EUSTACHIAN TUBE DYSFUNCTION AND OTITIS MEDIA

Cuneyt M. Alper, M.D.
Professor, Department of Otolaryngology and Clinical and Translational Research Institute
Director, Pediatric Otolaryngology Fellowship Program
Division of Pediatric Otolaryngology
Children’s Hospital of Pittsburgh of UPMC
Department of Otolaryngology
University of Pittsburgh School of Medicine
Discoverers

- **Bartholomeus Eustachius (1510-1574)**
  - First description of “auditory” tube in *Epistola de auditus organis*

- **Antonio Maria Valsalva (1666-1723)**
  - Maneuver for testing the patency and for the treatment of middle ear effusion

- **Joseph Toynbee (1815-1866)**
  - Extensive cadaver work

- **Adam Politzer (1835-1920)**
  - Middle ear inflation to treat ear diseases
Various forms of OM

- Otitis Media
- Acute OM
- Recurrent AOM
- Persistent AOM
- Middle ear effusion
- Otitis Media with effusion (OME)
- Chronic OME
- Suppurative OM
- Chronic suppurative OM (CSOM)
- CSOM with cholesteatoma
Questions

- What are the risk factors for OM?
- What is the association between ME and ET?
- What does the ET do?
- Why is ET necessary?
- What happens if ET does not open at all?
- What causes ET dysfunction?
- Do viral infections affect ETF?
- What are the test methods to assess the ETF?
- What are the treatment options for ETD?
What are the risk factors for OM?

- **Host Factors**
  1. Familial predisposition
  2. Immature/impaired immunology
  3. Method of feeding (breast or bottle)
  4. Sex
  5. Race

- **Anatomic/Physiologic Dysfunction**
  1. Eustachian tube dysfunction
  2. Cleft palate, submucous cleft

- **Environmental Factors**
  1. Day-care attendance
  2. Smoking in households

- **Infection**
- **Allergy**
What is the association between middle ear (ME) and Eustachian Tube (ET)?

- Anatomically contiguous structures
- Embryology - ME is extension of the ET
- Ectodermal indentation form the ear canal
- Endodermal indentation (pharyngeal pouch) at nasopharynx, extend laterally to form the ET, ME, Mastoid air cells
- These two meet to form the tympanic membrane
Eustachian TUBE

Ear

- inner ear
- middle ear
- ear drum

external ear
eustachian tube
Eustachian Tube: An Organ

- Organ, in biology, is a group of tissues in a living organism that have been adapted to perform a specific function.
- ET has, lumen surrounded by distinct mucosa, submucosa with secretory glands, surrounded by bone, cartilage and soft tissue, and muscles specific to its function, innervation and blood supply, lymphatics.
- ET, ME, Mastoid, TM form an organ system.
What does the ET do?

- Stays closed at rest with tissue pressures
- Protects ME from
  - Pressure changes in the nasopharynx
  - Sounds in the nasopharynx from talking or eating
  - Reflux of secretions /virus /bacteria
- Clearance of ME
  - Secretions, effusion, infection, organisms
- Equalizing the ME pressure to environment
  - With passive opening or active muscular function
Why is the ET necessary?

- First, we need to ask: “Why do we need air (gas, instead of fluid) in the middle ear?”
  - To enhance the hearing: When there is fluid, there is 15-35dB conductive hearing loss.

- Why do we need ME pressure equal to the ambient pressure?
  - To maximize hearing efficiency: When there is pressure difference TM stretches in or out which dampens the vibration/sound transmission.
What happens if ET does not open at all?

- Negative ME pressure develops
- TM gets retracted
  - TM fibers lose their strength
  - Retraction pockets, atelectasis of the ME, erosion of the ME ossicles, cholesteatoma may develop
- Transudation and exudation of effusion occurs
  - ME gets filled with effusion
Negative ME pressure complications and sequelae

- Effusion
- Retraction / Retraction pocket
- Atelectasis
- Adhesive otitis
- Ossicular erosion
- Hearing loss
- Cholesteatoma
- Temporal and intracranial complications
Why does negative ME pressure develop?

- “Physics”
- “Gas laws”
- Each specific gas in the ME is in constant exchange with gasses dissolved in blood
  - Each specific gas exchanges in the direction to equalize the partial pressures
  - Each gas exchanges independent of other
each gas is limited with its properties
ME Pressure-Regulation

The summed effect of all physiologic processes that contribute to maintaining near equivalence between ME and ambient pressures

- Maintains the neutral tympanic membrane position for efficient ambient-inner ear sound pressure transfer.
- Maintains the ME Pressure greater than that which precipitates pathology (≈ -200mmH2O ref ambient).
PATHWAYS FOR GAS EXCHANGE

- NP (ET) ↔ ME
- TYMPANUM ↔ MACS
- TYMPANUM ↔ MEM
- MACS ↔ MEM
- TYMPANUM (TM) ↔ EEC
- MEM ↔ BLOOD
- BLOOD ↔ RBC

1. FOR MOST PATHWAYS, EXCHANGE RATES ARE GAS SPECIFIC
2. GAS EXCHANGE DEPENDS ON EXTANT PRESSURE GRADIENT
EXCHANGE CHARACTERISTICS

- **TransTM exchange:**
  - Diffusion across a barrier
    - TM thickness, surface area
    - ΔPg (ME-ambient), Sg in TM

- **TransMEM exchange**
  - N2, H2O perfusion-limited
    - MEM blood flow (Q)
    - ΔPg (ME-blood), Sg
  - CO2, O2 diffusion-limited
    - MEM thickness, surface area
    - ΔPg (ME-blood), Sg

- **TransET exchange:**
  - Bolus transfer of gas volumes
    - ET opening time, Resistance,
    - ΔP (ME-NP)
What is the State of ME Gas Composition When ET Functions Normally?

**Inner Ear**
- PO2 = 40
- PCO2 = 46
- PN2 = 573
- PH2O = 47

**Round Window**
- PO2 = 103
- PN2 = 573

**Nasopharynx**
- PO2 = 37
- PCO2 = 37
- PN2 = 573
- PH2O = 47

**Middle Ear**
- PO2 = 40
- PN2 = 620
- PCO2 = 50
- PH2O = 47

**Mucosa**
- PO2 = 40
- PCO2 = 46
- PN2 = 573
- PH2O = 47

**Blood**
- PO2 = 158
- PN2 = 596
- PCO2 = 0.3
- PH2O = 5.7
What Happens to ME Gas Composition When ET Does Not Open?

What Happens to ME Gas Composition When ET Does Not Open?

**MEP = Ambient**

**Ambient-200 mmH2O**

**Ambient-400 mmH2O**
Why is negative ME pressure unsustainable?

- Any closed gas cavity in the body will eventually develop ~ -600mmH₂O pressure over weeks.
- TM retraction reduces the ME volume, maintains the pressure.
- When pressure decreases below -200-300 mmH₂O, fluid leaks into the ME cavity with or without protein (transudate/exudate).
- Fluid volume reduces the gas space and maintains the pressure.
- Eventually fluid fills the ME.
An in vivo chinchilla study was conducted to test the effect of negative pressure. Pressure levels for 1 hour were 0, -200, -400, and -600 mmH2O. No change in the histology of ME mucosa was observed at 0 and -200 mmH2O. At -400 mmH2O, there was edema and some effusion. At -600 mmH2O, disruption of capillaries and effusion filling the bullae were observed.
What causes ET dysfunction?

- Eustachian tube mechanical blockage
  - Inside the lumen, or extrinsic
- ET functional obstruction
  - Inability to open passively, despite large pressure differences between ME and ambient
    - Flying, diving
  - Inability to open with active function
    - Swallowing, yawning
- Could be continuous of intermittent
Risk factors for OM related to ET dysfunction


<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Yes</th>
<th>Maybe</th>
<th>No</th>
<th>Unknown</th>
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<tbody>
<tr>
<td>Infant age</td>
<td>+</td>
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<tr>
<td>Male gender</td>
<td></td>
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<tr>
<td>Genetic predisposition</td>
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<tr>
<td>Sibling order</td>
<td>+</td>
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<tr>
<td>Early onset of otitis media</td>
<td>+</td>
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<tr>
<td>Child day care</td>
<td>+</td>
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<tr>
<td>Season</td>
<td></td>
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<tr>
<td>Lower socioeconomic status</td>
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<tr>
<td>Smoking in household</td>
<td></td>
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<tr>
<td>Allergies</td>
<td>+</td>
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<tr>
<td>Use of pacifiers</td>
<td></td>
<td></td>
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<tr>
<td>Prone position in infancy</td>
<td>+</td>
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<tr>
<td>Wood-burning stoves</td>
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<td></td>
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<tr>
<td>Certain ethnic backgrounds</td>
<td>+</td>
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<td></td>
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<tr>
<td>Craniofacial malformations</td>
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<tr>
<td>Immature/impaired immunology</td>
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<tr>
<td>Lack of breast-feeding</td>
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<tr>
<td>Gastroesophageal reflux</td>
<td>+</td>
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</tbody>
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Developmental differences in ET-ME between infants and adults


<table>
<thead>
<tr>
<th>Anatomic Features of the Eustachian Tube</th>
<th>Compared with the Adult, in the Infant It Is</th>
<th>Reference</th>
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</thead>
<tbody>
<tr>
<td>Length of tube</td>
<td>Shorter</td>
<td>Sadler-Kimes et al, 1989&lt;sup&gt;6&lt;/sup&gt;; Ishijima et al, 2000&lt;sup&gt;7&lt;/sup&gt;</td>
</tr>
<tr>
<td>Angle of tube to horizontal plane</td>
<td>10° vs 45°</td>
<td>Proctor, 1973&lt;sup&gt;8&lt;/sup&gt;</td>
</tr>
<tr>
<td>Angle/length of TVP to cartilage</td>
<td>Variable vs stable angle, shorter attachment</td>
<td>Swarts and Rood, 1993&lt;sup&gt;9&lt;/sup&gt;; Suzuki et al, 2003&lt;sup&gt;10&lt;/sup&gt;</td>
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<tr>
<td>Lumen</td>
<td>Smaller area/volume</td>
<td>Kitajiri et al, 1987&lt;sup&gt;11&lt;/sup&gt;; Suzuki et al, 1998&lt;sup&gt;12&lt;/sup&gt;; Ishijima et al, 2002&lt;sup&gt;13&lt;/sup&gt;</td>
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<tr>
<td>Cartilage volume</td>
<td>Less</td>
<td>Takasaki et al, 2000&lt;sup&gt;14&lt;/sup&gt;</td>
</tr>
<tr>
<td>Cartilage cell density</td>
<td>Greater</td>
<td>Yamaguchi et al, 1990&lt;sup&gt;15&lt;/sup&gt;</td>
</tr>
<tr>
<td>Elastin at hinge portion of cartilage</td>
<td>Less</td>
<td>Matsune et al, 1993&lt;sup&gt;16&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ostmann’s fat pad</td>
<td>Relatively wider</td>
<td>Aoki et al, 1994&lt;sup&gt;17&lt;/sup&gt;; Orita et al, 2002&lt;sup&gt;18&lt;/sup&gt;; Orita et al, 2003&lt;sup&gt;19&lt;/sup&gt;</td>
</tr>
<tr>
<td>Mucosal folds</td>
<td>Greater</td>
<td>Sudo and Sando, 1996&lt;sup&gt;20&lt;/sup&gt;</td>
</tr>
<tr>
<td>Lumen glands</td>
<td>Variable type</td>
<td>Orita et al, 2002&lt;sup&gt;18&lt;/sup&gt;</td>
</tr>
<tr>
<td>Connective tissue lateral to tube</td>
<td>Different</td>
<td>Orita et al, 2003&lt;sup&gt;21&lt;/sup&gt;</td>
</tr>
<tr>
<td>Middle-ear volume</td>
<td>Smaller</td>
<td>Ikui et al, 2000&lt;sup&gt;22&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
Differences between a child and an adult
Causes of acquired ET dysfunction

- Changes in ambient pressure (flying/diving)
- Viral URI’s
- Nasal allergies
- GERD
- Adenoid hypertrophy
- Adenoiditis / Nasopharyngitis
- Sinusitis
- Nasopharyngeal mass
increased NP secretions
altered NP bacterial colonization
activation of inflammatory cascades
edema of nasopharynx
obstruction of ET orifice
viral OM
viral URI
Genetic Susceptibility
Genetic Susceptibility
high negative MEP
dysfunction
Viral URI is most common cause of acquired/intermittent ET dysfunction and negative ME pressure

Most viral URIs lead to negative ME pressure (more in children)

Negative MEP is from ET dysfunction

Most OM is preceded by viral URI (50-70%)

However, most viral URI does not lead to OME or AOM
Indirect methods in routine clinical care=
  ▶ Pneumatic otoscopy = retraction
  ▶ Tympanometry = negative pressure

A number of more complex and sophisticated test methods are available in a few research centers
Sonotubometry Testing
Forced Response Testing

Diagram showing the setup of the forced response testing, including the syringe pump, pressure and flow rate transducers, perforated tympanic membrane, middle ear, and eustachian tube. The graph shows the pressure (mm H2O) and flow (ml/min) over time (sec), with markers for opening and closing pressure, and swallow events.
Inflation-Deflation Testing
Tubomanometry Testing
Mass Spectrometer for Gas Analysis
Recommended / Followed Treatment Methods

- **Medical treatment**
  - Nasal topical steroids
  - Antihistamines
  - GERD diet
  - H-2 Blockers
  - Proton Pump Inhibitors

- **Adenoidectomy /Revision adenoidectomy**

- **Repeated Valsalva**

- **Auto-Inflation**
Potential New Surgical Methods

- New Surgical Treatment Methods*:
  - Laser tuboplasty
  - Cartilage framework tuboplasty
  - Balloon Dilation Tuboplasty

* EFFICACY OF THE NEW SURGICAL METHODS IS NOT YET SHOWN IN CONTROLLED STUDIES