SCALA MEDIA.
↓
CAUSES BULGING OF BASAL PORTION OF BASILAR MEMB.
↓
TOWARDS SCALA TYMPANI.
↓
INITIATES A WAVE, TRAVELS ALONG BM TOWARDS HELICOTREMA [like that of Arterial pulse wave]
↓
TRAVELLING WAVE.

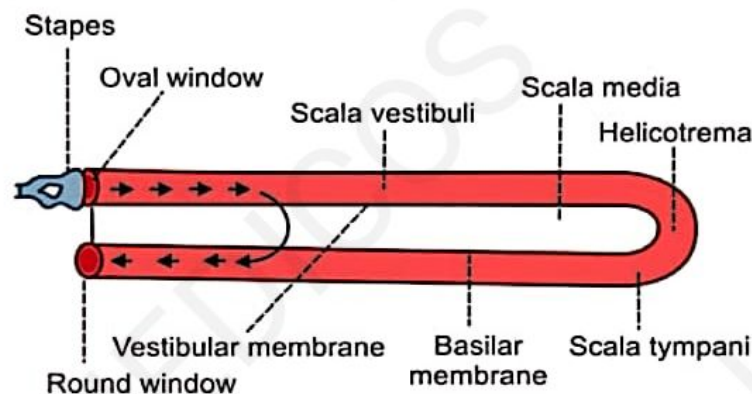


FIGURE 174.1: Diagrammatic representation of cochlea. Arrows show displacement of fluid

★ RESONANCE POINT →

THE PART OF BASILAR MEMBRANE, WHICH IS ACTIVATED BY TRAVELLING WAVE.

↳ THE WAVE AT ONE POINT (RESONANCE POINT) OF BASILAR MEMBRANE

BECOMES VERY STRONG & ACTIVATES THE BASILAR MEMBRANE.

RESONANCE POINT IMMEDIATELY VIBRATES BACK & FORTH.

TRAVELLING ^{POINT} WAVE stops.

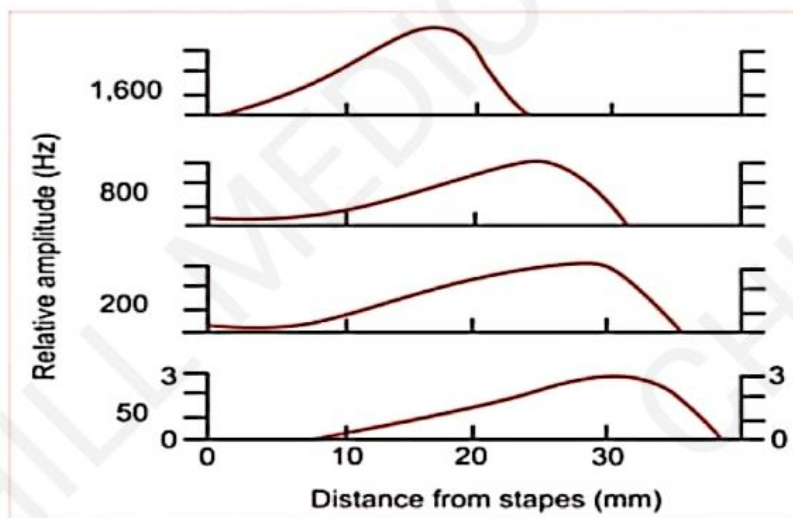


FIGURE 174.2: Traveling waves for different frequencies of sound

★ EXCITATION OF HAIR CELLS →

↳ STEREOCILIA OF HAIR CELLS IN ORGAN OF CORTI ARE EMBEDDED IN TECTORIAL MEMBRANE.

↳ HAIR CELLS ARE TIGHTLY FIXED BY CUTICULAR LAMINA RETICULARIS & PILLAR CELLS OR RODS OF CORTI.

↳ VIBRATION OF BASILAR MEMBRANE AT RESONANCE POINT CAUSES THE BASILAR FIBERS, RODS OF CORTI, HAIR CELLS & LAMINA RETICULARIS MOVE AS A SINGLE UNIT.

↳ MOVEMENT OF STEREOCILIA → GENERATION OF RECEPTOR A. POTENTIAL.

★ ELECTRICAL EVENTS →

• SOUND TRANSDUCTION IS A TYPE OF SENSORY TRANSDUCTION IN HAIR CELLS.

• 3 Types of ELECTRICAL EVENTS OCCURS :-

(a) RECEPTOR POTENTIAL (COCHLEAR MICROPHONIC POTENTIAL)

(b) ENDOCOCHLEAR POTENTIAL (ENDOLYMPHATIC POTENTIAL)

(c) ACTION POTENTIAL IN AUDITORY NERVE FIBERS.

* ROLE IN/OF HAIR CELLS →

1. INNER OF HAIR CELLS → THEY ARE RESPONSIBLE FOR SOUND TRANSDUCTION i.e. THESE RECEPTOR CELLS ARE PRIMARY SENSORY CELLS, WHICH CAUSE GENERATION OF AP IN AUDITORY NERVE FIBERS.

2. OUTER HAIR CELLS → THEY ARE SHORTENED DURING DEPOLARISATION & ELONGATED DURING HYPERPOLARISATION. THE PROCESS IS CALLED ELECTROMOTILITY OR MECHANOELECTRICAL TRANSDUCTION.

• OUTER HAIR CELLS ARE COCHLEAR AMPLIFIERS AS THEY ↑SE THE AMPLITUDE & SHARPNESS OF SOUND.

* PROPERTIES OF SOUND →

1) PITCH

2) LOUDNESS / INTENSITY

AUDITORY DEFECTS AND TESTS

AUDITORY DEFECTS ARE 2 Types.

- 1) CONDUCTION DEAFNESS
- 2) NERVE DEAFNESS

CONDUCTION DEAFNESS → Type of DEAFNESS THAT OCCURS DUE TO IMPAIRMENT IN TRANSMISSION OF SOUND WAVES IN THE EXTERNAL EAR / MIDDLE EAR.

CAUSES →

- 1) OBSTRUCTION OF EXTERNAL AUDITORY MEATUS WITH DRY WAX OR FOREIGN BODIES.
- 2) THICKENING OF TYMPANIC MEMBRANE DUE TO REPEATED MIDDLE EAR INFECTION.
- 3) PERFORATION OF TYMPANIC MEMBRANE (INEQUALITY OF PRESSURE ON EITHER SIDE)
- 4) OTITIS MEDIA (INFLAMMATION OF MIDDLE EAR)
- 5) OTOSCLEROSIS DUE TO ANKYLOSIS
↓
ABNORMAL IMMOBILITY & CONSOLIDATION OF A JOINT.

NERVE DEAFNESS → ★ THE DEAFNESS CAUSED BY DAMAGE OF ANY STRUCTURE IN COCHLEA, SUCH AS HAIR CELL, ORGAN OF CORTI, BASILAR MEMBRANE OR COCHLEAR DUCT OR THE LESION IN THE AUDITORY PATHWAY.

CAUSES →

- 1) DEGENERATION OF HAIR CELLS [DUE TO ANTIBIOTICS]
 ↳ STREPTOMYCIN
 ↳ GENTAMICIN
- 2) TUMOR AFFECTING VIII CRANIAL NERVE
- 3) DAMAGE OF COCHLEA BY PROLONGED EXPOSURE TO LOUD NOISE.

★ TESTS FOR HEARING →

- 1) BED SIDE TESTS :-
 - 1) WHISPERING TEST
 - 2) TICKLING OF WATCH TEST.
- 2) ROUTINE TESTS :-
 - 1) RINNE TEST
 - 2) WEBER TEST
 - 3) AUDIOMETRY

1. WHISPERING Test → IF SUBJECT IS NOT ABLE TO HEAR THE WHISPER, THEN HEARING DEFICIT IS SUSPECTED.

2. TICKLING OF WATCH Test → THE SUBJECT SUFFERING FROM HEARING DEFECTS CANNOT HEAR TICKLING SOUND OF WATCH.

ROUTINE TESTS →

1. RINNE Test → • BASE OF A VIBRATING TUNING FORK PLACED ON MASTOID PROCESS
↓
UNTIL SUBJECT CANNOT FEEL VIBRATION & CANNOT HEAR THE SOUND.

↳ AIR CONDUCTION VIA OSSICLES IS BETTER THAN BONE CONDUCTION.

↳ IN CONDUCTION DEAFNESS [VIBRATIONS IN AIR ARE NOT HEARD AFTER CESSATION OF BONE CONDUCTION].

↓
BONE CONDUCTION BETTER THAN AIR CONDUCTION.

↳ NERVE DEAFNESS [BOTH AIR CONDUCTION + BONE CONDUCTION ARE LOST].

2. WEBER Test → • BASE OF VIBRATING TUNING FORK PLACED ON VERTEX OF SKULL / MIDDLE OF FOREHEAD.

↳ IN UNILATERAL CONDUCTION DEAFNESS [deafness in ONE EAR]

↓
SOUND IS HEARD LOUDER IN DISEASED EAR.

↓
DUE TO ABSENCE OF MASKING EFFECT OF ENVIRONMENT NOISE.

↳ UNILATERAL NERVE DEAFNESS [SOUND HEARD LOUDER IN NORMAL EAR].

3. AUDIOMETRY → • TECHNIQUE USED TO DETERMINE NATURE & SEVERITY OF AUDITORY DEFECT.

• AUDIOMETER (INSTRUMENT) USED.

↓
AN ELECTRONIC FUNCTION GENERATOR OR OSCILLATOR [CONNECTED TO AN EAR PHONE].

↓
INSTRUMENT IS CAPABLE OF GENERATING SOUND WAVES OF DIFFERENT FREQUENCIES FROM LOWEST TO HIGHEST.

* AUDIOMETER HAS AN ELECTRONIC VIBRATOR ALSO.

↓
TO TEST BONE CONDUCTION FROM MASTOID PROCESS

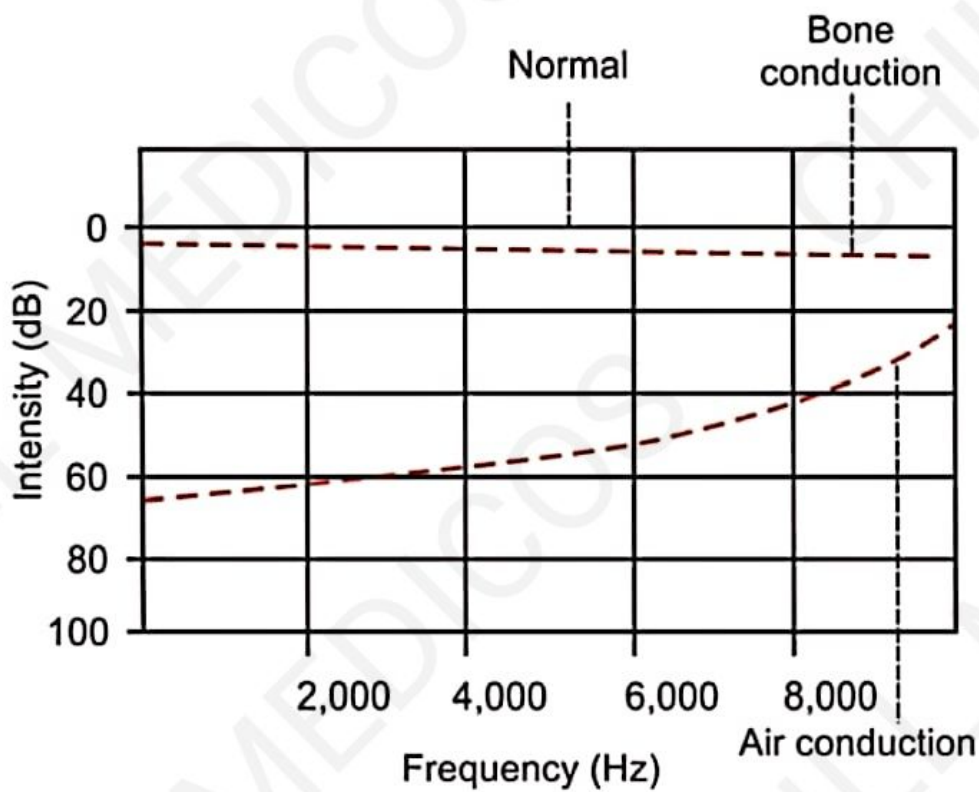
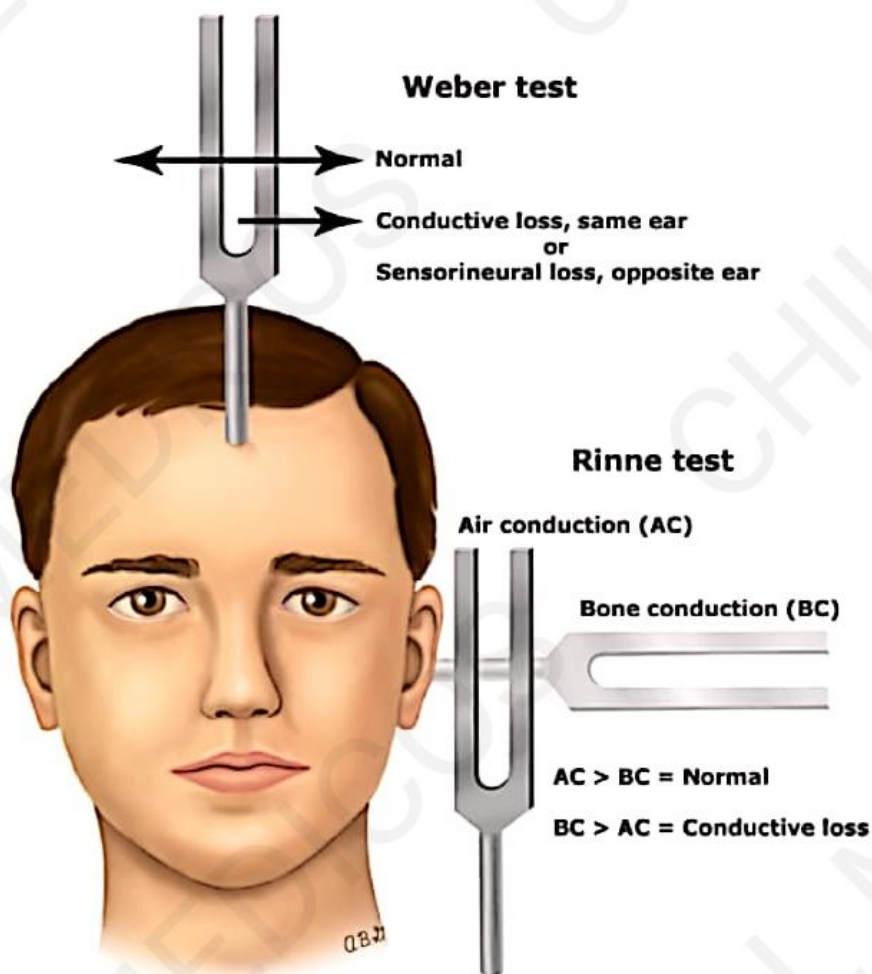


FIGURE 175.1: Audiogram in a patient with conductive deafness



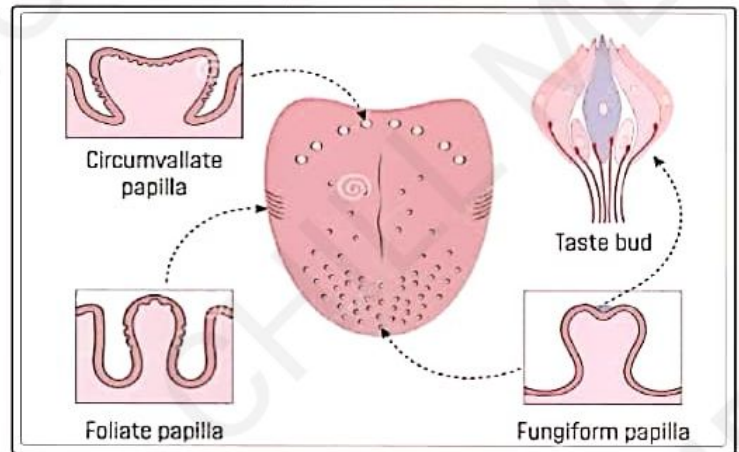
... TASTE SENSATION ...

TASTE BUDS →

- SENSE ORGANS FOR TASTE OR GUSTATORY SENSATION ARE TASTE BUDS.
- TASTE BUDS ARE OVOID BODIES.
- DIAMETER → 30-70 μ .
- NUMBER → 10,000 TASTE BUDS
- TASTE BUDS ARE PRESENT ON PAPILLAE OF TONGUE & ALSO IN MUCOSA OF EPIGLOTTIS, PALATE, PHARYNX AND PROXIMAL OESOPHAGUS.

• TYPES OF PAPILLAE :-

- (a) FILIFORM — SMALL, CONICAL
- (b) FUNGIFORM — ROUND, NEAR TIP
- (c) CIRCUMVALLATE — LARGE, POST. of tongue



★ STRUCTURE OF TASTE BUD :-

- IT IS A BUNDLE OF TASTE RECEPTOR CELLS, WITH SUPPORTING CELLS EMBEDDED.
- EACH TASTE BUDS — 40 CELLS.

TYPES →

- 1) Type I cells / SUBTENTACULAR CELLS
- 2) Type II CELLS
- 3) Type III CELLS
- 4) Type IV CELLS / BASAL CELLS.

- Type-II → TASTE RECEPTOR cell
- Type I & IV → SUPPORTING cells
- Type I, II & III → HAVE MICROVILLI

THERE IS A OPENING — TASTE — PORE

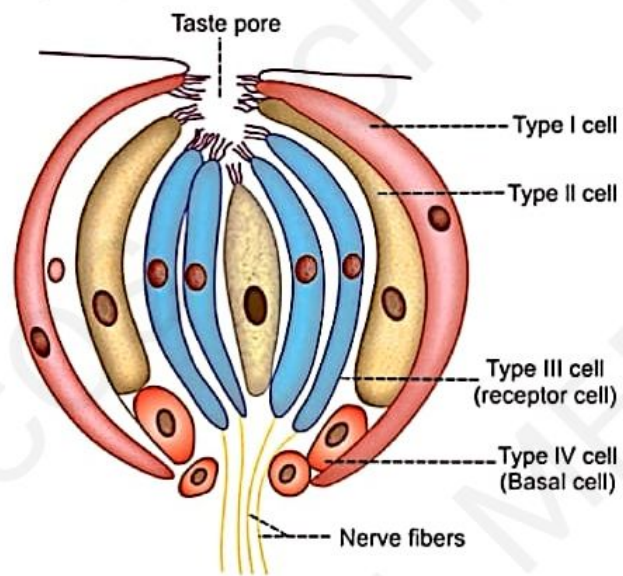


FIGURE 176.1: Taste bud

★ PATHWAY FOR TASTE SENSATION →

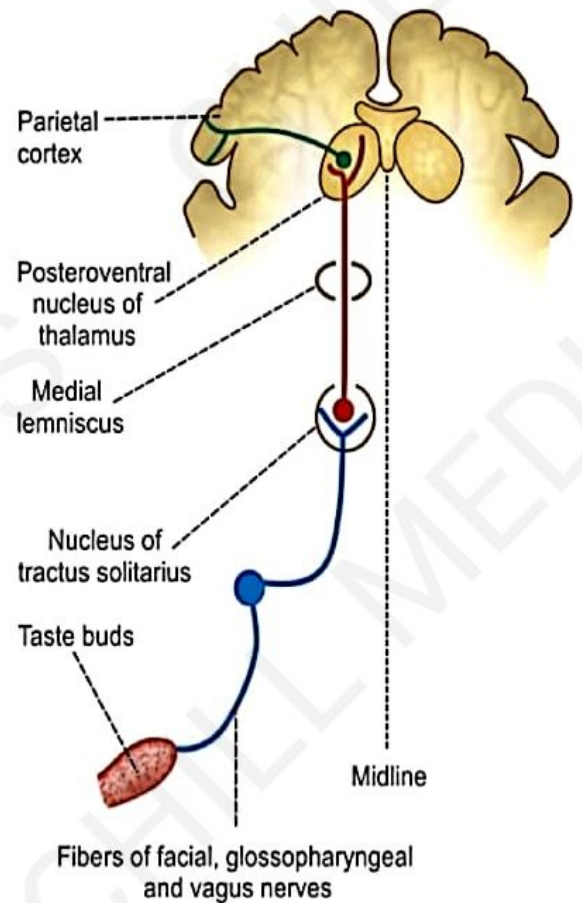
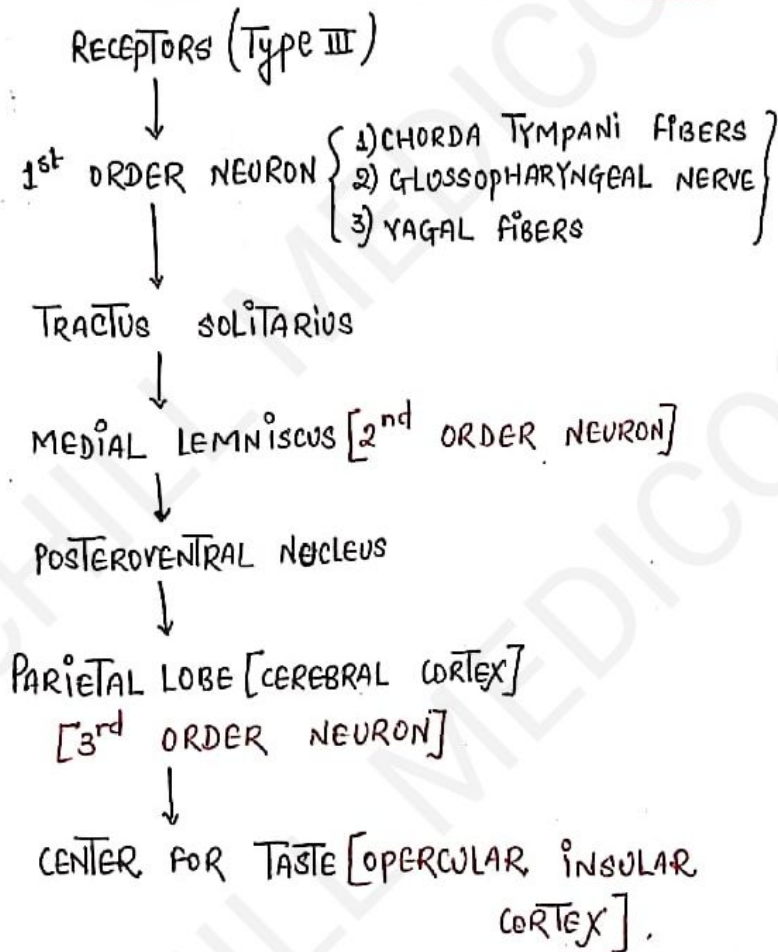


FIGURE 176.2: Pathway for taste sensation

★ TASTE SENSATIONS →

- 1) 5 Types →
- (a) SWEET
 - (b) SALT
 - (c) SOUR
 - (d) BITTER
 - (e) UMAMI

100 different tastes can be produced by combining those 5 primary tastes.

★ SWEET → PRODUCED MAINLY BY ORGANIC SUBSTANCE LIKE MONO/POLYSACCHARIDES, GLYCEROL, ALCOHOL, KETONES.

↳ INORGANIC SUBSTANCES - LEAD & BERYLLIUM ALSO GIVE SWEET TASTE.

↳ RECEPTOR FOR SWEET TASTE IS GPCR (G-protein coupled receptor).

↳ Sweet-Substance → CAUSE DEPOLARISATION VIA CYCLIC AMP.

★ SALT TASTE → • IT IS PRODUCED BY CHLORIDES, NITRATES, SULPHATES, BROMIDES & IODIDES.

↳ RECEPTOR - Epithelium sodium channel (ENaC)

↳ RECEPTOR RELEASE GLUTAMATE if Na^+ ENTERS AND CAUSES CAUSE DEPOLARISATION.

3. SOUR → By H^+ ions in ACID/ACID SALTS.

↳ RECEPTOR - ENaC

↳ H^+ CAUSES DEPOLARISATION

↳ BESIDES ENaC, HCN (Hyperpolarisation - Activated cyclic is ALSO INVOLVED → NUCLEOTIDE - GATED cation CHANNEL)

4. BITTER → ORGANIC SUBSTANCES LIKE QUININE, STRYCHNINE, MORPHINE, PICRIC ACID, BILE SALTS.

↳ CATIONS CAUSES BITTER TASTE.

↳ RECEPTOR - GPCR

↳ SUBSTANCE ACTIVATES PHOSPHOLIPASE C through G-Protein. WHICH CAUSES PRODUCTION OF Inositol Triphosphate (IP_3).

↓
DEPOLARIZATION BY RELEASING Ca^{2+} .

5. UMAMI → RECENTLY RECOGNISED, TASTE PRODUCED WHEN TASTE BUDS RESPOND TO GLUTAMATE PARTICULARLY MONOSODIUM GLUTAMATE (MSG).

↳ RECEPTOR METABOTROPIC GLUTAMATE RECEPTOR (mGluR4).

↳ GLUTAMATE CAUSES DEPOLARISATION.

*** APPLIED** →

1) **AGEUSIA** → LOSS OF TASTE SENSATION

• LESION IN FACIAL N., CHORDA Tympani or Mandibular division of trigeminal nerve causes loss of TASTE SENSATION in ant. $\frac{2}{3}$ rd of Tongue.

• LESION IN GLOSSOPHARYNGEAL N. - loss in Post. $\frac{1}{3}$ rd.

• TEMPORARY LOSS - DRUGS like captopril & penicillamine.

2) **HYPOGEUSIA** → ↓ in TASTE SENSATION.

3) **DYSGEUSIA** → DISTURBANCE IN TASTE SENSATION [due to Temporal lobe Syndrome]

4) **TASTE BLINDNESS** → RARE GENETIC DISORDER IN WHICH ABILITY TO recognise substance by TASTE is lost.

SENSATION OF SMELL

OLFACTORY RECEPTORS →

- THEY ARE SITUATED IN OLFACTORY MUCOS MEMBRANE, WHICH IS MODIFIED MUCUS MEMBRANE THAT LINES UPPER PART OF NOSTRIL.
- THE MUCUS MEMBRANE CONSISTS OF 10-20 millions OF OLFACTORY RECEPTOR CELLS.
- MUCOSA ALSO HAVE BOWMAN GLANDS (MUCUS - SECRETING).
- OLFACTORY RECEPTOR CELL IS A BIPOLAR NEURON.

↓
10-12 CILIA ARISE (NON-MYELINATED)
↓
LENGTH (2μ)
DIAMETER (0.1μ)

★ OLFACTORY PATHWAY →

• AXONS OF BIPOLAR OLFACTORY RECEPTORS
↓ (Pierces cribriform plate)
OLFACTORY BULB
↓
AXONS SYNAPSE WITH DENDRITES
OF MITRAL CELLS.
↓
OLFACTORY GLOMERULI
↓
OLFACTORY CORTIX
↓
OLFACTORY STRIA.

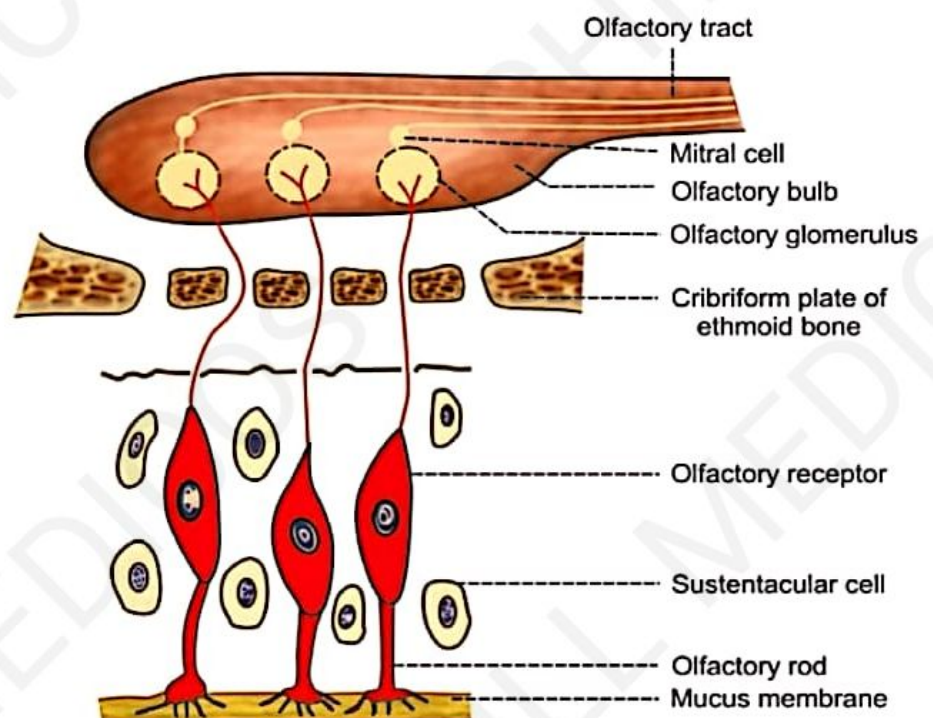
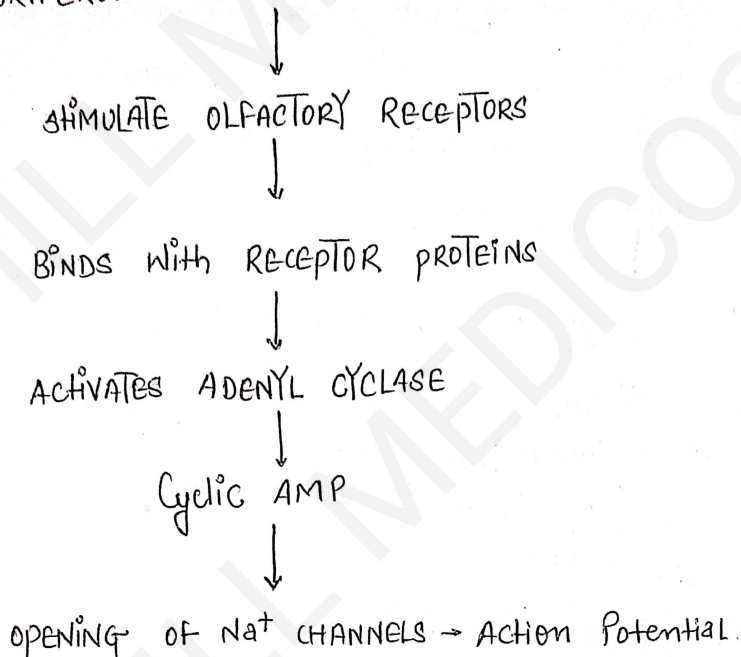


FIGURE 177.1: Olfactory mucus membrane and pathway for olfactory sensation

OLFACTORY TRANSDUCTION →

- It is the process by which olfactory receptor converts chemical energy into action potentials in olfactory nerve fibers.
- Odoriferous substance dissolve in mucus



CLASSIFICATION OF ODOR →

- Various types
- Some are →
 - (a) AROMATIC odor/RESINOUS odor — CAMPHOR, CLOVE.
 - (b) AMBROSIAL odor [MUSK]
 - (c) BURNING odor [TOBACCO, ROASTED COBEE]
 - (d) ETHEREAL odor [FRUITS, ETHERS]
 - (e) FRAGRANT odor/BALSAMIC o. — FLOWERS & PERFUMES
 - (f) NAUSEATING odor — DECAYED VEGETABLES & FECES
 - (g) GOAT odor — SWEET CHEESE
 - (h) REPULSIVE odor — BED BUG,
 - (i) GARLIC odor — GARLIC, ONION, SULFUR

ADAPTATION →

- Olfactory receptors are phasic receptors and adapt very rapidly.
- Within one second, adaptation occurs upto 50%.

Applied →

1) ANOSMIA → TOTAL LOSS OF SENSATION OF SMELL.

a) TEMPORARY ANOSMIA → is DUE TO OBSTRUCTION OF NOSE (AS IN COMMON COLD, NASAL SINUS).

b) PERMANENT ANOSMIA → is DUE TO LESION IN OLFACTORY TRACT, MENINGITIS AND DEGENERATIVE CONDITIONS SUCH AS - PARKINSON D. & ALZHEIMER DISEASE.

2) HYPOSMIA → It is THE REDUCED ABILITY to RECOGNIZE AND DETECT ANY ODOR.
↓
CAN BE TEMPORARY & PERMANENT.

3) HYPEROSMIA → It is THE ↑SED or EXAGERRATED OLFACTORY SENSATION.
↓
It is ALSO KNOWN AS OLFACTORY HYPERESTHESIA.
→ OCCURS IN BRAIN INJURY, EPILEPSY & NEUROTIC CONDITIONS.

*** FUNCTIONS OF MIDDLE EAR ***

- CONDUCTION → CONDUCT SOUND FROM THE OUTER EAR TO THE INNER EAR
- PROTECTION →
 - * CREATES A BARRIER THAT PROTECTS THE MIDDLE & INNER AREAS FROM FOREIGN OBJECTS.
 - * MIDDLE EAR MUSCLES MAY PROVIDE PROTECTION FROM LOUD SOUNDS.
- TRANSDUCER →
 - * CONVERTS ACOUSTIC ENERGY TO MECHANICAL ENERGY
 - * CONVERTS MECHANICAL ENERGY TO HYDRAULIC ENERGY
- AMPLIFIER →
 - * TRANSFORMER ACTION OF THE MIDDLE EAR.
 - * ONLY ABOUT $\frac{1}{1000}$ OF ACOUSTIC ENERGY IN AIR WOULD BE TRANSMITTED TO THE INNER-EAR FLUIDS (ABOUT 30 DB HEARING LOSS).
- IMPEDANCE MATCHING → AMPLIFICATION OF SOUNDS TO OVERCOME DIFFERENCE IN IMPEDANCE B/W AIR OF EAC AND THE FLUID OF INNER EAR.
- FILTERING → RESONANT FREQUENCY IS APPROXIMATELY 1000 Hz, FUNCTION AS BANDPASS FILTER.
- ACOUSTIC REFLEX → CONTRACTION OF STAPEDIUS MUSCLE IN RESPONSE TO LOUD SOUNDS.
 - * SMALL MUSCLE PROTECT THE HAIR CELLS IN THE INTERNAL EAR FROM LOUD SOUNDS.
 - * MAINTENANCE OF AIR PRESSURE ON EITHER SIDE OF THE TYMPANIC MEMBRANE.
- PREFERENTIAL SOUND PRESSURE APPLICATION → (PHASE DIFFERENCE).

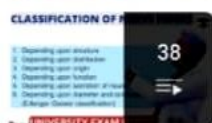
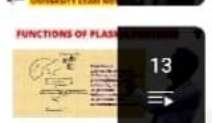
• YIELDING OF ROUND WINDOW MEMBRANE [push-pull effect]
IS NECESSARY AS INNER EAR FLUIDS ARE
INCOMPRESSIBLE.

• LARGE Tympanic membrane perforation - loss of this function
[push-pull effect] - NO MOVEMENT OF INNER
EAR FLUIDS.



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