EUSTACHIAN TUBE DYSFUNCTION AND OTITIS MEDIA

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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 eat diseases

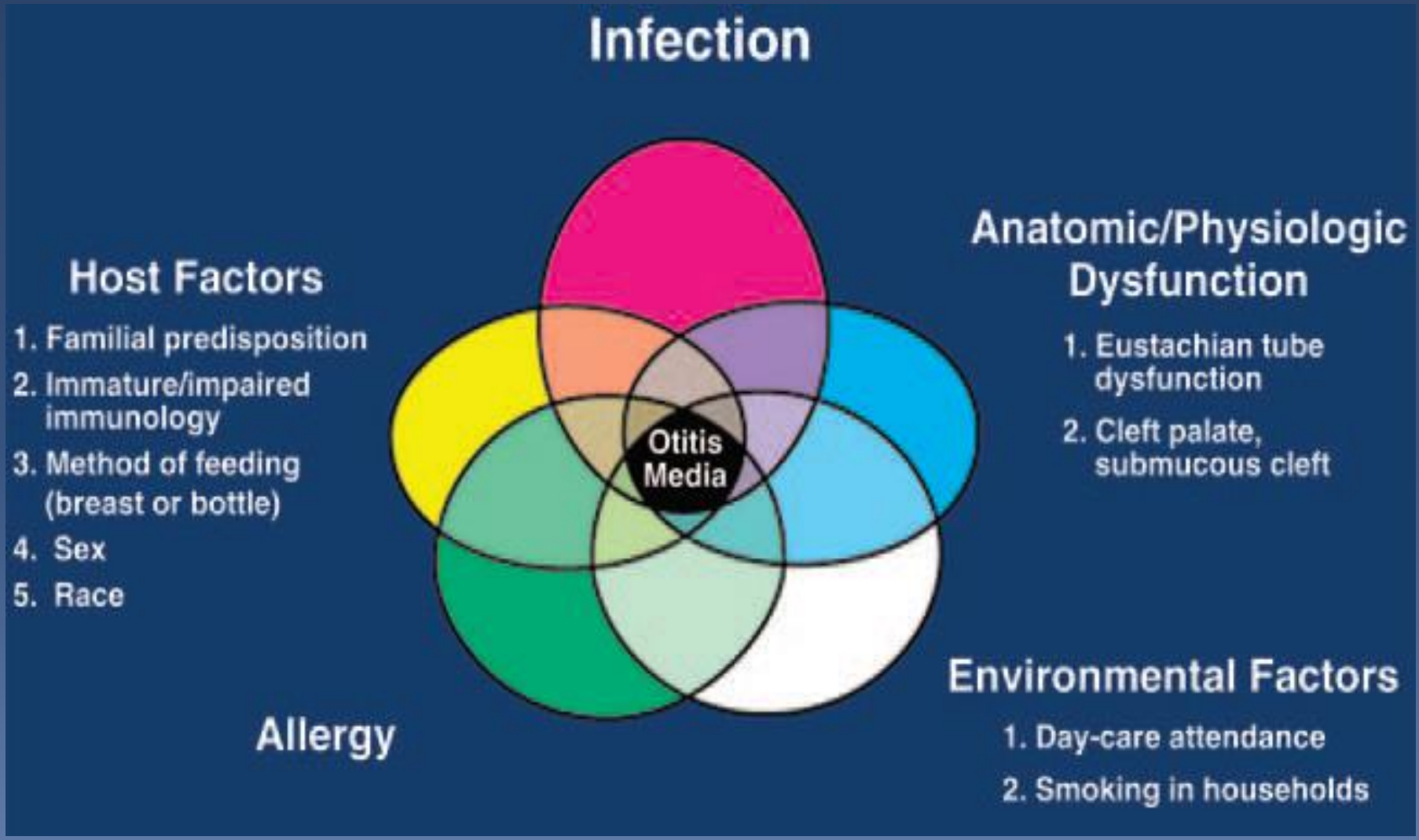
Various forms of OM

- 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?

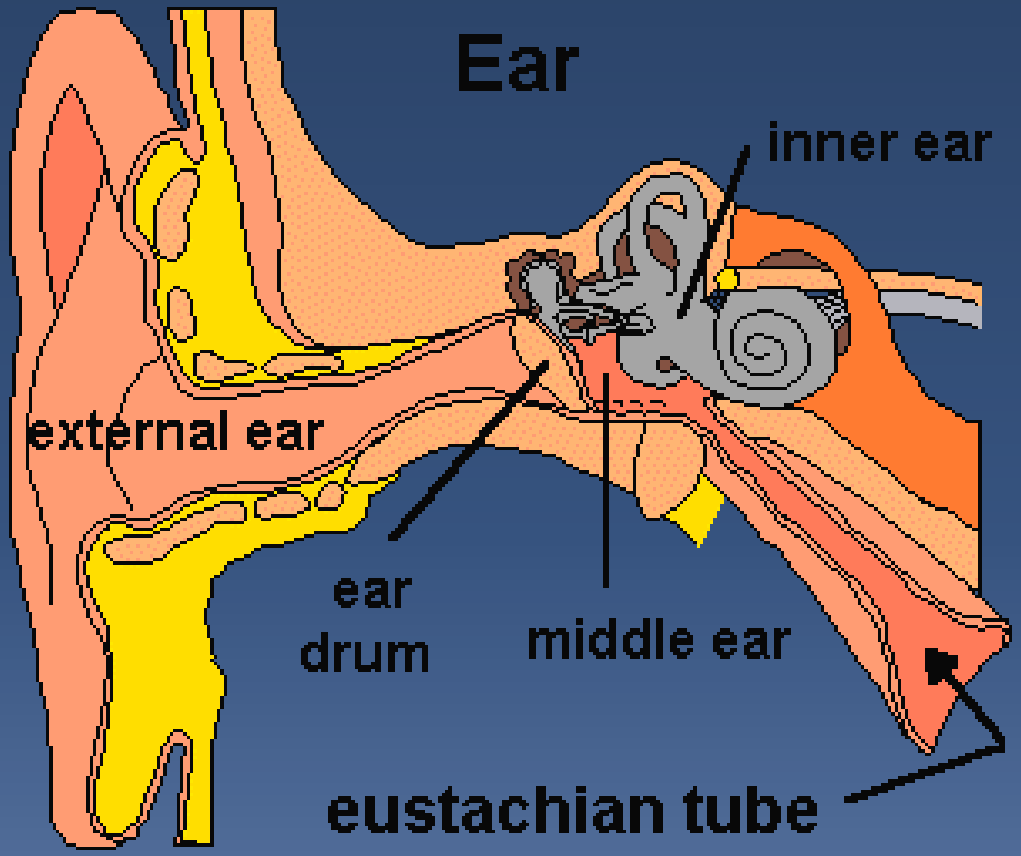
What are the risk factors for OM?



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**



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 loose 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 sequalae

- 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
  - Exchange rate of each gas is limited with its properties

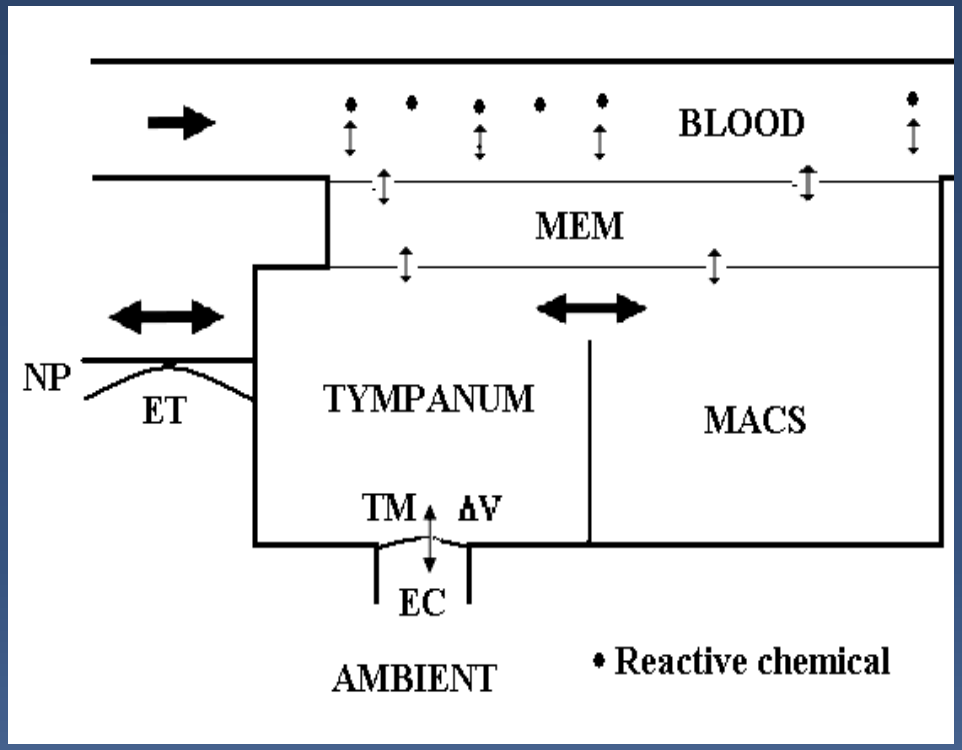
ME Pressure-Regulation

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

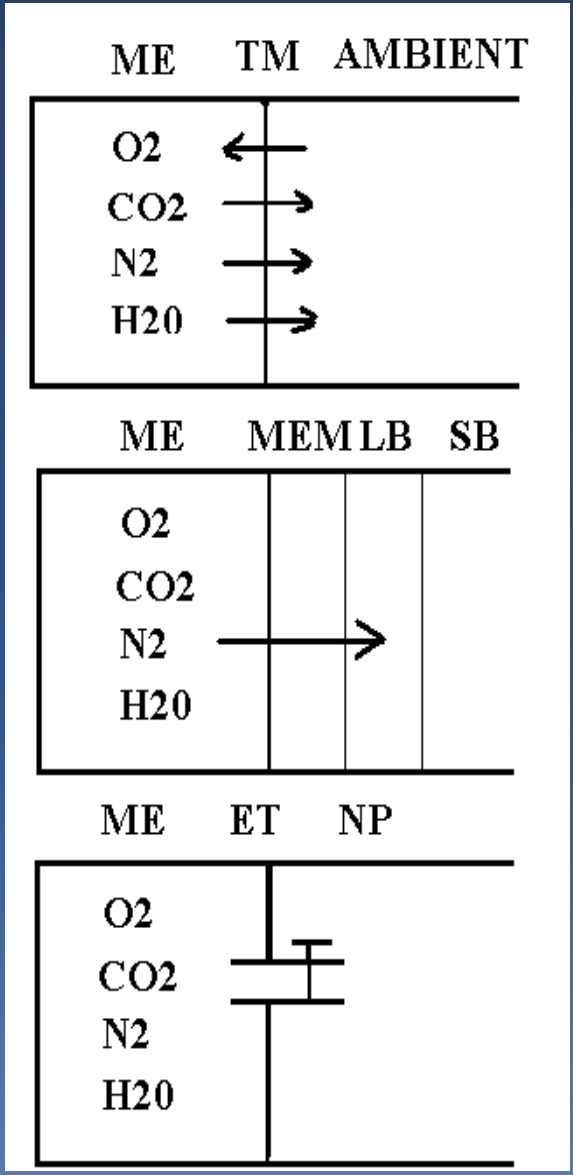
- NP (ET) ⟷ ME
- TYMPANUM ⟷ MACS
- TYMPANUM ⟷ MEM
- MACS ⟷ MEM
- TYMPANUM (TM) ⟷ EEC
- MEM ⟷ BLOOD
- BLOOD ⟷ RBC



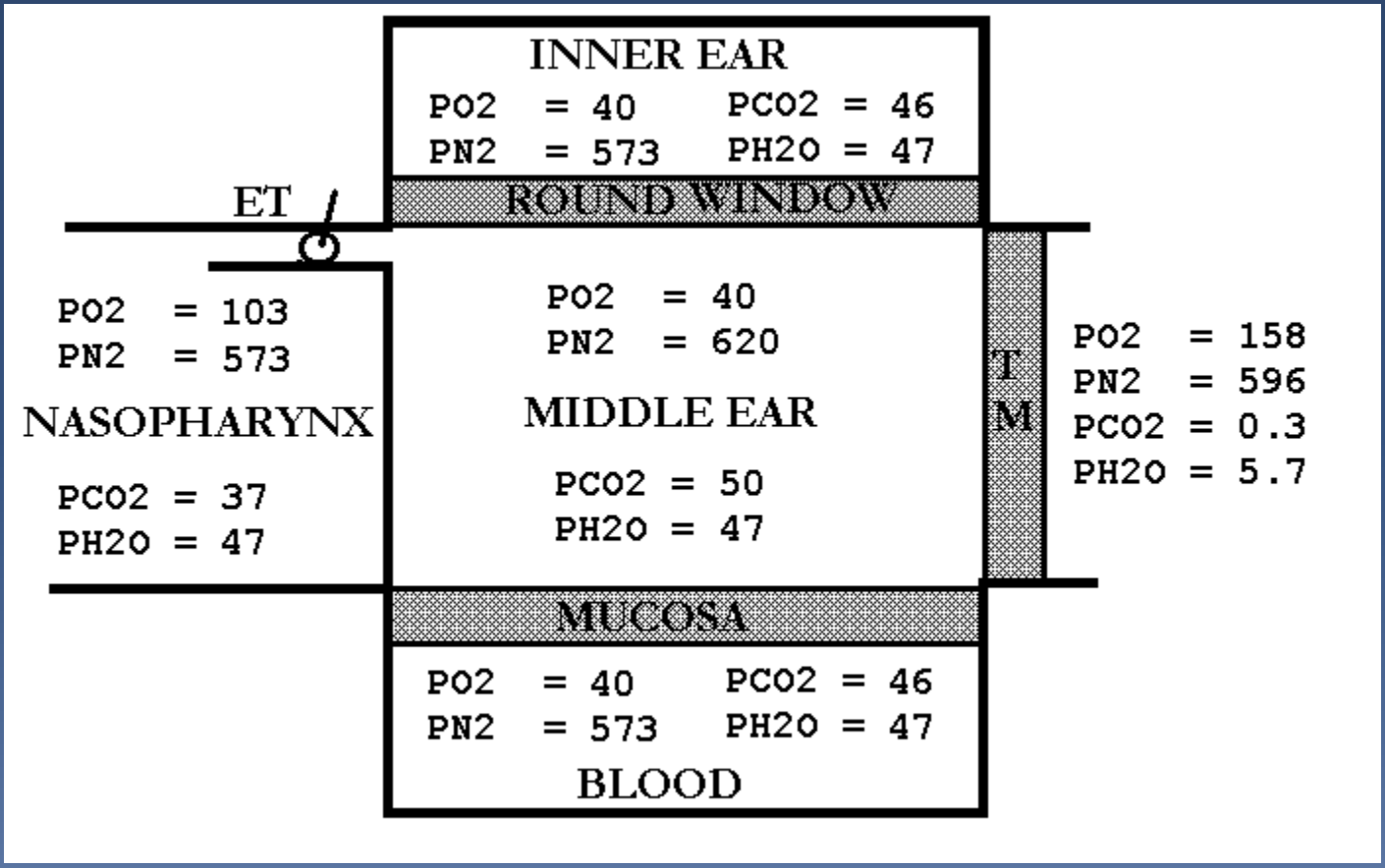
1. FOR MOST PATHWAYS, EXCHANGE RATES ARE GAS SPECIFIC
2. GAS EXCHANGE DEPENDS ON EXTANT PRESSURE GRADIENT

EXCHANGE CHARACTERISTICS

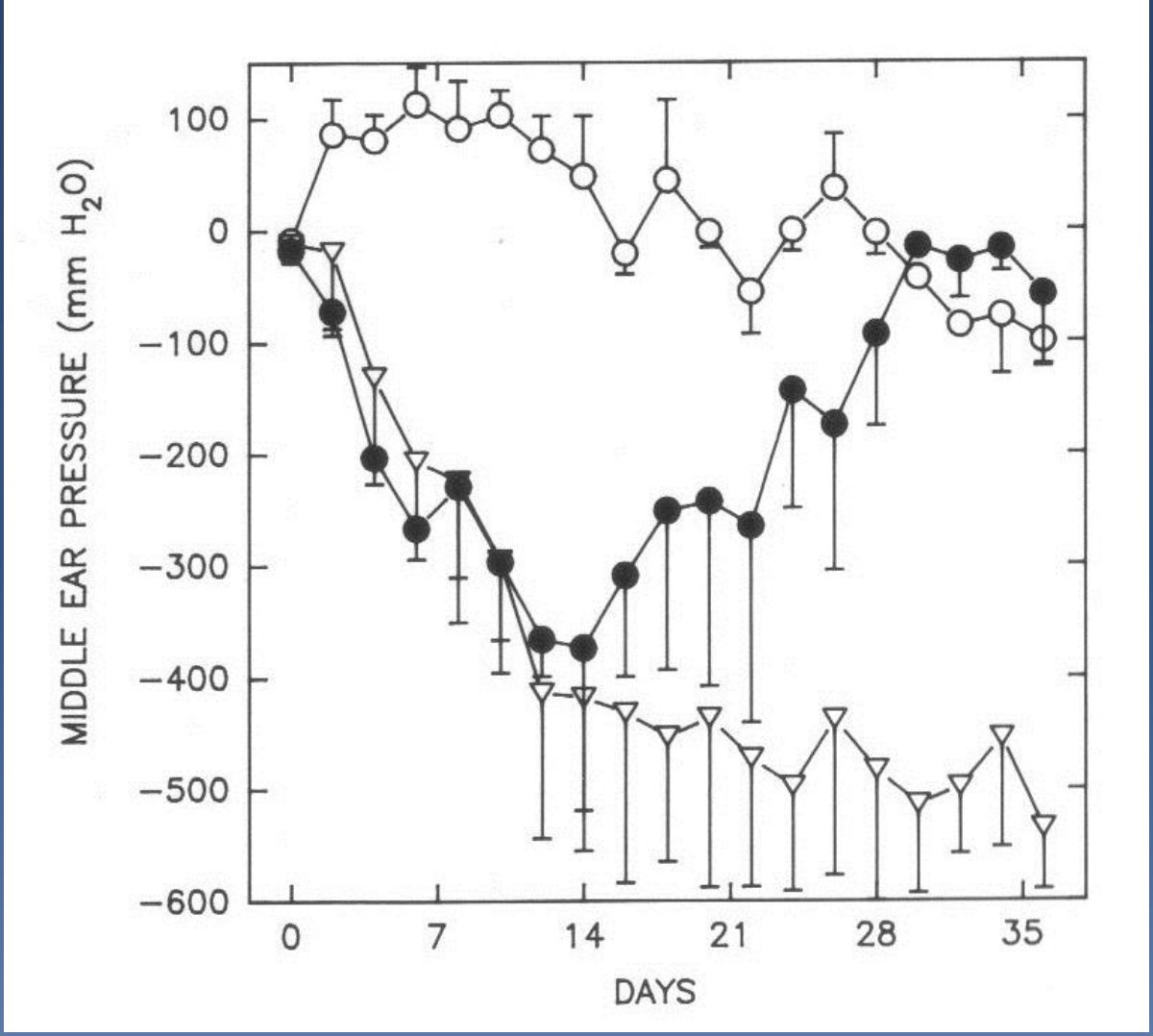
- **TransTM exchange:**
  - Diffusion across a barrier
    - TM thickness, surface area
    - ΔPg (ME-ambient), Sg in TM
- **TransMEM exchange**
  - N2, H2O perfusion-limited
    - MEM blood flow (Q)
    - ΔPg (ME-blood), Sg
  - CO2, O2 diffusion-limited
    - MEM thickness, surface area
    - ΔPg (ME-blood), 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?



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



Alper CM, Tabari R, Seroky JT, Doyle WJ. Magnetic resonance imaging of otitis media with effusion caused by functional obstruction of the Eustachian tube. Ann Otol Rhinol Laryngol 1997;106:422-431.

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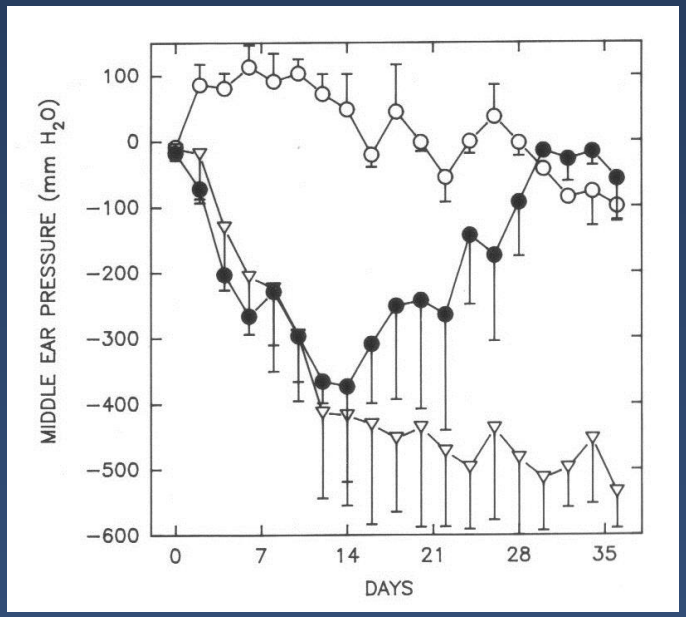
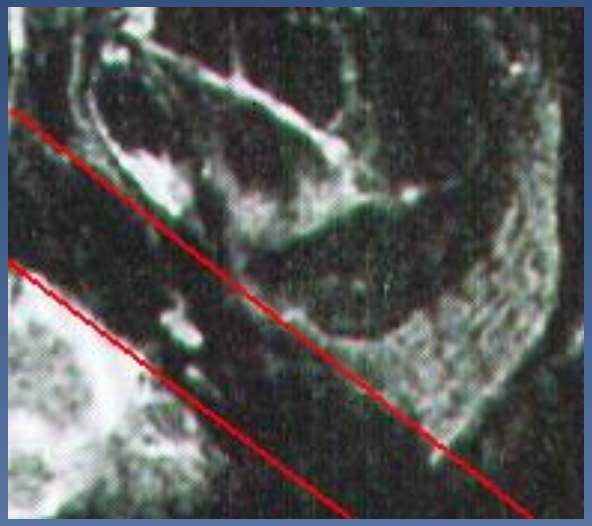
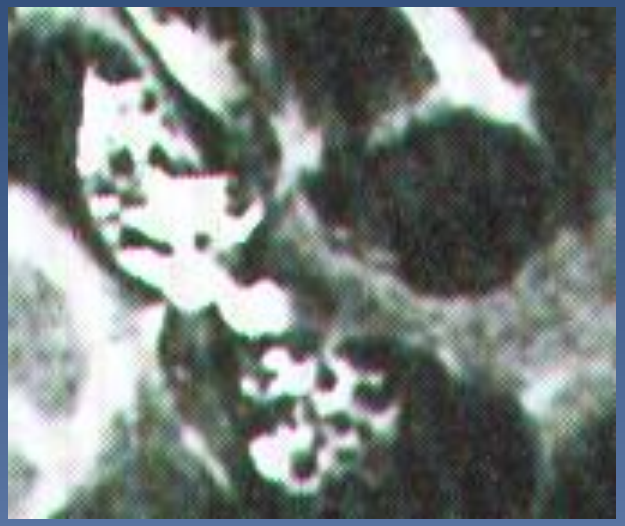


MEP= Ambient



Ambient-200 mmH20

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

Negative ME pressure

- An in vivo chinchilla study was conducted to test the effect of negative pressure
- Pressure levels for 1 hour
  - 0, -200, -400, -600 mmH2O
- No change in the histology of ME mucosa at 0 and -200mmH2O
- At -400mmH2O, there was edema, and some effusion
- At -600mmH2O, disruption of capillaries, and effusion filling the bullae

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

Bluestone CD. Eustachian Tube. Structure, Function, Role in Otitis Media. BC Decker Inc, Hamilton, 2005

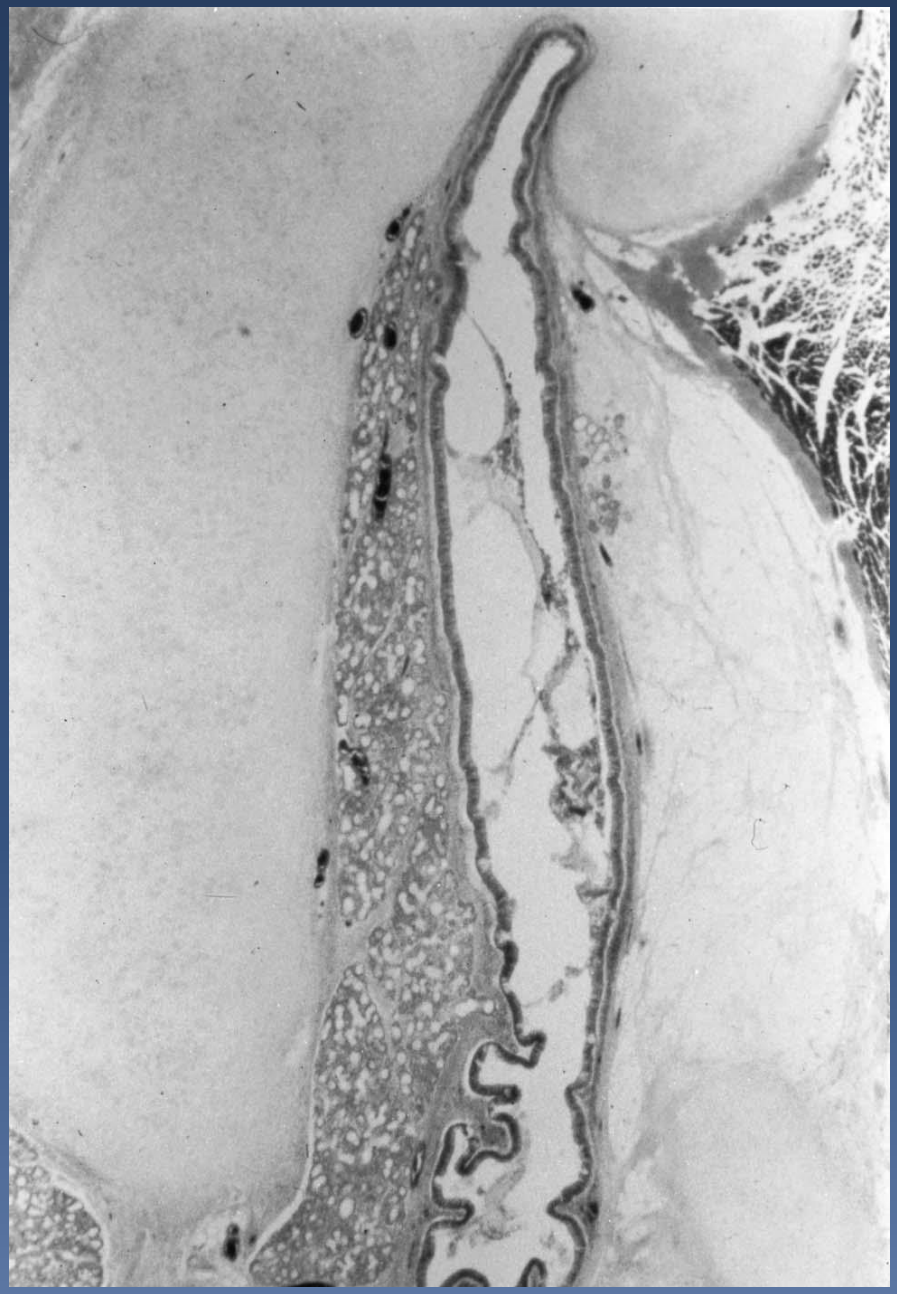
	Related to Eustachian Tube Dysfunction			
Risk Factor	*Yes*	*Maybe*	*No*	*Unknown*
Infant age	+			
Male gender				+
Genetic predisposition		+		
Sibling order	+			
Early onset of otitis media		+		
Child day care	+			
Season	+			
Lower socioeconomic status	+			
Smoking in household		+		
Allergies	+			
Use of pacifiers		+		
Prone position in infancy		+		
Wood-burning stoves		+		
Certain ethnic backgrounds	+			
Craniofacial malformations	+			
Immature/impaired immunology			+	
Lack of breast-feeding				+
Gastroesophageal reflux		+		

Developmental differences in ET-ME between infants and adults

Bluestone CD. Eustachian Tube. Structure, Function, Role in Otitis Media. BC Decker Inc, Hamilton, 2005

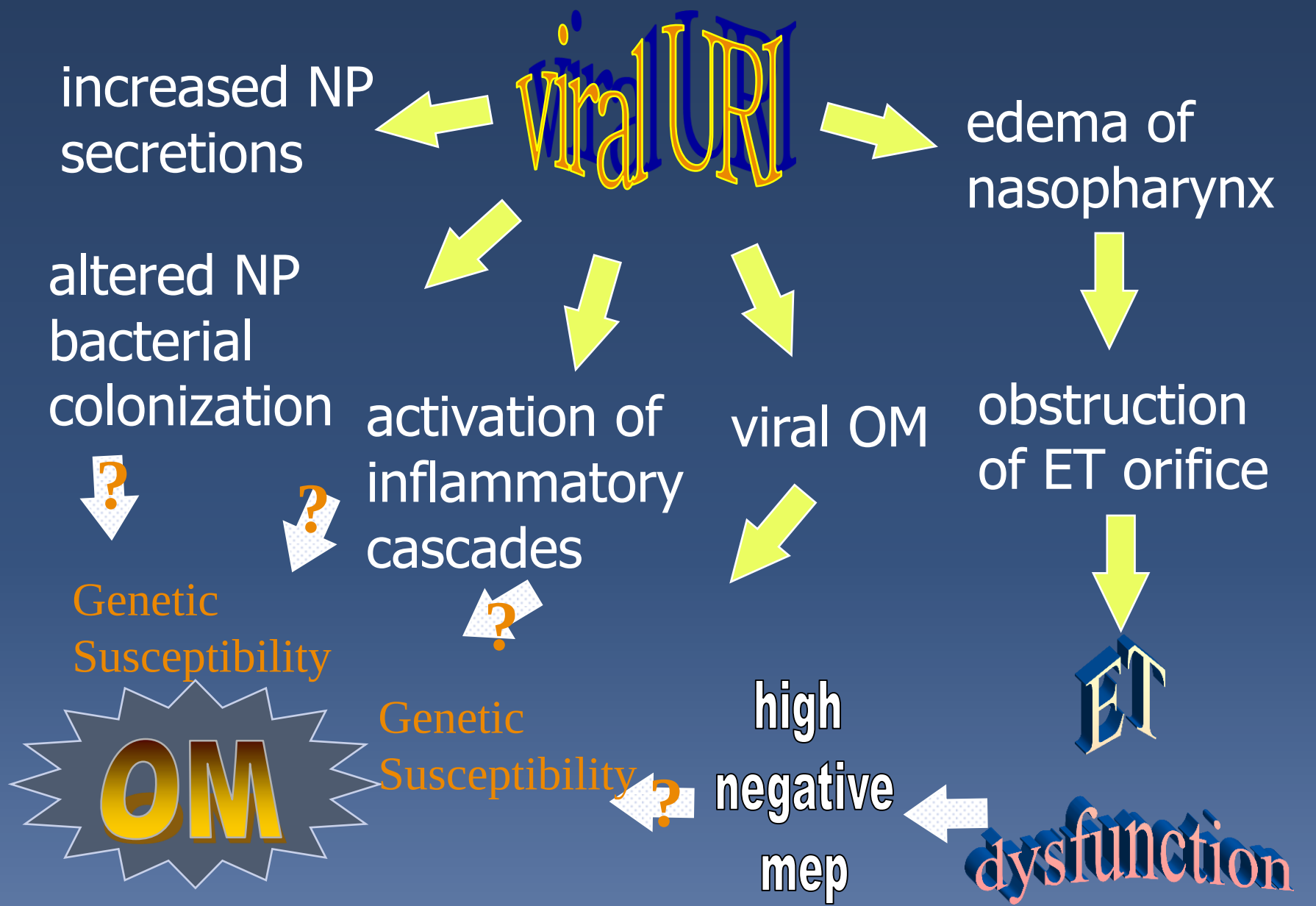
Anatomic Features of the Eustachian Tube	*Compared with the Adult, in the Infant It Is*	*Reference*
Length of tube	Shorter	Sadler-Kimes et al, 1989[6]; Ishijima et al, 2000[7]
Angle of tube to horizontal plane	10° vs 45°	Proctor, 1973[8]
Angle/length of TVP to cartilage	Variable vs stable angle, shorter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

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


viral URI
increased NP secretions
edema of nasopharynx
altered NP bacterial colonization
activation of inflammatory cascades
viral OM
obstruction of ET orifice
?
?
?
Genetic Susceptibility
Genetic Susceptibility
OM
high negative mep
?
dysfunction
ET

Outcome of viral URIs

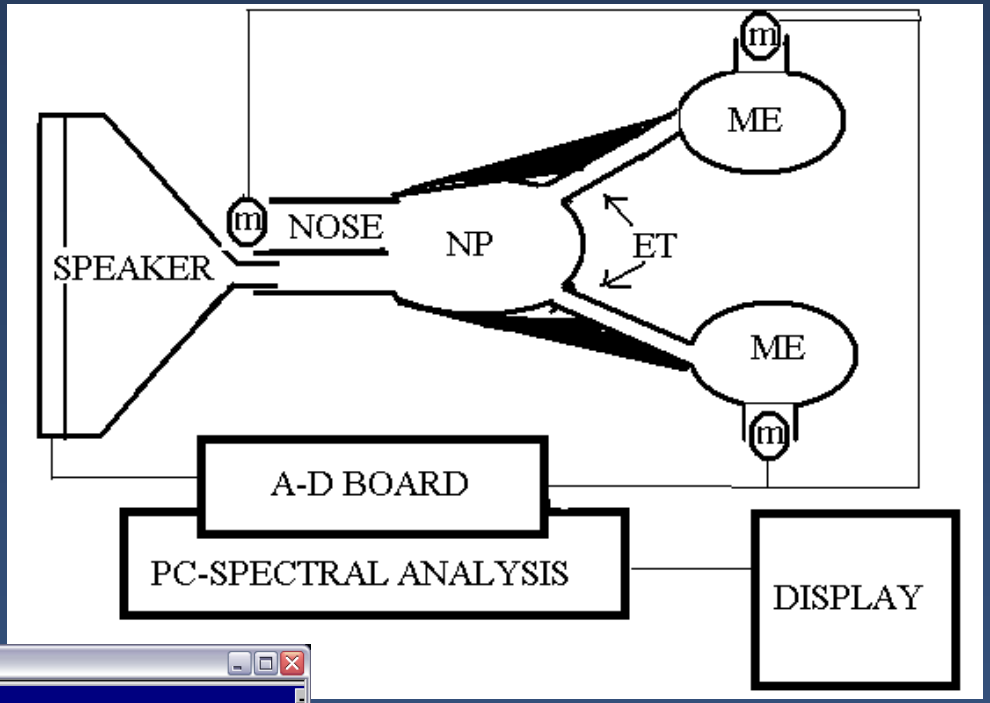
- Viral URI is most common cause of acquired/ intermittent ET dysfunction and negative ME pressure
- Most viral URIs lead to negative ME pressure (more in children)
- Negative MEP is from ET dysfunction
- Most OM is preceded by viral URI (50-70%)
- However, most viral URI does not lead to OME or AOM

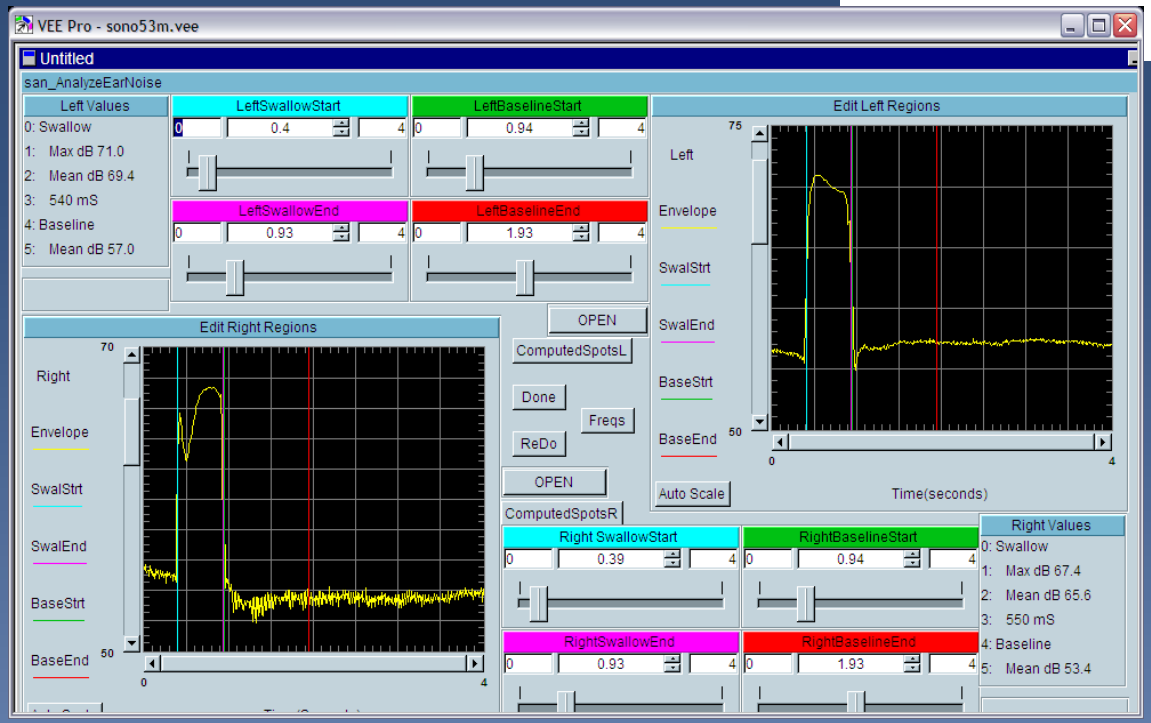
What are the test methods to assess the ETF?

- 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

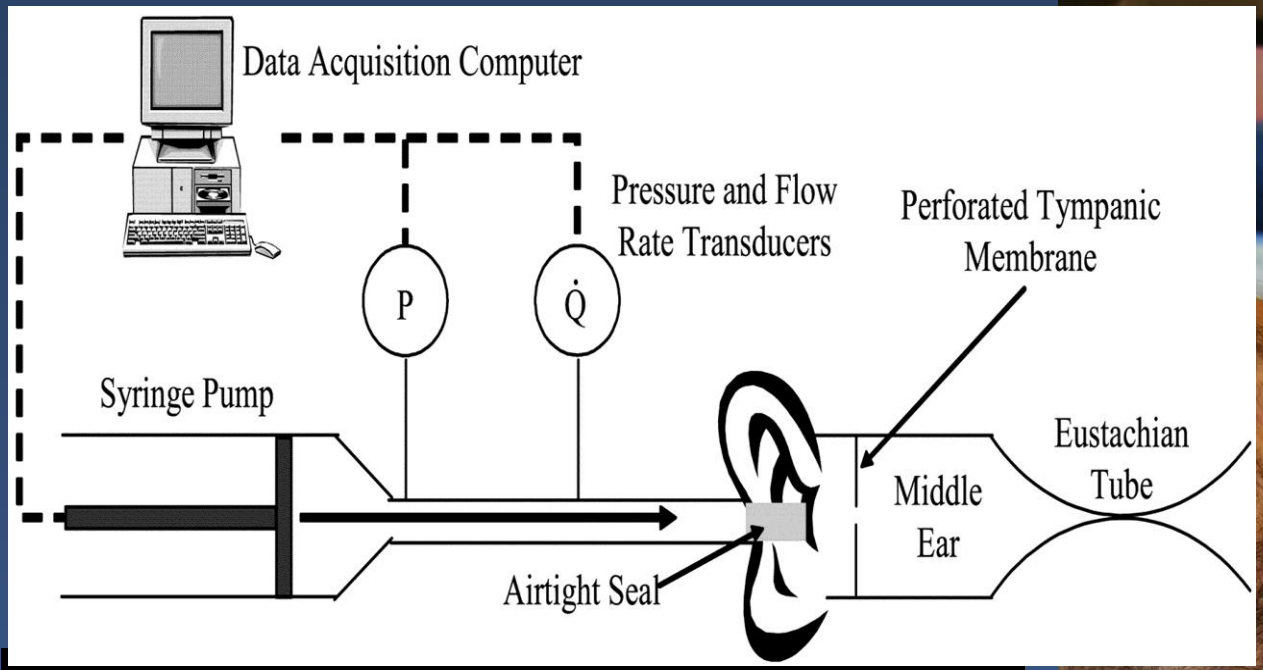
Pressure Chamber Testing

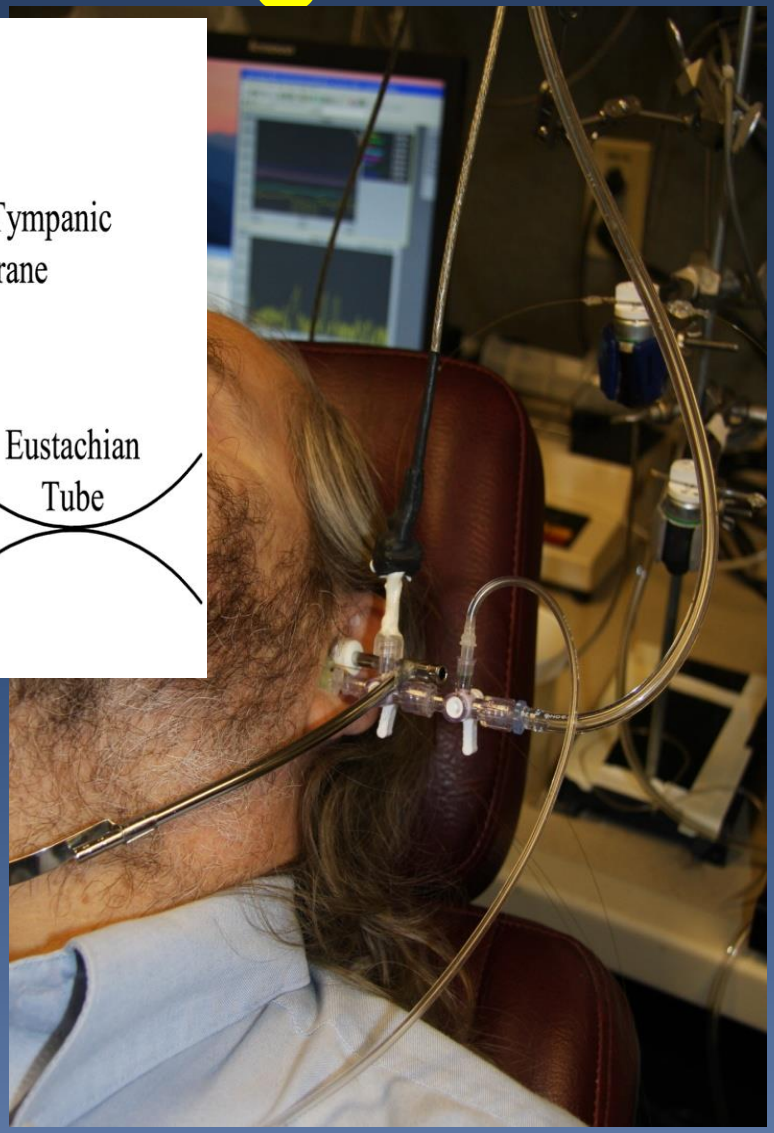
Sonotubometry Testing

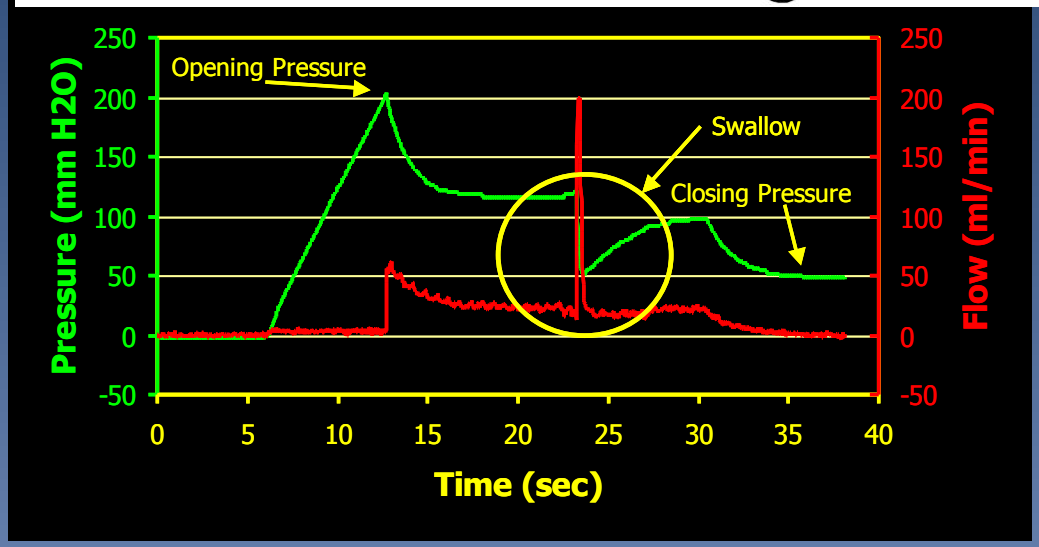




Forced Response Testing

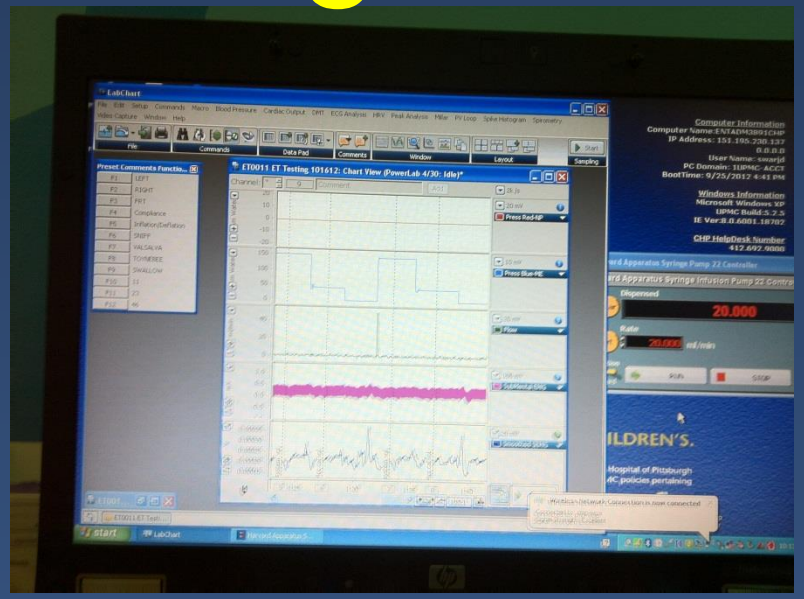




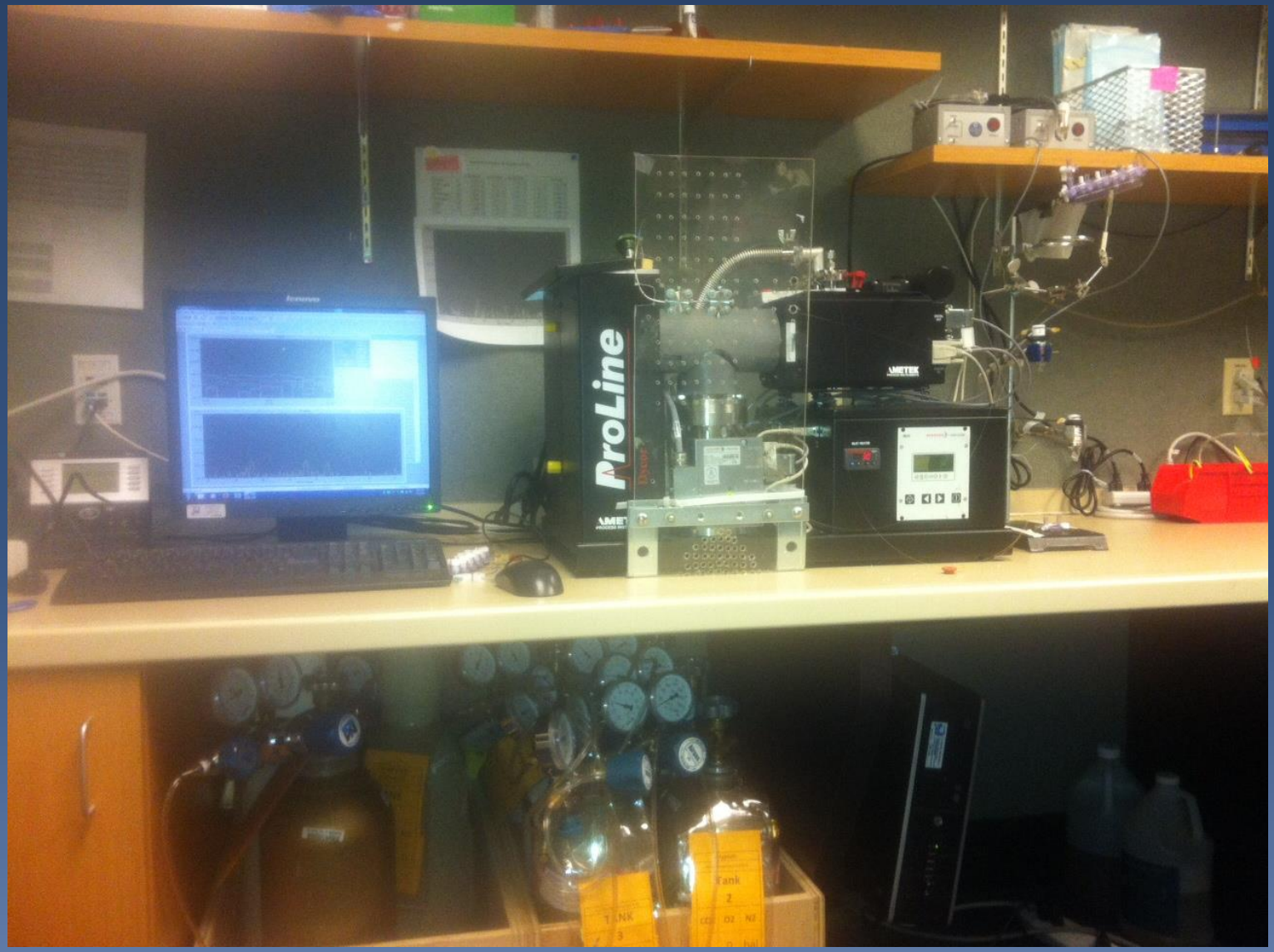


Inflation-Deflation Testing

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**