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THE EUSTACHIAN TUBE, THE OTOLOGIST'S ENIGMA

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The Eustachian tube (E.T.) has been an engima for the otologist for the last few decades. Various study groups have been established on this subject ;to mention a few :
- "The middle ear research" group set up in the University of Lund, Sweden, 1960.

- "The Bluestone group", in Pittsburgh (U. S. A.).
- "The Scientific research, of the Eustachian tube", group. 1988 under Tadami Kumazawa.

Although recognised as early as 500 BC, and designated by Alcmaeon of Sparta, the first definite description was given by the 16th century Italian anatomist, Bartolommeo Eustachio in 1564 in his magnum opus – 'De Auditus organis'.

The eustachian tube has an approximate length of 37 mm and forms an air conduct of the middle ear cavity, extending from its anterior wall to the rhinopharynx.

Grossly the tube can be divided into:

- (i) The Cartilaginous (medial two-thirds) part.
- (ii) The Osseous. (external one-third) part.
(Hollinshead, 1968).

"From the pharyngeal orifice, the tube travels upwards, backwardss and outwards. Thus the tympanic ostium is 2.0 to 2.5 cm. higher than the pharyngeal ostium in the adult", Pulec, 1970. On the contrary, in infants, the tube is relatively wider, shorter, and more horizontal in position, affording easier access for infection ascending from the pharyngeal ostium, Holborow, 1970.

The tubal mucosa shows a gradual transition from a pseudostratified ciliated columnar epithelium with abundant goblet cells at its pharyngeal end to a low columnar ciliated epithelium lining the entire tube, the latter extends into the middle ear as far as the promontory and thins to cuboidal epithelium, Lim et al, 1967; Hentzer, 1970.

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Tensor veli palatini, tensor tympani, the levator veli palatini and the salpingopharyngeus muscles control the mechanics of the eustachian tube, Proctor, 1973; Dickson, 1975. Holmquist et al., 1972.

Physiological aspects :

The middle ear (M. E.) is one of the gas pockets within the human body and various mechanisms, viz. ventilation, diffusion and perfusion, through the enclosing structures are involved (Rahn and Canfield, 1955).

Swen Ingelstedt 1976, considered the auditory tube to be in a state of equilibrium with two opposing forces acting on it.

1. The closing forces.
2. The opening forces.

The Closing Forces

They keep the compliant tube collapsed and can be subdivided into :

- a) Luminal factors.
- b) Extraluminal factors.

a) Luminal Factors (Mucosal Factors)

They further include :

- i) The viscous secretions by their 'cohesiveness' serve to form mucus layer which cilia are unable to propel and blocks the tubal lumen. Moreover, the 'adhesiveness' of the secretions even holds the opposing walls of the tube together.
- ii) The surface tension of the mucus film lining the air filled middle ear cavity and the eustachian tube orifice holds the mucus lining together and counters the opening forces.
- iii) The blood content of Mucosal Vessels.

The pressure dependant volumetric changes of the mucosa play an important role in the patency of the eustachian tube (E. T.)

Evidences in support are :-

Postaural Hydrostatic venous pressure changes. The mean volume of the air passing through the tube is reduced by 2/3 of the sitting position (90°), on recumbency, due to congestion in the internal jugular vein at the base of skull Perlman, 1939. Rundcrantz, 1969; Physical activity, increases sympathetic tone with a decongestive effect on the mucosa.

b) Extraluminal Factors: (Ingelstedt, 1976).

- i) Elasticity of tubal cartilage and peritubal tissues.

(Ostmann's fat pad, internal pterygoid muscle laterally, body of levator palati muscle inferiorly, middle meningeal and ascending pharyngeal vessels).

Due to elasticity of these constituents, the tube spontaneously resumes its collapsed shape after distension.

ii) The Hydrostatic Peritubal tissue pressure :

Due to high tissue vascularity, the peritubal pressure is similarly influenced by variations of the venous pressures with posture as in (a) (iii).

"The tube opens only during swallowing : there is increased loudness of the tuning fork, held at the nose during swallowing", Politzer, 1902.

"The eustachian tube patency obtained during swallowing gets compromised in acute and chronic inflammation and their sequelae". Miller, 1965; Flisberg, 1966; Sidentop, 1968; Holmquist, 1969; surmised from their studies that, a very high percentage of chronic otitis media cases were associated with an abnormal tubal function. Arnold and Cohen 1979 summarization of the pathogenesis of middle ear disorders, involved the presence of :

- 1) A mechanism inducing negative middle ear pressure. Alberti, 1974; Cooper, 1801; Politzer, 1862; Bezold, 1883 and Flisberg, 1963.
- 2) Pathophysiological changes associated with an inflammatory mucosa. Tos, 1974; Lim, 1967; Sade, 1990.

Cooper 1801 described deafness resulting from a closed eustachian tube and devised myringotomy to treat such deafness.

The Opening Forces :

They are namely :

- a) The muscular forces.
- b) The middle ear nasopharynx pressure gradient.

A) The Muscular Opening of the Tube :

In this case, the collapsed tube is opened by the contraction of the muscles, during swallowing or yawning.

The tensor veli palatini is the only muscle which functions to open the eustachian tube, Rich, 1920.

The tensor veli palatini dilates the collapsed tubal orifice in an inferior and antero-lateral vector, during swallowing or yawning, while relaxation of the muscle allows the tube

to resume its closed state Holmquist, 1972. Hollinshead, 1968.

The tensor tympani muscle exerts a traction on its attachment on the malleus and increases the middle ear pressure, by a medial displacement of the drum thus facilitating tubal opening. Moreover, the origin of this muscle from the cartilaginous portion of the tube and adjoining area may influence the opening of the tube at this point. Holmquist, 1972. "The levator palatini which forms the bottom of the tube, merely supports the opening". Holmquist, 1972. Rich, 1920. The salpingopharyngeus muscle plays a minor role in the tubal dynamics. Holmquist, 1972; Rich, 1920; Ingelstedt, 1976.

B) Pressure opening of the tube (Ingelstedt, 1972) :

In this case the collapsed tube is opened solely through distension by pressure without any muscular activity.

During deflation, the flow of gas occurs from the middle ear to the nasopharynx e. g. owing to relative overpressure in the middle ear during ascent, or by a relative underpressure in the nasopharynx during a reversed Valsalva's Manoeuvre.

Dysfunction of the eustachian tube and its consequences:

CONSEQUENCES OF TUBAL DYSFUNCTION

Tubal dysfunction due to mechanical or functional factors may predispose to a "High Risk" state of "High Negative Pressure" (H.N.P.) Cantekin, 1979 and Bluestone, 1979.

The high Negative Pressure developed as a result of continuous gas absorption in a closed middle ear may lead to one of the following sequelae:

1) The early retraction of the tympanic membrane, due to slight to moderate negative pressure.

2) The state of sterile transudation in the middle ear, from the hyperemic mucosa due to increase in negative middle ear pressure. Consequent to O₂ and N₂ absorption (ex-vacuo theory).

3) The state of retraction of the tympanic membrane with consequent development of adhesive otitis media and its sequelae, due to persistent tubal occlusion. (Shambaugh, Glasscock, 1990; Bluestone, Cantekin, 1978; Berry, M. Tos 1972).

The various clinical phases can be diagnosed and arrested

at individual levels. Failure to control the pathology at an early stage may lead to irreversible changes.

Sade and Lutnz 1990, propounded, in tubal dysfunction there is :

- Compromised Ventilation :-

Leads to sinking of the tympanic membrane in the region of posterior tympanum corresponding to the area of profuse gas absorption, i.e. the region lined by non-ciliated flat cells.

- Compromised Mucociliary Clearance:-

Leads to stagnation of inflammatory exudate causing weakening of the corresponding part of the tympanic membrane predisposing collapse.

Ojala, 1953 recognised a close association of exudative middle ear disease and further suggested the following stages :-

1) An acute stage of Otosalpingitis with eustachian tube obstruction and inflammatory reaction through-out the entire middle ear cleft resulting in mucosal damage and the production of fibrinous exudate.

2) An intermediate stage of organisation of the exudate and formation of adhesions. In this stage, the mucosal lining of the middle ear and mastoid air cells is oedematous and there is a residual exudate sometimes containing cholesterol crystals. The mastoid air cells become filled with connective tissue and there is surrounding bone sclerosis.

3) The terminal stage is which mature fibrous adhesions are present together with diminished aeration of mastoid air cells. In addition to mucosal changes there may be evidence of bone absorption especially affecting the long process of the incus.

Ojala, 1953 further stated that the commonest sites of adhesions were the oval and round windows, ossicles, Prussak's space and epitympanum.

Cawthorne, 1956, stated that the drums in adhesive otitis media were opaque and adherent to the promontory.

In the post operative period, state of 'High negative pressure' (HNP) consequently causes retraction or

invagination of the neotympanum. The commonest sites involved may be:

- 1) The 'Flaccid', pars flaccida region.
- 2) The 'Atrophied and Weakened', postero-superior quadrant of the pars tensa region. Khanna and Tonndorf, 1972.

The early retraction pockets are reversible while the persistent retraction pocket may progress to atelectasis and may adhere irreversibly to the ossicles or surrounding structures or both. Collection of squamous debris and associated enzymes in these pockets amounts to the formation of a frank cholesteatoma. In fact there is a progressive transition from a retraction pocket to Stenotympania to adhesive otitis and finally to cholesteatoma. Zechner, 1983; Tos, 1973; Venker, 1973; Bluestone et al. 1978.

Under these circumstances treatment modalities change from early self inflation of the middle ear cavity to surgical lysis of the adhesions with or without ossicular reconstructions and ultimately tympanomastoidectomy as required.

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