THEORIES OF PNEUMATISATION

1. Theory of Active Endodermal epithelization
2. Mesodermal theory of potential space formation in bone marrow
3. Theory of mastoid plate retraction
4. Hypothesis of Whitmaack
5. Bateman theory
6. Diamant theory
7. Redui Theory
8. Tumarkin theory
9. Thorburn

THEORY OF ACTIVE ENDODERMAL EPITHELISATION

- Resorption of diploic bone on active invasion by epithelial cells originating from primitive tympanum.
- Epithelial invasion gives rise to mastoid air cells and air cells in relation to labyrinth.
- Vital process of pneumatisation begins in most cases before birth though **mastoid eminence** becomes prominent after birth only.

MESODERMAL THEORY OF POTENTIAL SPACE FORMATION IN BONE MARROW

- Mesodermal theory of potential space formation in bone marrow with aeration through breakdown of walls partly.

THEORY OF MASTOID PLATE RETRACTION

- This theory sees the growth of mastoid as a response to its muscle bearing duties resulting in a separation of the inner and outer plates due to pull of Muscles.
- In the potential space thus formed air enters simply as a result of atmospheric communication via eustachian tube.
- Blockage of this middle ear communication before completion of pneumatisation will result in variations or loss of pneumatization.

HYPOTHESIS OF WHITTMACK

- Mucous membrane influences the development of mastoid air cell system and normal mucosa results in a well developed mastoid air cell system.
- During birth **meconium or vernix caseosa** may enter the middle ear and produce a pathological type of mucosa and **infantile otitis** may accomplish same effect.
- This pathological mucosa will lead to inhibition or retraction of the process of pneumatisation of air cells.
- The pathological types of mucosa govern development of chronic suppurative otitis media.
**BATEMAN THEORY**

- Stated that COM is a result of infection in acellular mastoid.

**DIAMANT THEORY**

- Stated that degree of pneumatisation is determined by hereditary factors.

**REDUI THEORY**

- Necrotizing otitis media in infants can arrest pneumatisation by destroying the mucosa and replacing it with connective tissue and eventually a sclerotic bone.

**TUMARKIN THEORY**

- Eustachian tube obstruction with resultant intratympanic vacuum leads to arrest in pneumatisation.
- This occurs in young children as a result of infection and enlargement of adenoids.

**THORBURN THEORY**

- It's not the persistent eustachian tubal obstruction which is the common feature of chronic middle ear disease. Although intramastoid negative pressure is probably the limiting factor in pneumatisation.
- Intramastoid negative pressure can develop by persistent obstruction of ventilation between the mesotympanum and epitympanoantral segment without persistent eustachian tubal obstruction.

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**THEORIES OF OLFACITION**

Olfactory Pathway ➔ Olfactory epithelium ➔ Glomerular olfactory bulb ➔ Olfactory Tract ➔ Olfactory trigone ➔ Medial and lateral striae ➔ Hypothalamus ➔ central connection with amygdala and hippocampus (EBTT SHAH)

How can we detect so many different scents if our genes only code for about 1000 olfactory receptors?

**THEORIES OF ODOUR QUALITY**

**VIBRATION THEORY OF OLFACITION**

- The Vibration theory of smell proposes that a molecule's smell character is due to its vibrational frequency in the infrared range.
- The current vibration theory has recently been called the "swipe card" model
Pathophysiology

- As proposed by Luca Turin, the odorant molecule must first fit in the receptor's binding site.
- Then it must have a vibrational energy mode compatible with the difference in energies between two energy levels on the receptor, so electrons can travel through the molecule via inelastic electron tunneling, triggering the signal transduction pathway.
- The odor character is encoded in the ratio of activities of receptors tuned to different vibration frequencies, in the same way that color is encoded in the ratio of activities of cone cell receptors tuned to different frequencies of light.

Drawback

- Although vibration theory explains odor character, it does not explain intensity: why some odors are stronger than others at the same concentrations.
- Molecules that are mirror-image rotations of one another (i.e. stereoisomer); although they contain the same atoms, they can smell completely different (Vibration theory cannot explain this phenomenon).

SHAPE THEORY OF OLFACATION

- The Shape theory of smell states that a molecule's particular smell is due to a 'lock and key' mechanism by which a scent molecule fits into olfactory receptors in the nasal epithelium.
- More widely accepted than vibration theory.
- Shape pattern theory can explain specific anosmia i.e. (The inability to smell one specific compound amid otherwise normal smell perception) eg : specific anosmia to androstenone, a molecule found in armpit sweat and pork.

PENETRATION AND PUNCTURE THEORY

- Dave’s theory states that odorant molecules is able to penetrate the olfactory receptor cell and diffuse through it leaving a hole through which leakage of ions occur initiating a nerve impulse. Different shapes of molecules may leave different size holes. Depending upon the time taken by odorant molecule to diffuse and the time taken by membrane to heal (HOLE SHARPNESS FACTOR) the quality of odour varies.

ODOTOPE THEORY

- Odotope theory, also known as weak shape theory, is a theory of how olfactory receptors bind to odor molecules. The theory proposes that a combination of shape factors determine the coupling. The word itself is an analogy to epitopes.

THEORIES OF STIMULATION

OLFACATORY PIGMENT THEORY
Rosenberg proposed that odorant molecules form complex with certain olfactory pigments giving them an increased electrical conductivity.

**ENZYME THEORY**

- Activity of certain enzymes can be altered by adsorption of odorant molecules.

**PENETRATION AND PUNCTURING THEORY**

- During excitation of nerve axon, K moves out and Na moves in the cell. During resting phase pumping back of ions occurs.
- It is the exchange of ions or Puncturing of the membrane that sets off electrical discharge along the nerve.
- Dave’s theory states that odorant molecules is able to penetrate the olfactory receptor cell and diffuse through it leaving a hole through which leakage of ions occur initiating a nerve impulse.

**THEORIES OF PHONATION**

- There are currently two main theories as to how vibration of the vocal folds is initiated: (M A N)

**THE MYOELASTIC THEORY**

**AERODYNAMIC THEORY**

- A third theory, the NEUROCHRONAXIC THEORY, was in considerable vogue in the 1950s, but has since been largely discredited.
- These two theories are not in contention with one another and it is quite possible that both theories are true and operating simultaneously to initiate and maintain vibration.

**MYOELASTIC THEORY**

- The myoelastic theory states that when the vocal cords are brought together and breath pressure is applied to them, the cords remain closed until the pressure beneath them—the subglottic pressure—is sufficient to push them apart, allowing air to escape and reducing the pressure enough for the muscle tension recoil to pull the folds back together again. Pressure builds up once again until the cords are pushed apart, and the whole cycle keeps repeating itself. The rate at which the cords open and close—the number of cycles per second—determines the pitch of the phonation.

**AERODYNAMIC THEORY**

- The aerodynamic theory is based on the Bernoulli energy law in fluids. The theory states that when a stream of breath is flowing through the glottis while the arytenoid cartilages are held together by the action of the Interarytenoid muscles, a push-pull effect is created on the vocal fold tissues that maintains self-sustained oscillation. The push occurs during glottal closing, when the glottis is
convergent, whereas the pull occurs during glottal opening, when the glottis is divergent. During glottal closure, the air flow is cut off until breath pressure pushes the folds apart and the flow starts up again, causing the cycles to repeat.

**NEUROCHRONAXIC THEORY**

- This theory states that the frequency of the vocal fold vibration is determined by the chronaxy of the recurrent nerve, and not by breath pressure or muscular tension.

**Theories of Hearing**

**PLACE THEORY/ RESONANCE THEORY (Helmholtz, 1857)**

- Place theory is a theory of hearing which states that our perception of sound depends on where each component frequency produces vibrations along the basilar membrane.

**FREQUENCY THEORY / TELEPHONE THEORY (Rutherford, 1886)**

- Entire basilar membrane vibrates. Pitch is related to rate of firing of individual auditory nerve fibers.
  - The basilar membrane moves up and down due to the displacement of the perilymph and endolymph fluid in the cochlea, caused by each individual sine wave.
  - The movement of the membrane causes the hair cells in the cochlea to become excited. Each nerve correlates with a specific frequency.
  - Once that specific wave enters the cochlea, its frequency and intensity is sensitive to a specific nerve and it causes that nerve to fire.
  - The nerve cannot send another message until the message has been sent and the nerve recovers. Each nerve fiber in the auditory nerve sends the information to the auditory cortex where it assembles the information and puts it together to perceive and interpret the auditory signal.
  - Frequency theory in essence claims that human beings do not actually experience sound waves themselves, but rather vibrations on the auditory nerve, the frequencies of which are identical to the frequencies of the sound waves coming into the ear.
  - **IN SHORT**, it claimed that all sounds were encoded to the brain by neurons firing at a rate that mimics the frequency of the sound.
  - However, because humans can hear frequencies up to 20,000 Hz but neurons cannot fire at these rates, the frequency theory had a **major flaw**.

**VOLLEY THEORY**

- Volley theory, claims that multiple neurons could fire in a volley to later combine and equal the frequency of the original sound stimulus. Through more research, it was determined that because phase synchrony is only accurate up to about 5000 Hz, volley theory cannot account for all frequencies at which we hear.
  - Sequential firing in groups of 2 - 5 fibers as each fiber has limitation of 1 KHz.
PRESENT THOUGHTS

- Ultimately, as new methods of studying the inner ear came about, a combination of place theory and frequency theory was adopted.
- > 5 KHz : Place theory
- <400 Hz : Frequency theory
- 400 – 5000 Hz : Volley theory

BEKESY’S TRAVELLING WAVE THEORY

- Sound stimulus produces a wave-like vibration of basilar membrane starting from basal turn towards apex of cochlea.
- It increases in amplitude as it moves until it reaches a maximum & dies off. Sound frequency is determined by point of maximum amplitude.
- High frequency sounds cause wave with maximum amplitude near to basal turn of cochlea.
- Low frequency sound waves have their maximum amplitude near cochlear apex.
- Georg von Bekesy won Nobel prize for his traveling wave theory in 1961

THEORIES OF BONE CONDUCTION

COMPRESSION THEORY

- Skull vibration from sound stimulus leads to vibration of bony labyrinth & inner ear fluids

INERTIA THEORY

- sound stimulus leads to skull vibration but ear ossicles lag behind due to inertia.
- Out of phase movement of skull & ear ossicles leads to movement of stapes footplate causing vibration of inner ear fluids.

OSSEO-TYMPANIC THEORY

- Sound stimulus → skull vibration but mandible condyle lags behind due to inertia.
- Out of phase movement of skull & mandible → vibration of air in external auditory canal → vibration of tympanic membrane.

TONNDORF’S THEORY

- sound stimulus → skull vibration → rotational vibration of ear ossicles → movement of stapes footplate

THEORIES OF NASAL POLYPOSIS

- Most Accepted theory

THEORY OF EPITHELIAL RUPTURE
I.e. Inflammation and Tissue oedema ➔ Epithelial rupture ➔ prolapse of Lamina propria ➔ Adjacent epithelium tries to cover up the defect forming lining of polypoidal tissue ➔ If not covered up fast lamina propria continues to grow leading to polyp formation.

- Other theories include:
  1. Adenoma theory of Billroth
  2. Fibroma theory of Hoppman
  3. Necrotising Ethmoiditis theory of Woakes
  4. Taylor’s Glandular Cystic Theory
  5. Glandular Hyperplasia Theory (Krajina)
  6. Mucosal Exudate Theory of Hayek
  7. Blockade theory of Jenkins
  8. Periphelebitis and perilymphangitis theory (Eggston and Wolff)
  9. Proetz theory (for antrocoanal polyp)
  10. Bernoulli's phenomenon
  11. Mucopolysaccharide changes
  12. Mill's theory
  13. Ewing's theory
  14. Infections
  15. Vasomotor imbalance theory

**ADENOMA FIBROMA THEORY OF BILLROTH**

- Interpreted nasal polyp to be adenomas that began growing under the nasal mucosa pushing the epithelium and nasal glands outwards.
- However Hopmann disagreed with this hypothesis saying that the glandular tissue found in the tissue samples of nasal polypi studied contained only mucous glands normally found in the nasal mucosa and concluded that nasal polypi could be soft fibromas and used the term fibroma theory to explain this.
- These two theories are not currently accepted at present.

**NECROTIZING ETHMOIDITIS THEORY OF WOAKES**

- This theory suggests that ethmoiditis causes periostitis and ostitis of ethmoid bone causing bone necrosis.
- The necrotic bone initiates mucosal reaction leading on to mucosal oedema and polyp formation.
- This theory has been flawed from the very begining as no evidence of bone necrosis could be found in the polypoidal tissue studied so far.

**TAYLOR’s GLANDULAR CYST THEORY**

- Evidently this theory is based on the presence of cystic glands and mucous filled cysts in the nasal polypoid tissue.
- The probable cause for the formation of these glandular cysts could be oedema of submucosa causing obstruction to the drainage of mucoid glands present in the nasal mucosa.
- These mucous cysts expands outwards pushing the nasal mucosa causing the polyp to occur.
Taylor in his meticulous study has proved that mucous glandular cysts usually occur after the polyp has formed and hence he believed that glandular cysts could be caused by nasal polyposis and not vice versa.

**MUCOSAL EXUDATE THEORY OF HAYEK**

- Hayek believed that nasal polyp formed due to accumulation of exudate localised deep in the mucosa.
- This accumulation of exudate causes the mucosa to bulge leading to polyp formation.
- Nasal mucosal glands and tubuloalveolar glands are also displaced outwards. These glands are hence found in the distal part of the polyp.

**BLOCKADE THEORY OF JENKINS**

- This theory is based on the premise that development of nasal polypi is almost always preceded by certain degree of nasal mucosal inflammation.
- The inflammation could be the result of either infection / allergy. Histologically polyp itself is accumulation of intracellular fluid dammed up in a localized tissue.
- If this blockage persists polyp develop, if the blockage covers a large area then multiple polypi forms. This theory doesn't explain why nasal polyp prefers certain areas of nasal cavity.

**PERIPHLEBITIS / PERILYMPHANGITIS THEORY OF EGGSTON AND WOLFF**

- This theory is based on the premise that recurrent infections of nasal mucosa blocks intercellular fluid transport mechanism in the mucosa.
- This is always associated with oedema of lamina propria.
- This theory is based on the demonstration of chronic vascular changes in the nasal mucosa in response to inflammation.
- Histologically these changes are supposed to be rather diffuse and hence cannot be used to explain the pathogenesis of nasal polypi which can always be localised to certain areas of nasal cavity.

**GLANDULAR HYPERPLASIA THEORY OF KRAJINA**

- According to Krajina chronic inflammation of nasal mucosa cause local hyperplasia of nasal mucosal glands.
- These hyperplastic glands will cause bulging of nasal mucosa. In addition to glandular hyperplasia changes that occur in the blood vessels will cause oedema in the region of the middle meatus.
- This in turn increases nasal mucosal oedema. Studies have shown that the number of nasal mucosal glands are the same in polypoidal as in the normal tissue.

**PROETZ THEORY FOR DEVELOPMENT OF ANTROCOANAL POLYP**

- Proetz suggested that this disease could be due to **faulty development of the maxillary sinus ostium**, since it was always been found to be **large** in these patients.
- Hypertrophic mucosa of maxillary antrum sprouts out through this enlarged maxillary sinus ostium to get into the nasal cavity.
The growth of the polyp is due to **impediment to the venous return** from the polyp. This impediment occur at the level of the maxillary sinus ostium. This venous stasis increases the oedema of the polypoid mucosa thereby increasing its size.

**BERNOULLI'S PHENOMENON**

- Pressure drop next to a constriction causes a suction effect pulling the sinus mucosa into the nose.

**MUCOPOLYSACCHARIDE CHANGES**

- Jakson postulated that changes in mucopolysaccharides of the ground substance could cause polyp

**MILL'S THEORY**

Mills postulated that antrochonal polyp could be maxillary mucoceles which could be caused due to obstruction of mucinous glands

**EWING'S THEORY**

- Ewings suggested that an anomaly which could occur during maxillary sinus development could leave a mucosal fold close to the ostium.
- This fold could later be aspirated into the sinus cavity due to the effects of inspired air causing the development of antrochonal polyp

**INFECTIONS**

**VASOMOTOR IMBALANCE THEORY**

Why Osteomeatal area is Most commonly affected during Polyp Formation ???

- Osteomeatal complex is most common site of development of polyp.
- It is proposed that 'Touching mucous membranes' in the narrow ostiomeatal complex results in the release of pro inflammatory cytokines from epithelial cells.
- Another possibility is an influence of special airflow, air current and pressure in the upper part of the nose i.e. Bernoulli’s phenomenon
- Finally, it may be of significance that the nerve endings near the borderline between the nose and paranasal sinuses are thin and may easily become damaged by cytotoxic proteins, released by eosinophils

**Preauricular sinus theories**

- Embryological fusion: Incomplete fusion of Hillocks
- Ectodermal infolding: Isolated ectodermal infolding
- Incomplete closure of dorsal part of first pharyngeal groove: (accepted)