Middle Ear Disorders

Last updated: May 11, 2019

Findings indicating normal middle ear function:

1. Positive Rinne test
2. Normal TM in otoscopy
3. Normal tympanogram (A type) + positive stapedial reflex
4. Detectable OAE (otoacoustic emissions)

TYMPANIC MEMBRANE PERFORATION (TMP)

ETIOLOGY

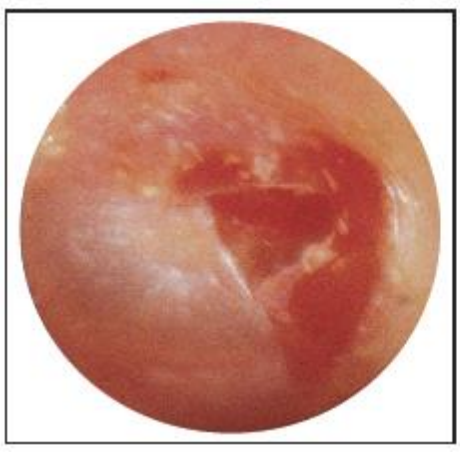
A. Infection of:
a) middle ear!!!
b) rarely - ear canal (usually *Aspergillus niger*)

B. Trauma:
a) foreign bodies
b) sudden overpressure (e.g. explosion, slap, diving accident)
c) sudden negative pressure (e.g. strong suction applied to ear canal)

C. Iatrogenic intentional **myringotomy** (± ventilating tube placement)

Possible additional lesions:
1) dislocations of ossicular chain, displacement of fragments of ossicles
2) fracture of stapedial footplate
3) perilymph fistula
4) facial nerve injury.

Fig. Traumatic rupture of the tympanic membrane



Fresh tympanic membrane rupture in the right ear. Otoscopy reveals a triangular perforation with hemorrhagic margins.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

SYMPTOMS & SIGNS

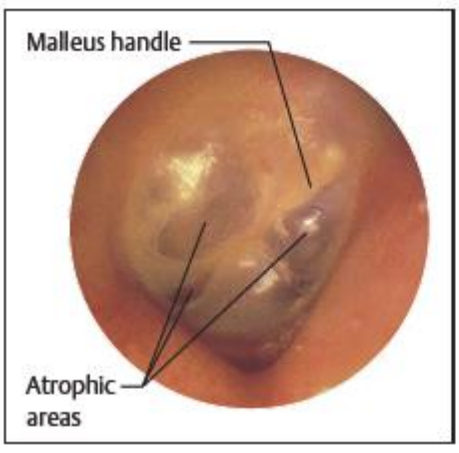
- sudden **severe pain** → **bleeding** from ear.
- CONDUCTIVE **hearing loss & tinnitus**.
- **vertigo** suggests injury to inner ear.
- *audible whistling sounds* during sneezing, nose blowing.
- perforation renders ear more susceptible to infection (esp. if water enters ear canal) - **purulent otorrhea** may begin in 24-48 h.
  N.B. *perforation is* absolute contraindication to irrigation for cerumen removal; history of perforation also is absolute contraindication (unless personal knowledge derived from prior examination indicates intact drum).
- perforations uncomplicated by infection are never painful.

Eardrum tends to heal itself (even eardrums that have been perforated multiple times often remain intact)!

- at times, perforation heals with thin neomembrane *without fibrous middle layer* (consisting only of mucosal and squamous epithelial layers):
  – may be so thin that it *can be mistaken for perforation* instead of healed perforation.

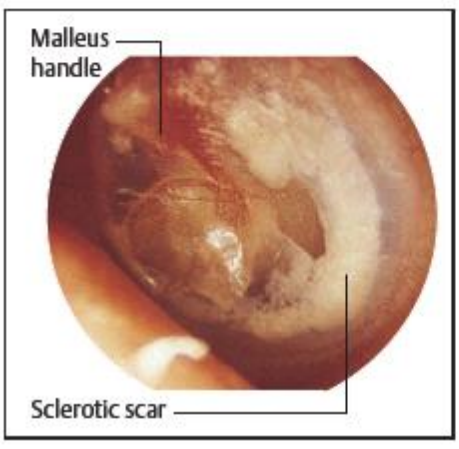
  - *may retract deeply* into middle ear (may presage formation of cholesteatoma).
- if squamous epithelium covers perforation edge (leaves no raw surface) → perforation persistence.

Fig. Atrophic scars of the tympanic membrane



Right tympanic membrane is heavily scarred but intact. Most of the scarring is atrophic.

Fig. Sclerotic scar of the tympanic membrane



Left tympanic membrane has a sclerotic scar in its posteroinferior quadrant. Atrophic areas are also evident.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

DIAGNOSIS

1. Most are diagnosed with **routine OTOSCOPY**.
2. Small perforations may require **OTOMICROSCOPY**.
3. **TYMPANOMETRY** may reveal abnormalities consistent with perforation, but confirmation still requires examination.
4. In rare cases, *if TMP diagnosis remains questionable* → **fill ear canal** with sufficient distilled water to cover tympanic membrane → have patient perform **Valsalva maneuver → stream of bubbles** is caused only by TMP; negative test result is suggestive but not definitive.
5. Always perform **AUDIOMETRY** - upon initial TMP diagnosis and again before any repair attempt.
   - ***mild (!!!) conductive hearing loss*** is consistent with perforation.
   - audiometry often reveals normal hearing.
   - conductive component of ≥ 30 dB indicates *ossicular discontinuity*.

TREATMENT

Many perforations can be monitored without need for medical treatment.

- ear is kept dry; *aseptic technique* when examining ear.
- displaced flaps of tympanic membrane are ***replaced in their original positions*** (using local anesthetics and microscopic control) to facilitate healing.
- if infection is present / likely to occur → oral PENICILLIN V for 7 days.
- **topical 2%** ACETIC ACID (5 drops tid) - if ear becomes infected (no eardrops should be used prophylactically!) - lowers pH (environment unfavorable to microbial growth).
- *spontaneous perforation closure is usual*; if it does not occur within 2 mo → **REPAIR OF TYMPANIC MEMBRANE**;
  - if CONDUCTIVE hearing loss persists (discontinuity of ossicular chain), middle ear should be explored surgically and repaired.
  - if SENSORINEURAL hearing loss or VERTIGO persist for hours - inner ear concussion but may also indicate inner ear penetration → prompt exploratory tympanotomy to repair damage as soon as possible.

Office treatments (esp. if perforation is small and involves neither umbo nor annulus):

a) ***cauterize edges*** of TMP with caustic (e.g. 10% trichloroacetic acid), and then apply small patch of ***cigarette paper***; mechanical stripping of perforation margin before applying patch slightly increases success rate.

b) ***fat-plug tympanoplasty***: obtain small plug of fat from postauricular sulcus; anesthetize perforation margins with phenol solution; mechanically débride edges with microcup forceps; fat is tucked into perforation, extending both into canal and into middle ear space.

c) ***irritant oil method*** (very successful office treatment, which requires 6-10 weekly office visits): freshen perforation by stripping margin using microcup forceps (if necessary, use small amount of phenol solution for anesthesia); apply ***cotton ball*** (that is 1-2 mm larger than perforation diameter) to TMP; patient instills solution of irritative and aromatic oils daily into ear; change cotton weekly (repeat edge freshening if no progress is seen).

TYMPANOPLASTY (successful in > 90% cases)

- approach can be *permeatal*, *endaural*, or *postaural*.
- incision may be made behind ear or entirely through ear canal.
- most commonly used grafting material is ***postauricular fascia***; others - temporalis fascia, tragal perichondrium, vein graft.
- graft may be placed either medially (underlay) or laterally (overlay) to perforation.

FOLLOW-UP

Several annual visits should be minimum once healing is verified:

1. Risk of *CHOLESTEATOMA* formation
2. Risk of *REPERFORATION* (≈ 10%)

- perforations in ***pars tensa*** rarely lead to complications (exception - perforations located at annulus - risk of cholesteatoma from migration of surface epithelium into middle ear).
- perforations in ***pars flaccida*** are more frequently associated with complications.

INFECTIOUS (s. BULLOUS) MYRINGITIS

- viral, bacterial or fungal.
- primary causes include ***otitis externa***, ***trauma***, and, on rare occasion, ***foreign bodies***.
- clinical features:
  - sudden severe **pain**; persists for 24-48 h.
  - **vesicles on tympanic membrane**: viral, acute bacterial (esp. *Streptococcus pneumoniae*), mycoplasmal infection.
  - **hearing loss, sensation of congestion** and **fever** suggest bacterial otitis media.
  - no middle ear effusion!!!
- diagnosis - PNEUMATIC OTOSCOPY;
  - TM becomes red and thickened, light reflex disappears;
  - bacterial inflammation in middle ear results in bulging of TM with possible perforation.

TREATMENT

- **antibiotic therapy** (as for acute otitis media).
- pain may be relieved:
  a) rupturing vesicles with myringotomy knife
  b) narcotic.
- myringotomy, tympanostomy may be necessary to drain middle ear cavity.

Patients with *recurrent myringitis* are taught to use **acidifying drops** (e.g. acetic acid) after every exposure to water.

ACUTE OTITIS MEDIA

- classified as **subacute** if persists > 3 weeks; **chronic** if > 3 months.

PATHOPHYSIOLOGY

- **EUSTACHIAN TUBE DYSFUNCTION**:

A. Most commonly: viral URI involving nasopharynx → obstruction of eustachian tube → negative pressure inside middle ear cavity → transudation.
- middle ear is extension of upper respiratory tract and is lined by ciliated epithelium.
- microorganisms migrate from nasopharynx by:
  a) moving over surface of eustachian tube's mucous membrane
  b) propagating in lamina propria of mucous membrane (as spreading cellulitis or thrombophlebitis).
- allergy may also play important role in obstruction.

B. In minority (e.g. children *with neuromuscular disorders),* eustachian tube is hypotonic - predisposed to reflux of nasopharyngeal contents into middle ear.

ETIOLOGY

- to become pathogenic bacteria must adhere to mucosal lining; **viral** infections that damage mucosal linings of *respiratory tracts facilitate ability of bacteria to become pathogenic.*
- most common **bacteria**:
  NEWBORNS - *Str. pneumoniae*, Gr-negative enteric bacilli (particularly *E. coli*), *Staph. aureus*.
  AFTER NEONATAL PERIOD, E. coli becomes rare; 4 main pathogens:
  Str. pneumoniae (30-40%)
  H. influenzae (20%)
  Moraxella catarrhalis (10%)
  Str. pyogenes
  – when otitis becomes **chronic**, *Pseudomonas* species predominate.

Predisposing factors:
1) second-hand smoking
2) daycare attendance (greater risk factor than parental smoking!!!)
3) bottle feeding in supine position (→ eustachian reflux)
4) Down syndrome (decreased function of immunoglobulins A, G2, G4)
5) palate disorders (e.g. cleft palate) – affected eustachian opening

CLINICAL FEATURES

- Native Americans and Inuit > whites > African Americans.
- ***any age*** (most common **children 3 mo ÷ 3 yr**; after *eruption of permanent teeth*, incidence drops dramatically).
  – in USA, it is most common affliction necessitating medical therapy for children < 5 years!!!
  – 70% of all children experience ≥ 1 attack before their 2nd birthday!
  – some infants experience 1st attack shortly after birth and are considered ***OTITIS PRONE*** (i.e. at risk for recurrences).

1. Persistent severe **EARACHE** (infant ear pulling) - first complaint.
2. Conductive **HEARING LOSS**.
3. **SYSTEMIC SIGNS** (fever, nausea, vomiting, diarrhea) may occur in young children.
4. **Spontaneous perforation** → **OTORRHEA** (bloody → serosanguineous → purulent);
   – with perforation patient experiences ***rapid relief of pain and fever***.
   – *pulsation of otorrhea* is common.
   – otorrhea lasts 1-2 days before spontaneous healing occurs; otorrhea may persist if perforation is accompanied by mucosal swelling or polypoid changes, which can act as ball valve.

COMPLICATIONS:
1) postauricular abscess
2) acute mastoiditis, petrositis (Gradenigo syndrome), labyrinthitis
3) **FACIAL PARALYSIS**
   - in setting of ACUTE otitis media (within 7-10 days), facial nerve weakness is due to ***edema of nerve*** within bony canal - recovery can be expected with conservative treatment of acute otitis media.
   - in case of CHRONIC otitis media, paralysis likely is secondary to ***erosion of osseous facial canal*** → immediate surgical intervention:
     a) *incomplete paralysis* - simple mastoidectomy without exposure of nerve (incision of perineurium is contraindicated - facilitates spread of infection)
     b) *complete paralysis* - decompression of nerve.
4) **MENINGITIS** (most common intracranial complication!!!), epidural abscess, subdural empyema, brain abscess, sigmoid sinus thrombosis, otitic hydrocephalus.
5) bacteremia, bacterial endocarditis.

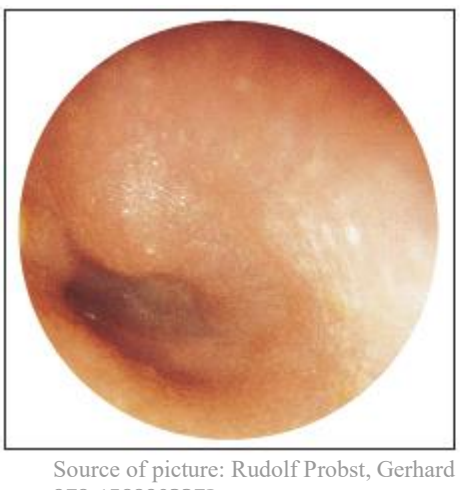
DIAGNOSIS

Diagnosis is usually made clinically!

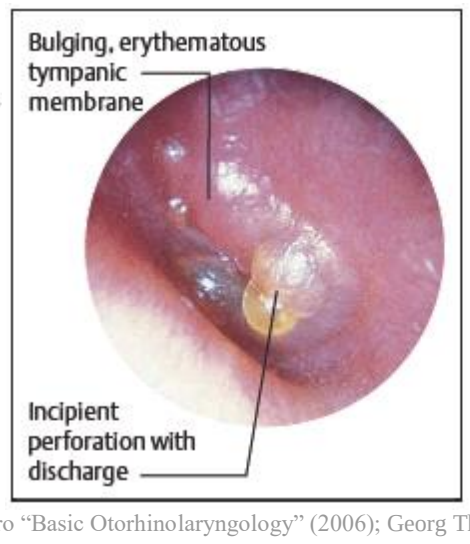
Pneumatic Otoscopy (standard of diagnosis) – erythematous tympanic membrane may bulge; indistinct landmarks, displaced light reflex, poor tympanic mobility!!!, effusion!!!

Erythema can result from crying or fever and, by itself, does not establish diagnosis!

Tympanometry is objective diagnostic documentation!



Bulging, thickened, undifferentiated left tympanic membrane in acute otitis media.



Otoscopic findings in acute otitis media. The left tympanic membrane is bulging and erythematous. Incipient perforation with discharge is evident in the posteroinferior quadrant.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

Culture exudate via tympanocentesis;
N.B. *nasopharyngeal* cultures, cultures from *spontaneous perforation* or *tympanostomy tube* do not correlate well with causative agent and should be discouraged!
– indications for TYMPANOCENTESIS:
  1) immunosuppressed children
  2) neonates
  3) antimicrobial treatment failure
  4) complications of AOM

- perform tympanocentesis without anesthesia, after sterilization of ear canal with isopropyl alcohol or povidone-iodine solution;
- insert needle through anterior portion of tympanic membrane;
- aspirate contents into sterile trap for identification of microbes;
- hole is small enough to permit healing within day or two.

CT / MRI may be necessary to determine if ***complication*** has occurred; otherwise, imaging studies are unnecessary.

- **hearing**, **tympanometry** should be monitored until resolution is complete.

TREATMENT

Self-limiting disease (provided patient does not develop complication – 80% recover without treatment) - chorus of advocates recommends withholding antibiotic therapy (esp. in Europe)!

ANTIBIOTIC THERAPY for 10-14 days is generally indicated in USA*:

- ***first line agents***: AMOXICILLIN 40 mg/kg/d q8h 10 days [drug of choice], TMP-SMX, macrolides (e.g. ERYTHROMYCIN-SULFISOXAZOLE, AZITHROMYCIN, CLARITHROMYCIN).
- ***second line agents*** (high-dose AMOXICILLIN, AMOXICILLIN-CLAVULANATE, CEFPROZIL, CEFUROXIME, CEFPODOXIME, CEFDINIR) - used if ***first-line agents fail*** or when there is ***risk for penicillin-resistant* S. pneumoniae / *β-lactamase–positive* H. influenzae / M. catarrhalis *infection***:
  a) therapy within last month with erythromycin-sulfisoxazole, TMP-SMX, azithromycin, or ampicillin.
  b) patients < 2 years
  c) attend daycare center
  d) contact with individuals treated with antibiotics.
- ***third line agents***: CEFTRIAXONE, LEVOFLOXACIN, CIPROFLOXACIN.

*concerns about *antibiotic-resistant germs* in USA led to recommendation to *withhold a/b treatment in most cases* (**painkillers** must be enough for most children!)

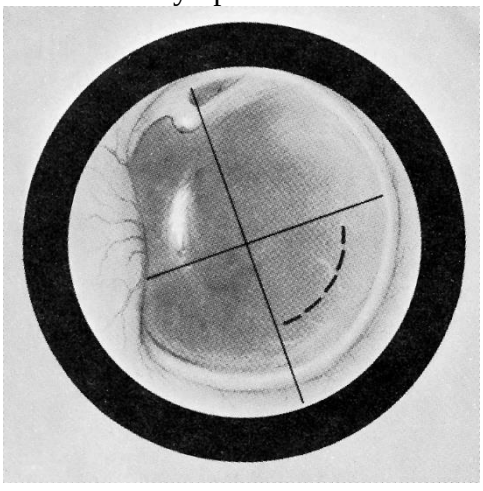
For improving eustachian tube function (should not be used – studies indicate that such drugs may even prolong illness!!!):

a) **topical vasoconstrictors** (e.g. PHENYLEPHRINE 0.25% 3 drops q 3 h into each nasal cavity while patient is supine with neck extended).
b) **systemic sympathomimetics** (e.g. EPHEDRINE SULFATE, PSEUDOEPHEDRINE, PHENYLPROPANOLAMINE); not recommended for children.

MYRINGOTOMY indications:

a) **bulging** tympanic membrane with **intractable pain**.
b) **severe or persistent** pain, fever, vomiting, diarrhea.
c) **neonates** (bacteremia is common in all neonates!!!).

- done by enlarging tympanocentesis hole with microalligator forceps or suction tip.
- TM can safely be incised in all quadrants *except posterior superior quadrant* (where incus and stapes lie; facial nerve and round window are generally protected from anyone but clumsiest of surgeons).
- instill antibiotic drops → suction middle ear.
- patient experiences prompt relief of local symptoms.



Indications for TYMPANOSTOMY TUBE insertion:

1) suppurative complication of temporal bone (e.g. mastoiditis)
2) history of repetitive attacks (tube design permits tube placement for longer than 2 years!).

Prevention of recurrences:

1) conjugated heptavalent ***pneumococcal vaccine***
2) SULFISOXAZOLE or AMOXICILLIN daily for 3-8 months (indicated if 3 episodes in past 6 months, or 4 episodes in past 12 months have occurred).
3) TYMPANOSTOMY TUBE.

N.B. normally, 50% children have ***persistent effusion*** 2-3 weeks after therapy!

ACUTE MASTOIDITIS

*- bacterial infection in mastoid process (osteitis, periosteitis) resulting in **coalescence of mastoid air cells** ± **destruction of mastoid process cortex***

- disease of very young.
- results from progression (complication) of **acute purulent otitis media**.
  CHRONIC MASTOIDITIS usually is due to **cholesteatoma**.
  – if acute otitis persists beyond 2 weeks, there is associated thickening of mucoperiosteum in air cells around periantral area → blockage of antral drainage;
  – trapped secretions in mastoid air cell system cause intense pressure, venous stasis, and local acidosis → dissolution of bone calcium → coalescence of mastoid air cell system (coalescent mastoiditis)..
- responsible bacteria are same as in acute otitis media;
  – STREPTOCOCCAL mastoiditis is preceded by *early perforation* and profuse otorrhea.
  – PNEUMOCOCCAL mastoiditis is less symptomatic but just as destructive; *advanced coalescence of mastoid air cells* may precede perforation of tympanic membrane.

CLINICAL FEATURES

- exacerbation of aural pain (persistent and throbbing), fever, otorrhea (creamy, profuse, lasting > 3 weeks).

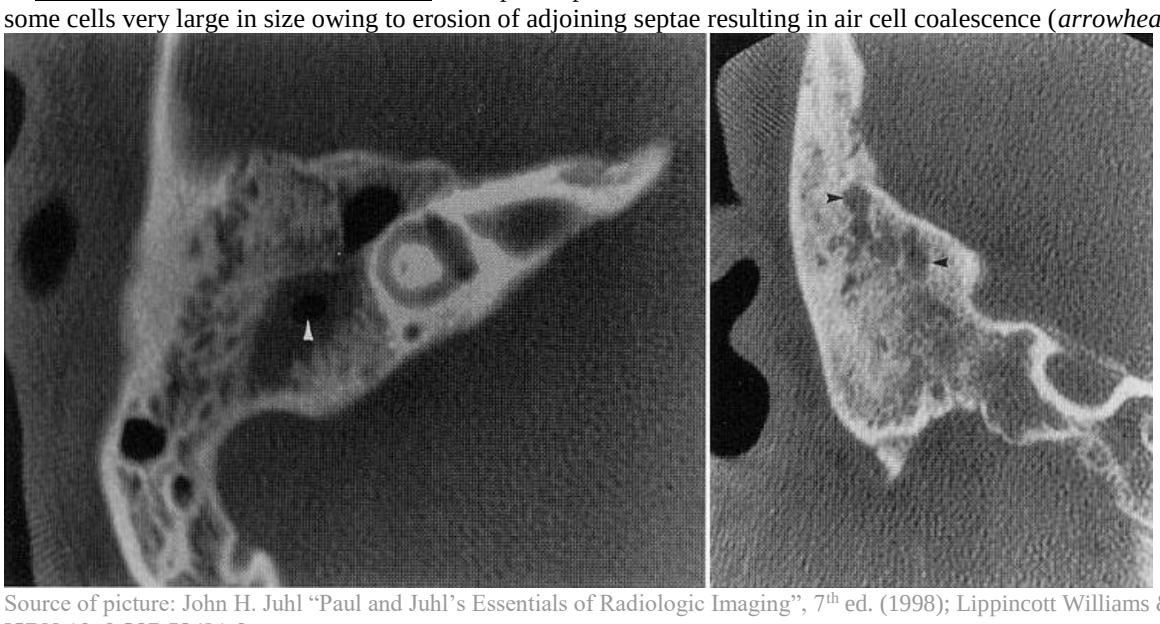
DIAGNOSIS

CT scans:

acute otitis media - mastoid air cells filled with purulent fluid (soft tissue density);
coalescent mastoiditis (conversion from mucosal disease to bone disease) - cell partitions become indistinct, eventually leading to bony sclerosis; treatment with antibiotics will not restore mastoid air cells to normalcy.

Plain radiographs are not reliable!

A. Axial CT of acute mastoiditis - mastoid air cells opacified with fluid and hyperemic mucosa; small fluid level in maxillary antrum (*arrowhead*); mastoid septae well mineralized, and air cells of normal size.
B. Coronal CT of coalescent mastoiditis - complete opacification of all mastoid air cells, with areas of mastoid sclerosis; some cells very large in size owing to erosion of adjoining septae resulting in air cell coalescence (*arrowheads*).



Source of picture: John H. Juhl "Paul and Juhl's Essentials of Radiologic Imaging", 7th ed. (1998); Lippincott Williams & Wilkins; ISBN-10: 0-397-58421-0 >>

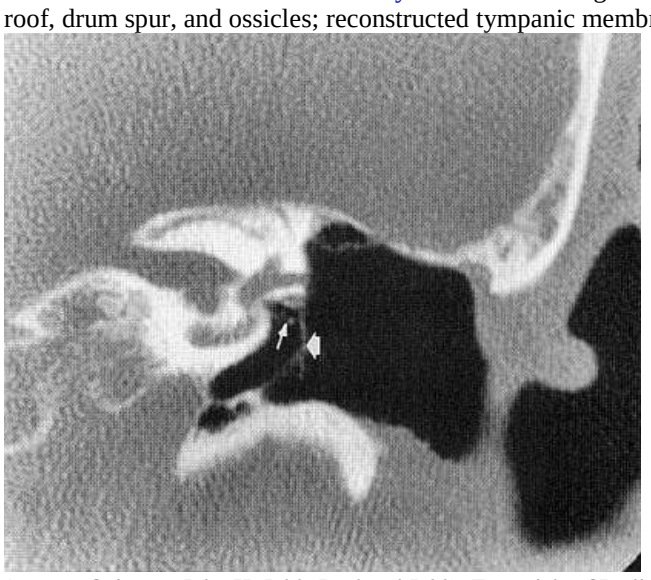
TREATMENT

- *simple mastoiditis:* **antibiotic therapy** for > 2 wk (i/v AMPICILLIN or CEFUROXIME) + **tympanostomy tube**.
- *coalescent mastoiditis, osteitis, subperiosteal abscess, meningitis* require complete exenteration of mastoid air cells (MASTOIDECTOMY).
  - antibiotics are started without any delay!
  - *TYMPANOSTOMY TUBE* is usually placed during mastoidectomy (acts as drain).
- if open mastoid surgery is not undertaken, use of **single, high-dose intravenous STEROIDS** is warranted - to decrease mucosal swelling → to promote natural drainage through aditus ad antrum into middle ear.

MASTOIDECTOMY

a) **radical mastoidectomy** - extensive removal of outer mastoid cortex, all mastoid air cells, posterior-superior external canal wall, and majority of ossicles.
b) **modified radical mastoidectomy** - leaves ossicles mostly intact.
c) **intact-canal-wall mastoidectomy** - leaves ossicles and external canal intact.

Coronal CT of radical mastoidectomy - evidence of surgical resection of mastoid air cells, mastoid antrum, external canal roof, drum spur, and ossicles; reconstructed tympanic membrane (*large arrow*) can be seen, as can stapes (*small arrow*):



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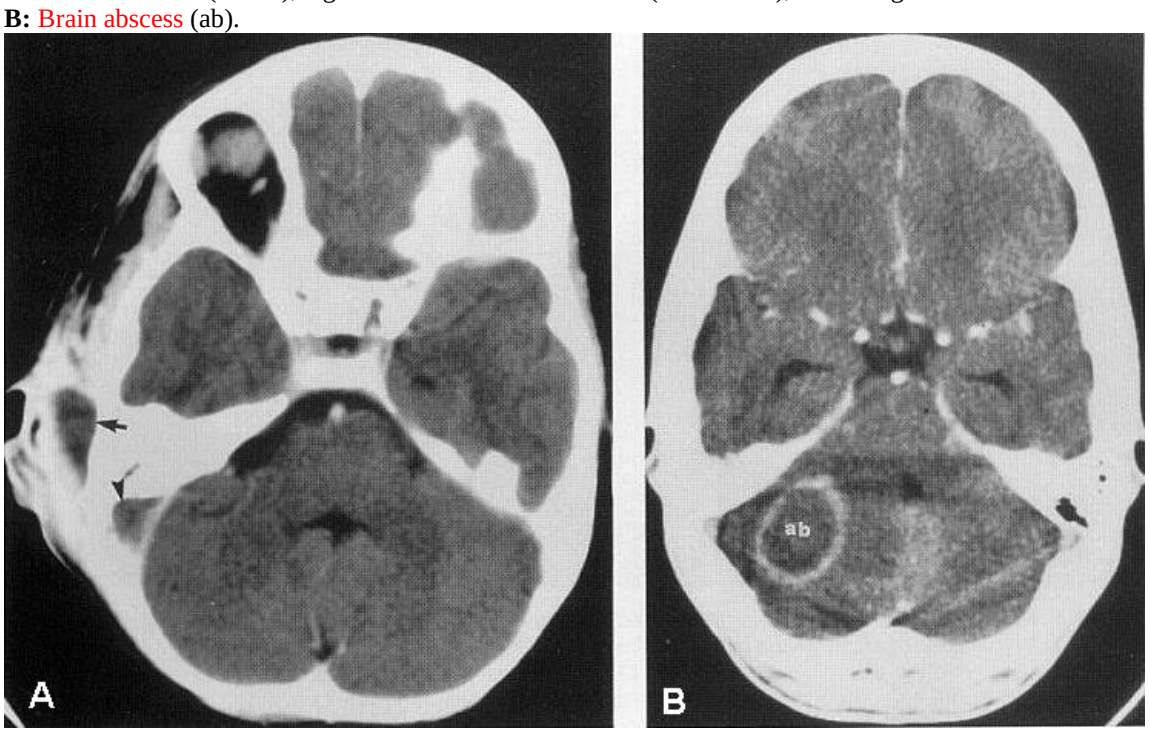
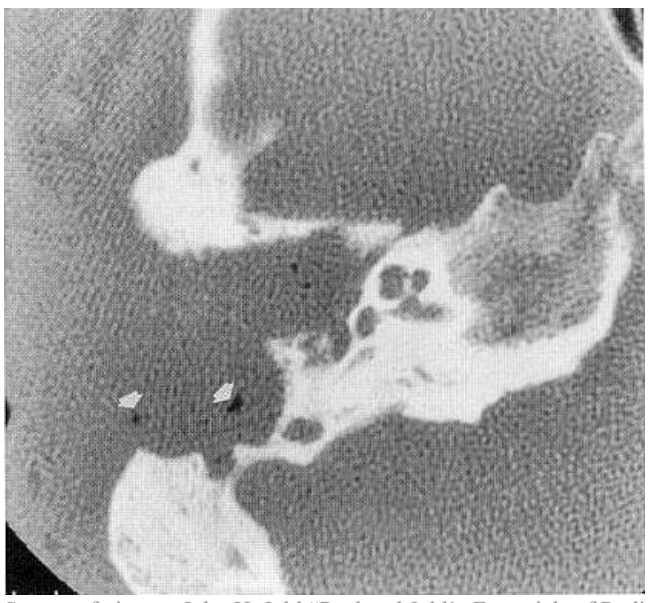
COMPLICATIONS

- extension into *petrous air cells* → *ACUTE PETROSITIS* → *GRADENIGO SYNDROME* (apical petrositis).
  - in cranial nerve palsies, **IV steroids** speed recovery.
- break through outer mastoid cortex into *subperiosteal space* → *POSTAURICULAR SUBPERIOSTEAL (BEZOLD) ABSCESS* - postauricular redness, swelling, tenderness, fluctuation over mastoid process; pinna displaced laterally and inferiorly (ear "sticks out"); postauricular skin crease obliterated*.
  *if crease remains, process is lateral to periosteum.
- extension through *inner mastoid cortex* → *SIGMOID SINUS THROMBOPHLEBITIS*.
- extension into *brain* → middle / posterior fossa *BRAIN ABSCESS*.
- occasionally mastoid becomes infected with anaerobic organisms (gas bubbles in area of infection) → walls are slowly eroded → inflammatory debris slowly evacuates → large air-filled *automastoidectomy cavity* (resembles surgical mastoidectomy).

Axial CT:
A: Bezold abscess (*arrow*); sigmoid sinus does not enhance (*arrowhead*), indicating thrombosis due to thrombophlebitis.
B: Brain abscess (ab).



Axial CT - chronic *Bacteroides fragilis* otomastoiditis with automastoidectomy - entire mastoid and middle ear cavity destroyed; note small air bubbles within mass (*arrows*):



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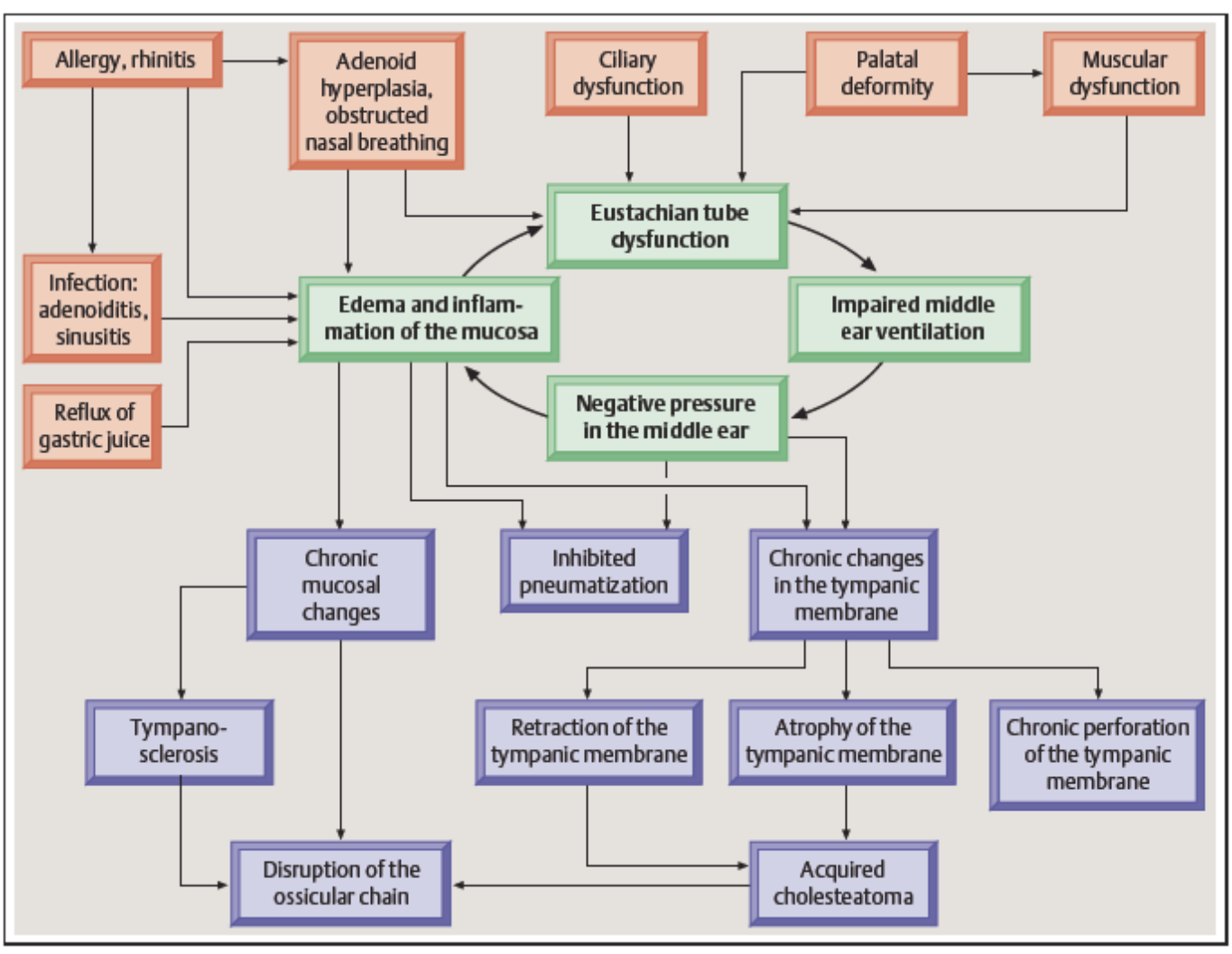
CHRONIC (s. SEROUS) OTITIS MEDIA

- NONPURULENT EFFUSION IN MIDDLE EAR resulting from:
  a) **incomplete resolution of acute otitis media** (it is expected - does not require re-treatment with antimicrobials!).
  b) **obstruction of eustachian tube** (e.g. adenoids, allergies)
     – normally, middle ear is ventilated 3-4 times/min (as tube opens during swallowing);
     – O_2 is absorbed by blood vessels of middle ear mucous membrane.
     – if eustachian tube is blocked, relative negative pressure develops.

- very common in *children* (esp. at late infancy); prevention - avoid recumbent milk feeding (e.g. bottle in bed) and exposure to cigarette smoke.
  in *adults*, cause is **nasopharyngeal mass** until definitively proven otherwise! (at minimum, indirect mirror examination or flexible nasopharyngoscopy should be performed)
- effusion may be sterile but usually ***contains pathogenic bacteria*** (same as in acute otitis media).



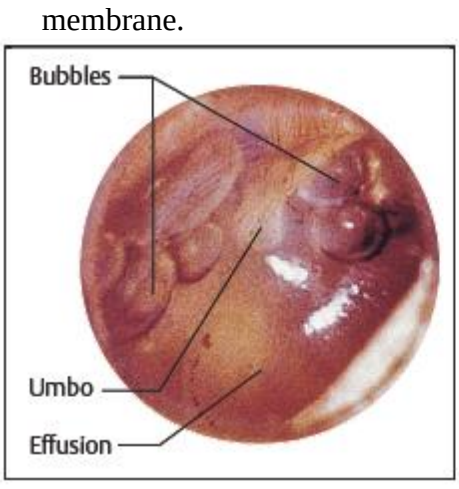
Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

CLINICAL FEATURES

1. Conductive **HEARING LOSS** ("glue ear")
2. Sensation of **AURAL FULLNESS**
3. No signs of acute otitis media (i.e. no signs / symptoms of acute local or systemic illness – no pain, no fever, etc.).

DIAGNOSIS

Otoscopy:
- tympanic membrane retracts (displacement of light reflex and accentuation of landmarks).
- effusion develops → amber / gray appearance of tympanic membrane with radial vessels on it, immobility of tympanic membrane, air-fluid level or air bubbles seen through tympanic membrane.



Serous effusion behind an uninflamed tympanic membrane, which is slightly retracted. Air bubbles are clearly visible.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

Tympanometry - *maximal compliance with negative pressures* in ear canal.
- particularly useful in small children (whose external auditory canals may be too small or too collapsible to permit adequate visualization of tympanic membrane);
- in those < 7 months of age, tympanometry is unreliable (excessive compliance of external auditory canal).

Audiometry indications:
1) if effusion persists for ≥ 3 months
2) language delay, learning problems
3) suspected significant hearing loss.

TREATMENT

a) trial of **antibiotic therapy** (as for acute otitis media).

b) *antimicrobials are not indicated* for initial treatment; indicated only if effusions persist for > 3-4 months.

Opening of eustachian tube, middle ear ventilation:

1) correct **underlying condition** in nasopharynx (e.g. adenoidectomy).
2) **systemic sympathomimetics** (e.g. ephedrine sulfate, pseudoephedrine, phenylpropanolamine).
3) **antihistamines** relieve allergic eustachian tube obstruction.
4) ***politzerization*** (inflation of eustachian tube and tympanum by forcing air into nasal cavity when patient swallows).

N.B. *antimicrobials are only medications* that have shown to increase rate of effusion clearance in randomized controlled trials!!!

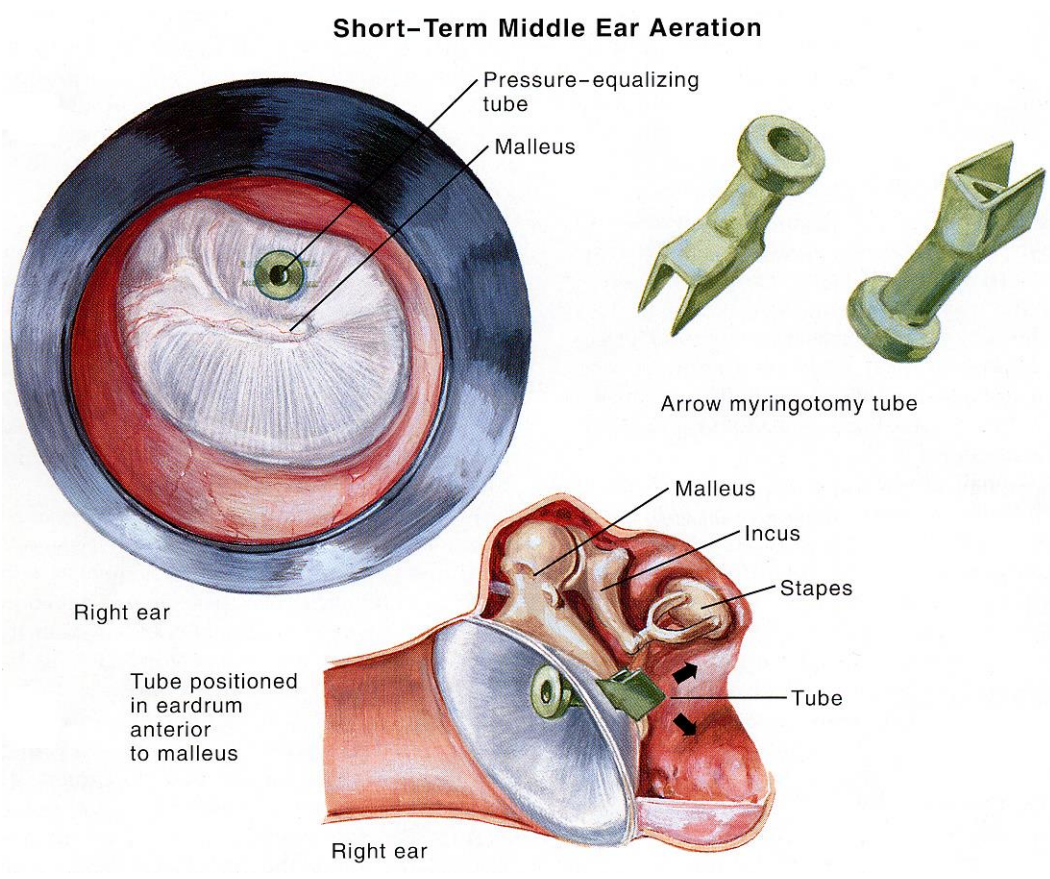
SURGERY indications:

a) effusion persists > 3-4 months;
b) severe (> 20 dB) conductive hearing loss;
c) risk for speech development delays.

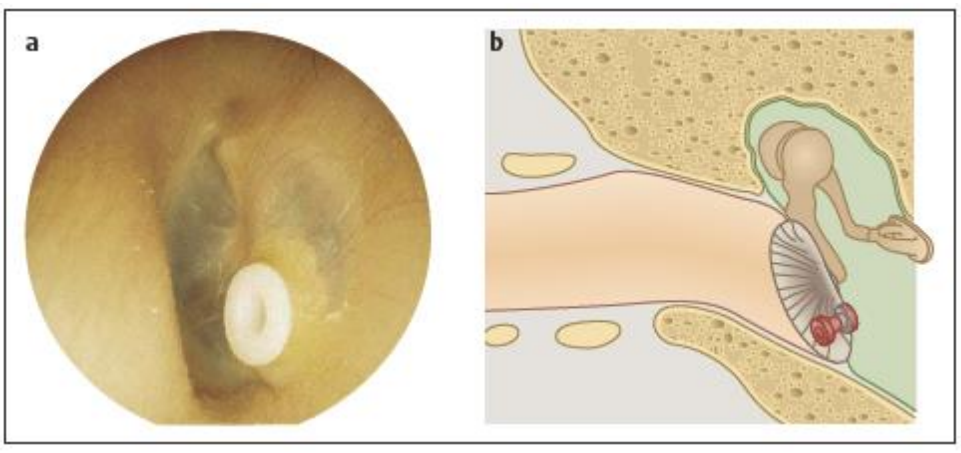
- most cases spontaneously resolve in spring and summer - conservative approach is often warranted at these times of year.

MYRINGOTOMY → fluid aspiration → insertion of TYMPANOSTOMY TUBE!!!

- tubes self-extrude 9-12 months after placement.
- in adults, aspiration may be enough.
- ***complications of tube***:
  1) persistent otorrhea (most common complication - occurring in 15%)
  2) tympanosclerosis
  3) persistent perforation
  4) granulation tissue formation, cholesteatoma, sensorineural hearing loss.



Source of picture: Frank H. Netter "Clinical Symposia"; Ciba Pharmaceutical Company; Saunders >>



Placed in the anteroinferior quadrant of the tympanic membrane, the myringotomy tube provides ventilation of the tympanic cavity through the ear canal.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

ADENOIDECTOMY

- *adenoidectomy* was once principal treatment (*tube placement* is now favored).
- as effective as tube placement.
- rationales:
  1) large adenoids occlude nasopharynx and choanae → excessive nasopharyngeal pressure during swallowing → eustachian tube **reflux**.
  2) extremely large adenoids may physically **occlude** eustachian tube orifice.
  3) adenoidectomy is removal of potential **source of inflammation and infection** at eustachian tube orifice.
- consider adenoidectomy in patients requiring second set of ventilation tubes.
- contraindication - submucosal cleft palate (mucosa intact but absent muscles in medial portion of soft palate; associated with notching of posterior hard palate and bifid uvula) – avoid adenoidectomy in such patients → regurgitation into nasal passages will surely occur!
- tonsillectomy has no effect!

CHRONIC SUPPURATIVE OTITIS MEDIA

- ***permanent perforation*** *of tympanic membrane +* ***chronic otorrhea*** *± permanent changes in middle ear.*

differentiate from CHRONIC SEROUS OTITIS MEDIA - middle ear effusion ***without perforation***, which persists > 3 months.

- initiated by episode of tympanic perforation & acute infection (recurrent AOM, traumatic perforation, placement of ventilation tubes): inflammation, ulceration, granulation tissue formation (destroying surrounding bony margins).
- bacteria translocate from external auditory canal; *P. aeruginosa* is most commonly recovered organism (48-98%), then *S. aureus*.

1. **SAFE EAR DISEASE (s. tubotympanic disease)** - **CENTRAL perforations of pars tensa** - some tympanic substance remains between rim of perforation and bony sulcus tympanicus.
2. **UNSAFE EAR DISEASE (s. atticoantral disease)** - *complications* (labyrinthitis, facial paralysis, intracranial suppuration, cholesteatoma, etc) are more likely to occur:

persistent chronic suppurative otitis media ***after appropriate medical treatment*** should alert physician to consider CHOLESTEATOMA!

a) **ATTIC perforations of pars flaccida** lead into epitympanum.

b) **MARGINAL perforations of pars tensa** - no tympanic substance between edge of perforation and bony sulcus tympanicus.

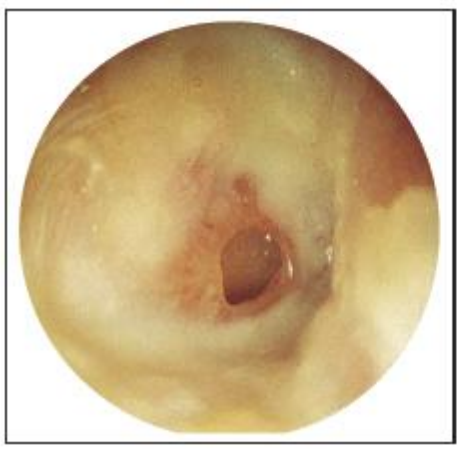
DISCHARGE & DEAFNESS

- typically persistent disease, insidious in onset; often causes severe destruction and irreversible sequelae.
- conductive **HEARING LOSS**.
- painless (!!!), serous ÷ purulent **OTORRHEA** (may be foul-smelling → social stigma).
  N.B. fever, vertigo, pain should raise concern about intratemporal / intracranial complications!
- *exacerbations*:
  – may follow URI or occur when water enters middle ear.
  – persistent exacerbations may produce **AURAL POLYPS** (granulation tissue that prolapses from middle ear through perforation into ear canal - almost invariably associated with cholesteatoma!!!) and **destructive changes in middle ear** (e.g. necrosis of long process of incus).

DIAGNOSIS

- examination with **operating microscope and adequate suction** equipment is required (in young children, short-acting, general anesthetic is sometimes required).

Fig. Chronic suppurative otitis media



Chronic perforation of the tympanic membrane with purulent discharge.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

TREATMENT

Responds more to topical than to systemic therapy!

- swimming is contraindicated during treatment.

Successful TOPICAL THERAPY consists of 3 important components:

1) **antibiotic drop** - must cover *P. aeruginosa* + *S. aureus* (antibiotics that meet this initial criterion are AMINOGLYCOSIDES and **FLUOROQUINOLONES** [drugs of first choice!!!])
   + **steroids** (in combination with a/b) – reduce granulation tissue!

   CIPROFLOXACIN + HYDROCORTISONE are typically used

   – 0.3% topical antibiotic solution contains 3000 mcg/mL - concentration 100-1000 times MIC - topical therapy can not fail because *organism is resistant*!!!
   – emergence of resistance is extremely uncommon - rapid kill rates do not permit even mutant strains with higher MICs to survive.
   – sensitivity reports (from clinical laboratory) are irrelevant - sensitivity testing is designed for tissue concentrations achievable by systemic administration (e.g. pseudomonad with MIC of 48 mcg/mL is likely to be reported as resistant by clinical laboratory).

   Culture & sensitivity are of little benefit so long as therapy is topical

   – failures are almost always ***failures of delivery*** (infectious debris, granulation tissue, cholesteatoma, neoplasia, cerumen, etc) – if necessary, try systemic therapy.

2) regular aggressive **AURAL TOILET** (critical process in treatment - remove mucoid exudate or desquamated epithelium → topically applied preparations can penetrate affected tissues):
   a) traditionally, in otolaryngology, aural toilet has been achieved using ***microscope & microinstruments*** to mechanically remove such materials 2-3 times per day (just before administration of topical antimicrobial agents).
   b) ***aural irrigation*** (effective less burdensome alternative) - 50% peroxide irrigated through external auditory canal.

3) **control of granulation tissue** (granulation tissue can prevent topical agents from penetrating to infection site): topical steroids, cautery (e.g. silver nitrate sticks).

For exacerbations:

- ear canal & middle ear are **thoroughly cleaned** (suction and dry cotton wipes).
- 2% **ACETIC ACID** with 1% **HYDROCORTISONE** is instilled into ear (tid for 7-10 days).
- *severe exacerbations* → systemic broad-spectrum **antibiotic** for 3-4 weeks.

If otitis fails to respond to combination of topical and systemic therapy → TYMPANOMASTOIDECTOMY (± surgical removal of cholesteatoma).

- general and most desirable outcome is dry, nondischarging, healthy cavity.
- **hearing reconstruction** should also be performed, but it is often completed during second surgery once dry ear has been achieved.
- keep ears dry (any water entry into mastoid cavity will trigger ear infection).

PREVENTION

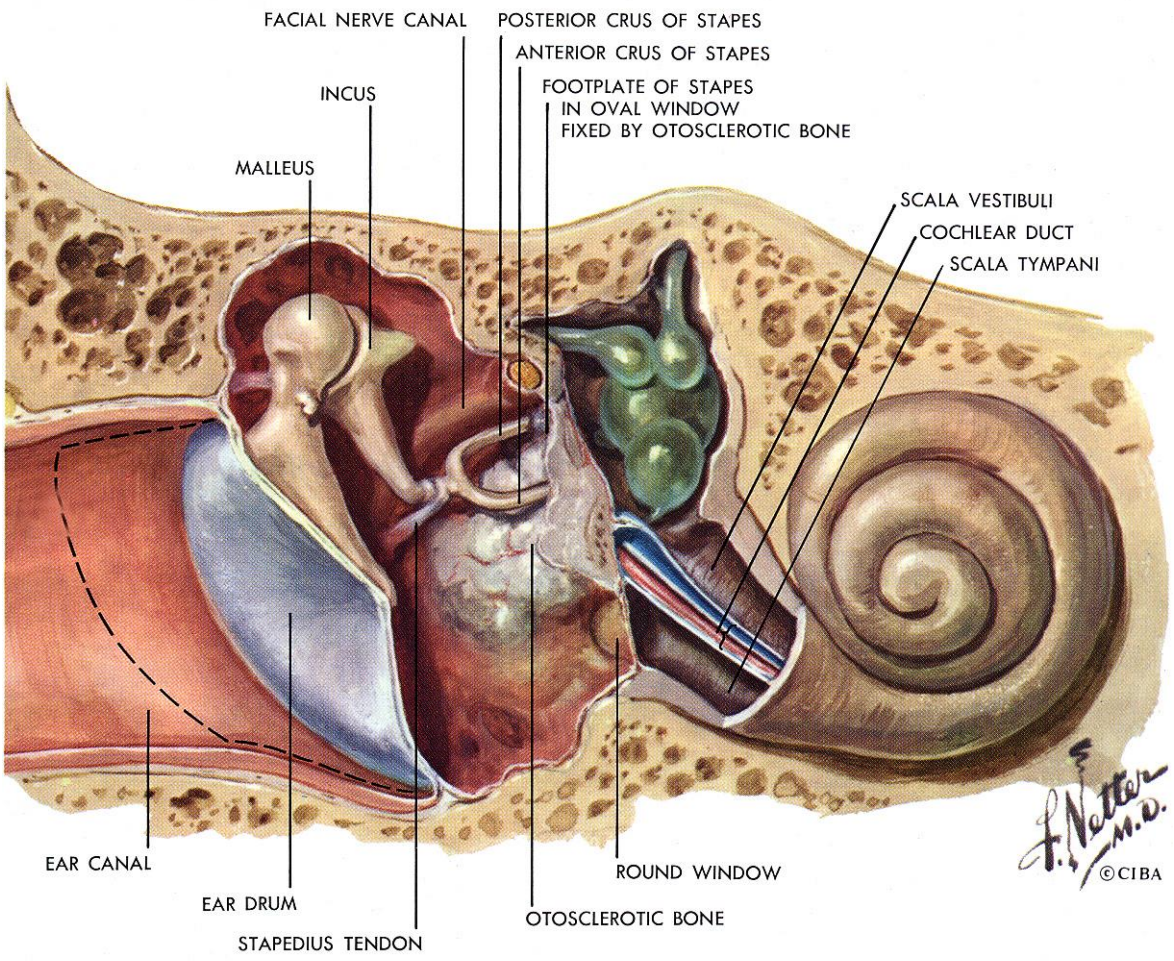
- swimming is not contraindicated if patients dry their ears afterward.
- **TYMPANOPLASTY** (surgery that seals perforation); for myringoplasty to be successful, ear should be dry preoperatively for ≥ 6 weeks.

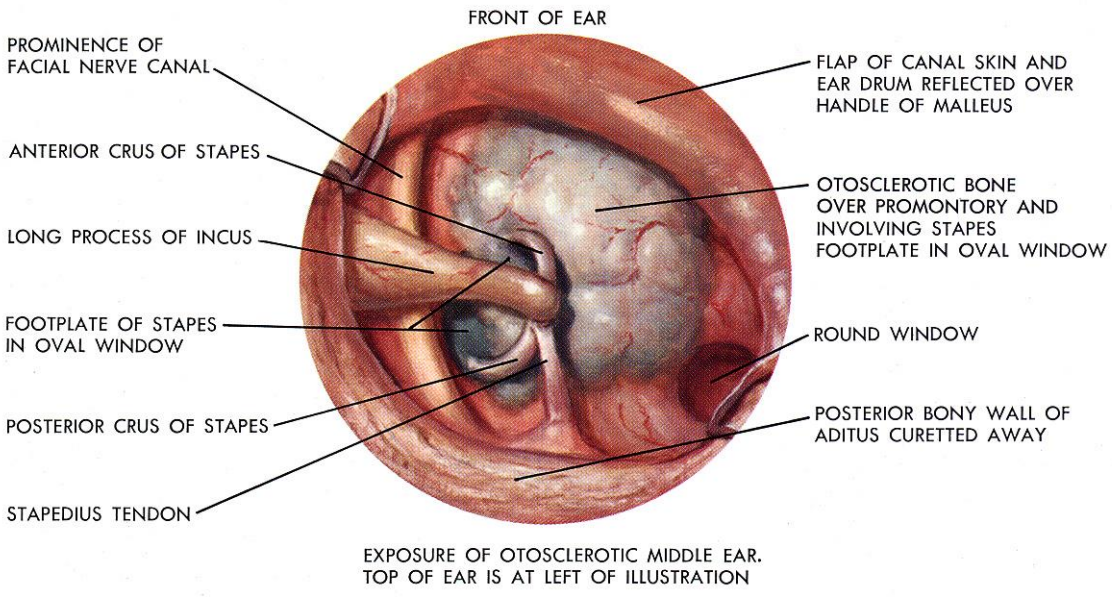
OTOSCLEROSIS

- ankylosis of stapedial footplate (local osseous dyscrasia of labyrinthine capsule).

Histology - pleomorphic replacement of normal bone: **early (active) stage** - osseous *rarefaction* (otospongiosis)* → maturation into *sclerotic* inactive foci - **end (stable) stage**.

*irregularly arranged, enlarging foci of new, immature bone interspersed with numerous vascular channels.





EXPOSURE OF OTOSCLEROTIC MIDDLE EAR.
TOP OF EAR IS AT LEFT OF ILLUSTRATION

Source of picture: Frank H. Netter "Clinical Symposia"; Ciba Pharmaceutical Company; Saunders >>

EPIDEMIOLOGY

- tends to be ***hereditary*** (probably autosomal dominant);
  - generally limited to **white population**.
  - 10% of white adults have foci of otosclerosis, but only 10% develop conductive hearing loss.
  - ***measles virus*** may play important role in gene activation! (measles vaccination is decreasing otosclerosis incidence).
- **women** : men = 2 : 1

CLINICAL FEATURES

Slowly progressive asymmetric ***CONDUCTIVE HEARING LOSS*** *in adult with normal tympanic membrane.*

- bilateral in 70% cases.
- becomes clinically evident after age 20 yrs.
- ***tinnitus*** may be present (resolves after successful surgical management).
- *pregnancy* and *estrogen therapy* accelerate progression.
- in 8% may progress to basal turn of cochlea (*RETROFENESTRAL OTOSCLEROSIS*), parts of labyrinth - *LABYRINTHINE OTOSCLEROSIS*) → uncorrectable ***sensorineural hearing loss***.

DIAGNOSIS

OTOSCOPY – **normal** (10% of patients demonstrate **SCHWARTZE sign** - reddish-blue hue over promontory and oval window niche areas - secondary to rich vascular supply associated with immature bone).

AUDIOMETRY - **conductive hearing loss**: low-frequency → high-frequency → maximal conductive loss of 50-65 dB across all frequencies.

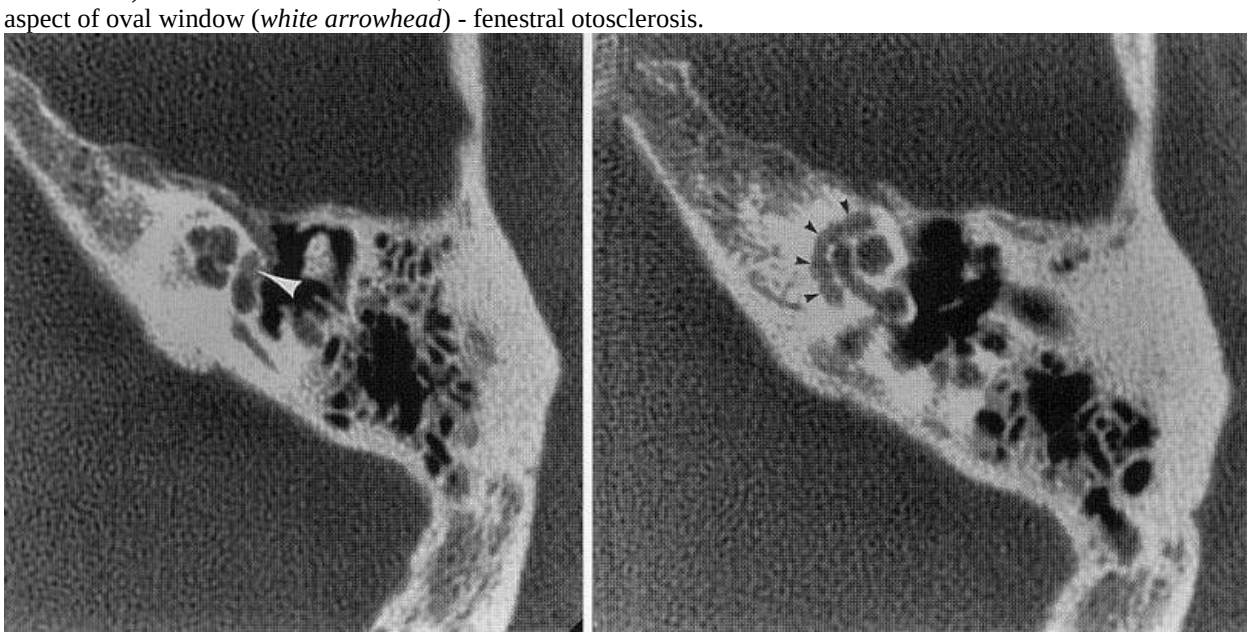
- stapes fixation produces **CARHART notch** at 2 kHz - elevation of bone conduction thresholds of 5 dB at 500 Hz, 10 dB at 1000 Hz, 15 dB at 2000 Hz, and 5 dB at 4000 Hz.

TYMPANOMETRY - **normal** (type A) tympanogram.

ACOUSTIC REFLEXES - abnormal (earliest evidence of otosclerosis!): ½ healthy population may show initial increase in compliance at stimulus onset, but **compliance increase at offset** is pathognomonic to stapedial fixation.

- advancing fixation affects both *ipsilateral* and *contralateral* acoustic reflexes, even in unilateral disease.

Axial CT of fenestral and retrofenestral otosclerosis - areas of demineralized bone surround basal turn of cochlea (*black arrowheads*) - retrofenestral otosclerosis; small focus of demineralization in area of fissula ante fenestram and anterior aspect of oval window (*white arrowhead*) - fenestral otosclerosis.



DIFFERENTIAL DIAGNOSIS

- various systemic hyperostoses (Paget's disease, fibrous dysplasia, craniometaphyseal dysplasia).

TREATMENT

1) trial with **HEARING AID**.
2) **fluoride** supplementation.
3) **MICROSURGERY** (indication - conductive hearing loss with **> 20 dB air-bone gap**):
   Many think surgical treatment for otosclerosis has reached perfection!
   a) total or partial STAPEDECTOMY and replacing it with prosthesis.
   b) STAPEDOTOMY.

CHOLESTEATOMA

- trapped squamous epithelium - can expand only at expense of bone that surrounds it → destructive lesion that ***can erode & destroy any important structure*** within temporal bone.

ETIOPATHOLOGY

a) healing of otitis media - stratified squamous epithelium of ear canal **migrates** to cover denuded areas in middle ear cleft.
b) **hyperplasia** of basal layer of stratified squamous epithelium of pars flaccida.
c) progressive **retraction** of pars flaccida or pars tensa.
d) squamous **metaplasia** in middle ear due to long-standing infection.

- once stratified squamous epithelium is established, it begins to desquamate & accumulate in ever-enlarging concentric layers.
- histology is indistinguishable from that of *sebaceous cysts* or *keratomas* in any other body portion: sac lined by keratinizing squamous epithelium and filled with keratin debris.
- *collagenases* in epithelium destroy adjacent bone.

1. CONGENITAL - squamous epithelium trapped during EMBRYOGENESIS.
   – typically in anterior mesotympanum or in perieustachian tube area (can obstruct eustachian tube), i.e. in ventral cephalad portion of middle ear cleft (vs. acquired cholesteatomas – in dorsal cephalad portion).
   – *intact normal-appearing tympanic membrane*!
   – identified in early childhood (6 mo to 5 y).

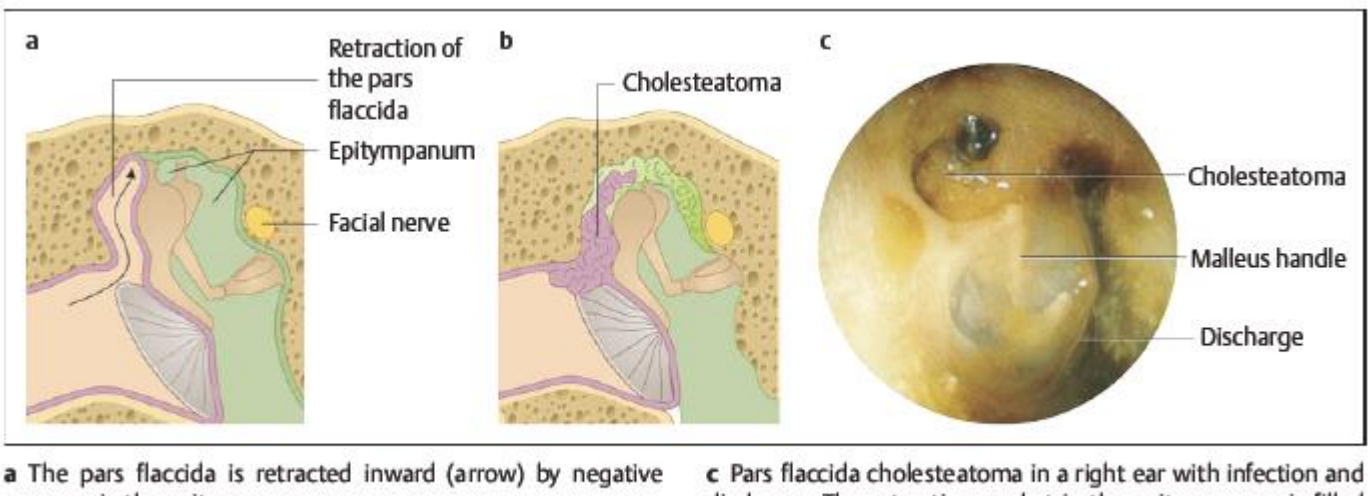
2. PRIMARY - result of progressive tympanic membrane RETRACTION:

Any retraction pocket can result in cholesteatoma if pocket becomes deep enough to trap desquamated epithelium!

   a) medial retraction of ***pars flaccida*** into epitympanum → lateral wall of epitympanum (scutum) is slowly eroded; ossicular destruction is common; may pass posteriorly through aditus ad antrum → erosion of tegmen mastoideum, dura exposure, erosion of lateral semicircular canal (deafness & vertigo).
   b) retracted ***posterior quadrant*** of tympanic membrane; likely to produce facial nerve exposure and destruction of stapedial superstructure.

typical primary attic cholesteatoma in earliest stages:

Fig. Pars flaccida cholesteatoma



a The pars flaccida is retracted inward (arrow) by negative pressure in the epitympanum.
b Epithelial debris creates a nidus for infection and inflammation, which leads to the actual cholesteatoma.
c Pars flaccida cholesteatoma in a right ear with infection and discharge. The retraction pocket in the epitympanum is filled with debris and epithelial residue.

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

3. SECONDARY - direct consequence of tympanic membrane INJURY (otitis, trauma, surgery, insertion of tympanostomy tube, etc) → epithelium implantation into middle ear.
   N.B. *posterior marginal perforations* are most likely to result in cholesteatoma (but even central perforations occasionally result in cholesteatoma).

Fig. Pars tensa cholesteatoma

Dry pars tensa cholesteatoma in the anterosuperior quadrant. The keratin debris is clearly visible. The location is somewhat unusual (the posterosuperior quadrant is more commonly affected).

Source of picture: Rudolf Probst, Gerhard Grevers, Heinrich Iro "Basic Otorhinolaryngology" (2006); Georg Thieme Verlag; ISBN-13: 978-1588903372 >>

CLINICAL FEATURES

- permanent, moderate **conductive hearing loss** - filled middle ear space, ossicular damage.
- hallmark symptom - **painless otorrhea**, either unremitting or frequently recurrent.

if cholesteatoma becomes infected, ***infection is extremely difficult to eradicate*** (cholesteatoma has no blood supply - systemic antibiotics cannot reach center of cholesteatoma) - otorrhea either persists or recurs.

COMPLICATIONS

- erosion of bony covering of lateral aspect of horizontal semicircular canal → perilymphatic fistula.
- erosion of bony covering of CN7 canal → CN7 palsy.
- erosion of tegmen tympani → CSF leak, intracranial abscess, meningoencephalocele; sometimes become large enough to distort normal brain and produce mass effects.

  POTENTIALLY FATAL **CNS complications** (brain abscess, meningitis)!!!

DIAGNOSIS

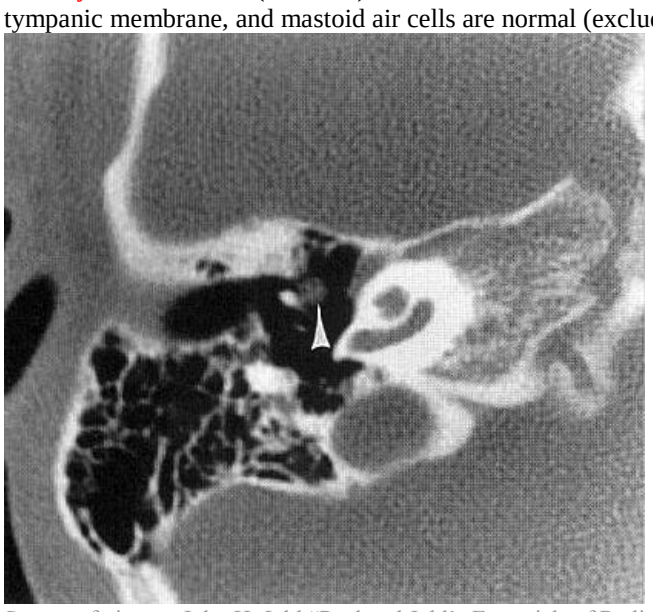
OTOSCOPY:

- **white debris** in middle ear
- mucopus **drainage** and **granulation tissue** that fill canal;
- cholesteatoma eventually produces **tympanic membrane perforation** (present in > 90% cases).
- destruction of ear canal bone adjacent to perforation.
- sometimes can be seen **retraction pocket**, collection of squamous epithelium, or absent tympanic membrane.

CT (imaging modality of choice) - ***nonenhancing mass eroding bone***, including ossicles, with sharply defined smooth margins, isodense with CSF.

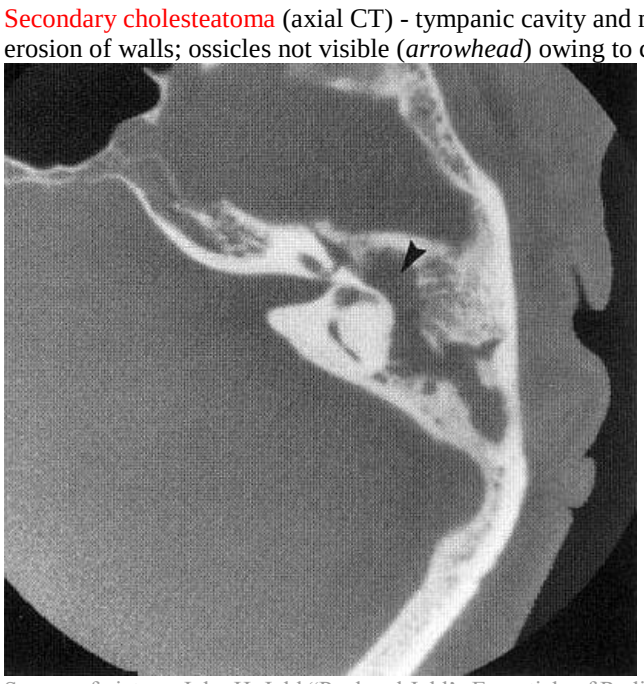
- CT cannot reliably determine full extent of disease.
- distinguishing between *cholesteatoma* and *pure mucosal disease* (e.g. postoperative granulation tissue) is difficult (even with MRI).

Primary cholesteatoma (axial CT) - small, rounded soft-tissue mass in middle ear cavity near malleus; tympanic cavity, tympanic membrane, and mastoid air cells are normal (excluding secondary cholesteatoma):



Source of picture: John H. Juhl "Paul and Juhl's Essentials of Radiologic Imaging", 7th ed. (1998); Lippincott Williams & Wilkins; ISBN-10: 0-397-58421-0 >>

Secondary cholesteatoma (axial CT) - tympanic cavity and mastoid antrum completely opacified, with areas of peripheral erosion of walls; ossicles not visible (*arrowhead*) owing to complete erosion by cholesteatoma:



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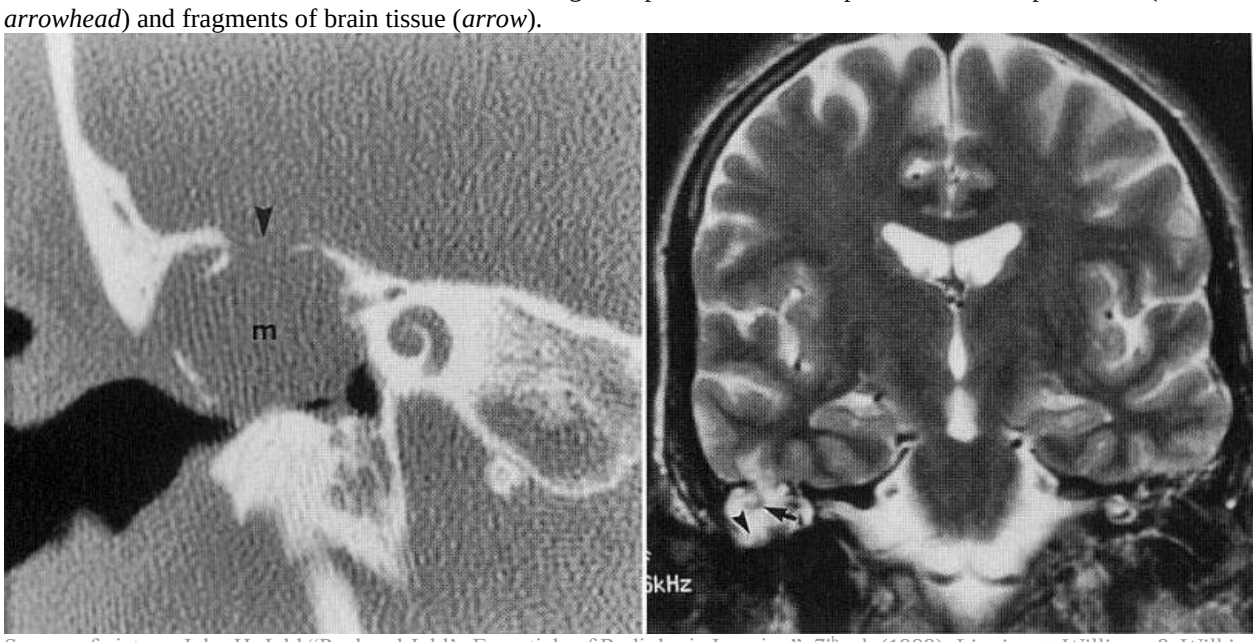
Tympanic cavity meningoencephalocele:
A: Coronal CT - large defect in tegmen tympani (*large arrowhead*) with soft tissue mass (m) in middle ear.
B: Noncontrast T2-MRI confirms that mass is meningoencephalocele and composed of cerebrospinal fluid (*small arrowhead*) and fragments of brain tissue (*arrow*).



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TREATMENT

- **antimicrobial therapy** should be **topical**, but systemic therapy is occasionally helpful adjunct.

Virtually all cholesteatomas ***should be excised***! (even in only hearing ear - cholesteatoma presents greater risk to residual hearing than surgery) by TYMPANOMASTOIDECTOMY – removing all debris, creating cavity that communicates with ear canal:

Canal-wall-up (closed) operations - maintain normal appearance, but higher risk of recurrences.

Canal-wall-down (open) operations - permanently rid patient of cholesteatoma; (semi)annual canal cleaning needed; vertigo may develop after exposure to water or cold air.

- if *ossicles* are involved, they must be removed (to avoid recurrence) → reconstruct ossicular chain (at end of procedure or as part of secondary operation).
- if ***membranous labyrinth is opened*** at any point during procedure, administer broad-spectrum intravenous **antibiotics + steroids** immediately.
- patient must be *monitored indefinitely* - recurrence can occur long after initial surgical excision.

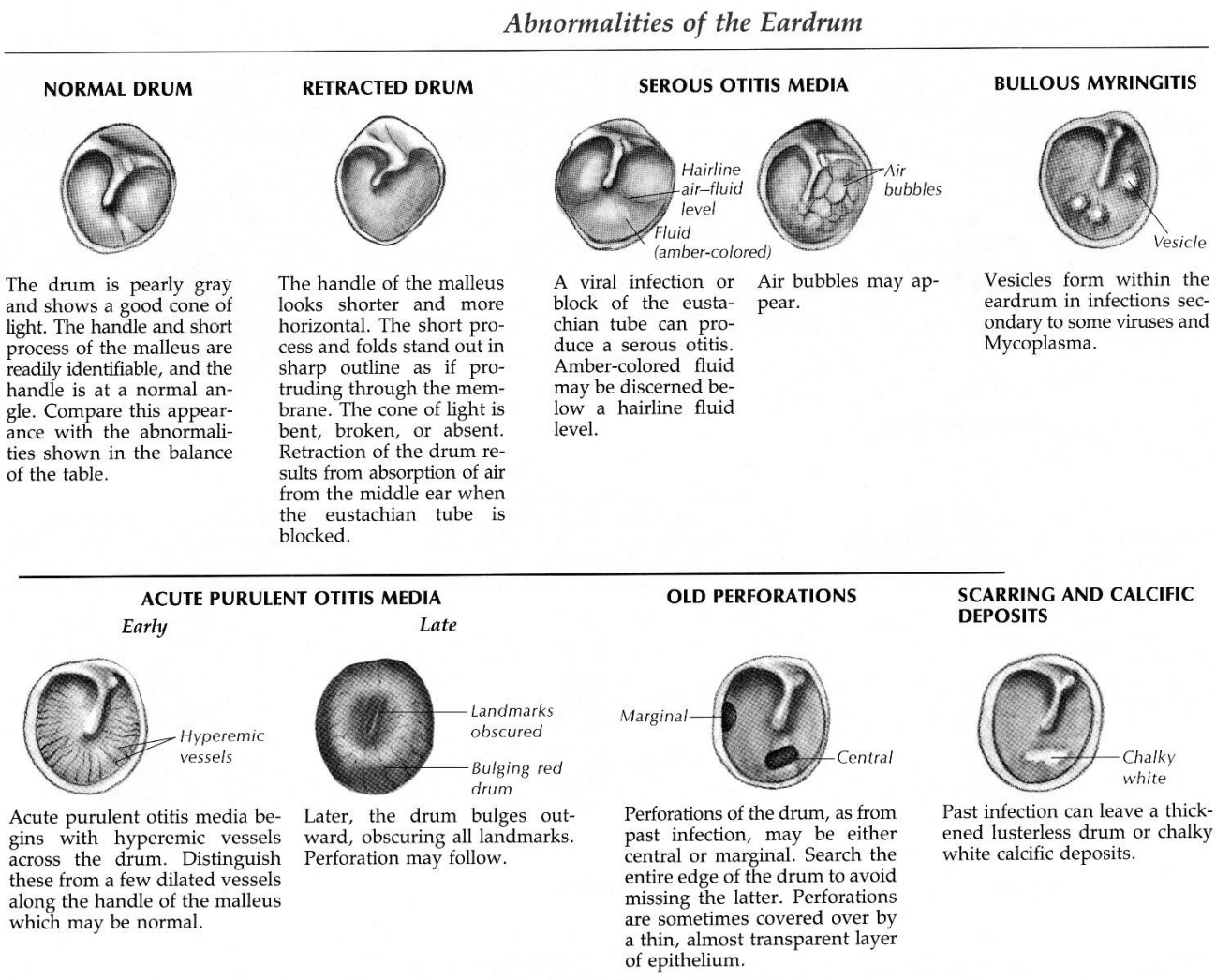
NEOPLASMS

Benign tumors: facial schwannomas, glomus tumors (s. paragangliomas, chemodectomas), choristomas, adenomatous tumors, Langerhans histiocytosis, etc.

- despite benign histopathological characteristics, benign lesions may be locally destructive.

Squamous cell carcinoma rarely originates in middle ear.

- persistent otorrhea of chronic otitis media may be predisposing factor.
- treatment - radiation therapy and resection of temporal bone.



Source of picture: Barbara Bates "A Guide to Physical Examination", 3rd ed. (1983); J.B. Lippincott Company; ISBN-13: 978-0397543991 >>

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