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Hearing Disorders

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Prevalence Estimates

Percentage	Number (millions)	
6.6%	19.8	"hearing impaired"
8%	24	PTA > 25 dB HL
10%	30	some hearing impairment

2010 census ~ 300 million

2

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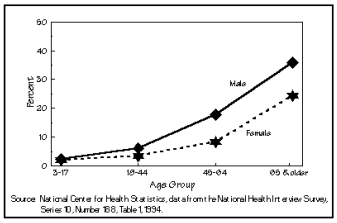
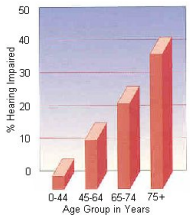
Prevalence by Age and Gender



Source: National Center for Health Statistics, data from the National Health Interview Survey, Series 10, Number 188, Table 1, 1994.



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Hearing Disorders in Adults

- Permanent (Sensorineural)
  - Presbycusis
  - Noise Induced Hearing Loss
  - Meniere's disease
  - Ototoxic diseases & drugs
  - Other
- Fixable (Conductive)
  - Otosclerosis
  - Other

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Presbycusis

- The progressive loss of hearing that occurs with age. AMA Encyclopedia of Medicine
- Prevalance
  - Conservative estimate ≈ 25% of those >65 have a handicapping hearing loss

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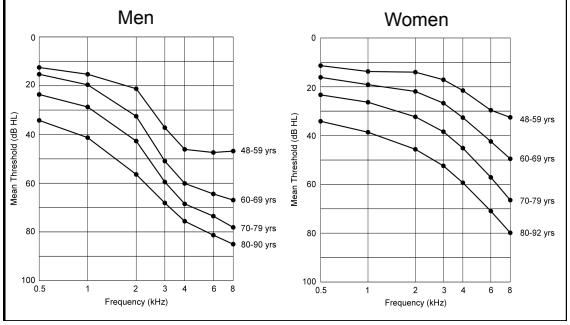
Hearing and Ageing

(Cruickshanks et al., 1998).



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Classic Categories
(Schuknecht)

- Sensory
- Neural
- Metabolic
- Mechanical

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Sensory Presbycusis

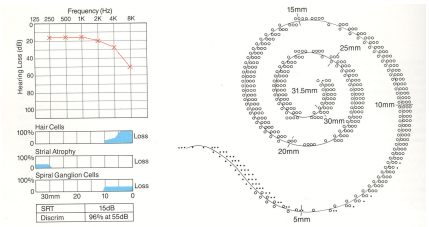

Figure 5.32 EARLY SENSORY PRESBYCUSIS (left ear) There is a loss of hearing in the high-est frequencies, and a concomitant loss of the hair cells in the basal turn of the cochlea. There is also evidence of secondary neural degeneration as shown by the loss of spiral gang-lion cells in the basal turn of the cochlea. (Large circles indicate a hair cell was present; dots indicate a hair cell was missing.)

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Sensorineural Presbycusis

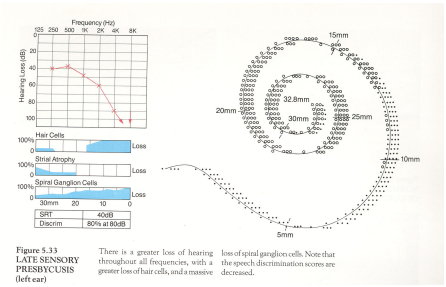

Figure 5.33 LATE SENSORY PRESBYCUSIS (left ear) There is a greater loss of hearing throughout all frequencies, with a greater loss of hair cells, and a massive loss of spiral ganglion cells. Note that the speech discrimination scores are decreased.

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Noise Induced Hearing Loss

- Permanent, sensorineural loss from chronic exposure to high-intensity sound
  - (> 80 dB A)
- Incidence
  - #1 occupational hazard
  - Estimated that at least 16 million in U.S. suffer from some form of NIHL
  - Majority of impairments in middle age

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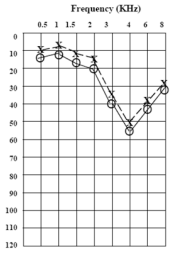
Noise Induced Hearing Loss

- Noise notch
  - Sensorineural
  - Worst at 4–6 kHz
  - LE often worse than right
- Tinnitus
  - Ringing or other sound



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Meniere's Disease

- Disease characterized by episodes of:
  - Vertigo
    - Nausea
    - Vomiting
  - Tinnitus
    - Roaring or buzzing
  - Hearing loss
  - Fullness

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Meniere's Disease

- Incidence
  - #3 cause of sensorineural hearing loss in adults
  - Low end = 46:100,000 (Stahle et al., 1978)
  - High end = 160:100,000 (Cawthorne & Hewlett, 1954)
- Prevalence = incidence X 25

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Meniere's Disease Sx

- Hearing loss - Configuration
  - Early stages
    - Classic = low frequency loss (rising)
    - Occasionally flat
    - Rarely high-frequency (sloping)
  - Later
    - Flattens out as loss progresses

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Hearing Loss Configuration

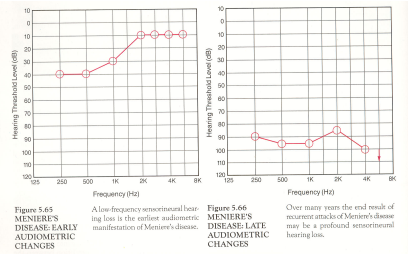

Figure 5.65 MENIERE'S DISEASE: EARLY AUDIOMETRIC CHANGES — A low-frequency sensorineural hearing loss is the earliest audiometric manifestation of Meniere's disease.

Figure 5.66 MENIERE'S DISEASE: LATE AUDIOMETRIC CHANGES — Over many years the end result of recurrent attacks of Meniere's disease may be a profound sensorineural hearing loss.

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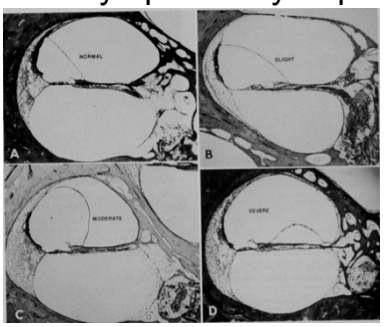
Endolymphatic Hydrops



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Diseases Causing Hearing Loss

- More Common
  - Bacterial meningitis
  - Sudden onset
- Less Common
  - Mumps
  - Measles
- Rare
  - diphtheria
  - whooping cough
  - typhoid
  - scarlet fever
  - chickenpox
  - flu
  - cold viruses
  - polio
  - herpes virus
  - other

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Major Ototoxic Drugs

- Antibiotics
  - Streptomycin
  - Neomycin
  - Kanamycin
  - Gentamicin
- Chemotherapueutics
  - Cisplatin
  - Carboplatin
- "Loop" Diuretics
  - Furosemide (lasix)
  - Bumetamide (bumex)
  - Ethacrinic acid (edecrin)
- Aspirin

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Otosclerosis

Disease of the bone of the otic capsule.

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Clinical Forms:

- Fixation of the stapes (most common)
- Cochlear impairment + stapes fixation
- Pure cochlear
  (labyrinthine, cochlear or retrofenestral)

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Stapes Fixation

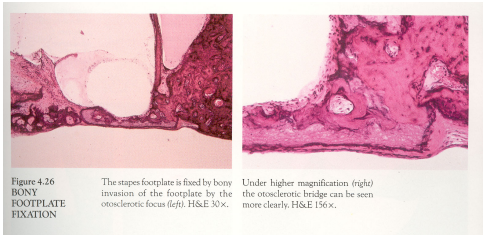

Figure 4.26 BONY FOOTPLATE FIXATION

The stapes footplate is fixed by bony invasion of the footplate by the otosclerotic focus (*left*). H&E 30×.

Under higher magnification (*right*) the otosclerotic bridge can be seen more clearly. H&E 156×.

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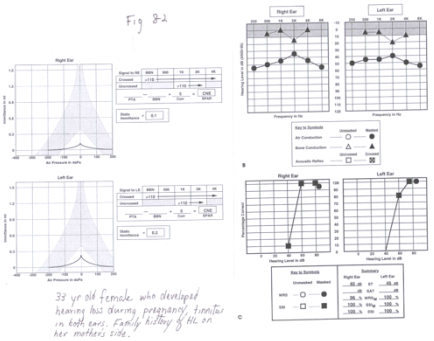
Otosclerosis



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Childhood Hearing Disorders

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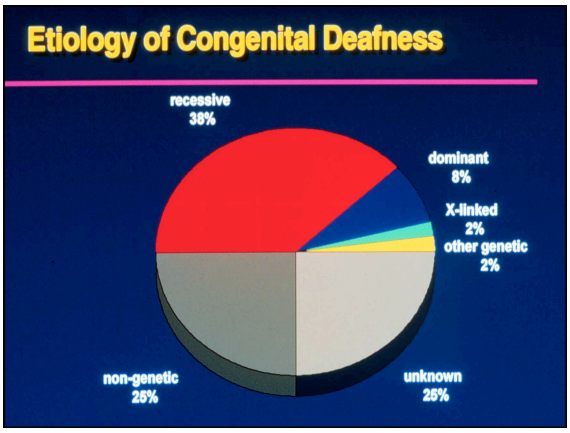
Prevalence

- Youngsters ≤ 17
  ~ 1% of have SRTs ≥ 26 dB HL
- Newborns
  ~1 per 1,000 live births

24



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"ABCDEFG's of Deafness"

(JCNH, 1981)

- A - Asphyxia
- B - Bacterial meningitis
- C - Congenital/perinatal infections
- D - Defects of the head or neck (e.g., cleft palate, pinna abnormalities)
- E - Elevated bilirubin
- F - Family history of childhood hearing impairment
- G - Gram birthweight less than 1,500 gms.

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Congenital Hearing Impairment

Inherited

(Endogenous)

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Incidence

- In general population = 1/2000 - 1/6000 births
- Among congenitally deaf ≈ 50% hereditary
- Pattern of inheritance
  - About 75 - 80% = recessive
  - About 20 - 25% = dominant
  - Rest = too rare to worry about

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Recessive Inheritance

- Defective gene must be present at the same locus on both chromosomes in the pair for defect to be present in offspring
  - 50% = carriers
  - 25% = affected offspring
  - 25% = completely normal

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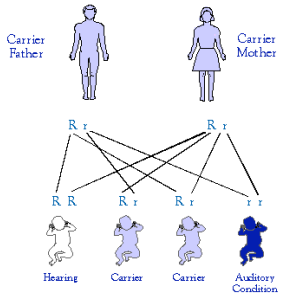
Recessive Inheritance



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Dominant inheritance

- Abnormality may be expressed when defective gene is in only one of pair of chromosomes
  - 50% = affected offsping
  - 50% = completely normal
  - No carriers

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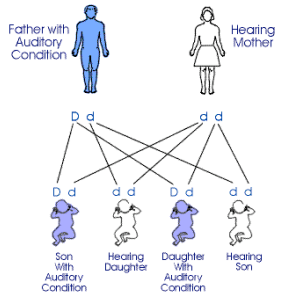
Dominant Inheritance



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Nonsyndromic Hearing Loss Genes

- Nomenclature:
  - Loci of hearing loss genes are numbered consecutively to reflect order of discovery.
  - Mode of inheritance is denoted by prefixes:
    - DFNA (dominant)
    - DFNB (recessive)
    - DFN (X-linked)

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Nonsyndromic Hearing Loss Genes

- Recessive
  - Estimate: at least 25 different genes involved
  - DFNB1 accounts for about 50%
  - DNA testing can identify deafness-causing mutations of this gene in most cases
- Dominant
  - Estimate: at least 22 different genes involved

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Major Recessive Syndromes

- 50% of recessive losses are syndromatic
- Pendred's Syndrome (1896)
  - Congenital hearing loss
  - Thyroid dysfunction (goiter) in adolescence
- Usher's Syndrome (1914)
  - Cochlear loss (congenital or degenerative).
  - Degeneration of inner layer of retina (retinitis pigmentosa)

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Major Dominant Syndromes

- Waardenberg's Syndrome (1951)
  - 20% = unilateral or bilateral hearing loss
  - 99% = lateral displacement of medial canthi (wide set eyes)
  - 78% = flat bridge of nose
  - 25% = iris heterochromea
  - 17% = white forelock

- Branchiootorenal Syndrome
  - Renal dysfunction
  - Variable hearing loss
    - Conductive, sensorineural, mixed
    - Mild - profound

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For your own info:

- 90% of congenitally hearing impaired have 2 normally hearing parents.
- 30% of offspring from 2 deaf parents have hearing losses also.
- More precise predictions can only be made by expert.

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Congenital Hearing Impairment

Acquired
(Exogenous)

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Congenital or Perinatal Infections

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The TORCH Complex

- T= toxoplasmosis
- O= other (syphilis)
- R= rubella
- C= CMV
- H= Herpes

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Toxoplasmosis

- Toxoplasma gondii parasite
- Transmitted across placenta
- Classic Sx Triad
  - Chorioretinitis
  - Hydrocephalus
  - Intracranial calcifications
- One estimate = 17% of infected infants will develop hearing loss

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Congenital Rubella: Sx Triad

- bilateral hearing loss
- cataracts (40%)
- heart anomalies (50%)

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Rubella Incidence

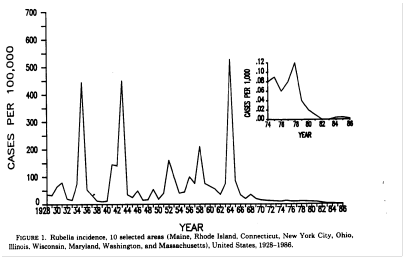

FIGURE 1. Rubella incidence, 10 selected areas (Maine, Rhode Island, Connecticut, New York City, Ohio, Illinois, Wisconsin, Maryland, Washington, and Massachusetts), United States, 1928–1986.

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Herpes Group of Viruses

- Cytomegalovirus
- Herpes simplex Type I
- Herpes simplex Type II
- Epstein-Barr
- Varicella-Zoster

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Cytomegalovirus (CMV)

- Incidence
  - Most common known microbiological cause of brain damage in infancy.
  - > 7000 children born each year with hearing loss from CMV
- Sensorineural Hearing Loss
  - Pattern is very variable
  - Most are bilateral, severe/profound
  - Majority show progressive loss.

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High Multiple Handicap Rate:

- ~50% have one or more educationally significant disabilities in addition to h/loss.
- 22% have two additional disabilities:
  - 19% MR
  - 13% CP
  - 10% Orthopedic problems
  - 10% LD
  - 9% Emotional/behavior problems
  - 6% blindness or significant visual impairment
- Score lower on standardized tests of academic achievement than h/i peers

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Herpes Simplex Type II

- Becoming one of the most common sexually transmitted diseases
  - 20-25% of the population
- Disease Process
  - 82% of neonatal infections are generalized throughout the body.
    - High mortality
    - Only 4 % of infected infants survive without being affected.

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Hypoxia/Anoxia

- Hypoxia - Amount of oxygen in air, arterial blood, or body tissues is below normal, but short of anoxia.
- Anoxia - Absence or almost complete absence of oxygen in air, arterial blood, or body tissues.
- Asphyxia
  - Impaired or absent exchange of O_2 and CO_2 in breathing.
  - Results in a lack of O_2 (anoxia) and increased CO_2 (hypercapnia) in the blood and tissues.
- Anemia - Deficiency of oxygen-transporting material (RBC's, hemoglobin) in the blood.
- Ischemia - Localized shortage of blood due to obstruction of the blood supply.

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Asphyxia

Apgar score

A: Activity (muscle tone)
P: Pulse
G: Grimace (reflex irritability)
A: Appearance (skin color)
R: Respiration

- Two points in each area
- < 7 = “at risk ”
- 3 or lower = "high risk "
- 1 min = asphyxia and need for ventilation
- 5 min = neurological impairment or death

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Hearing Loss

- About 4% with severe perinatal asphyxia develop sensorineural loss
- May damage CANS
- Possible cause of auditory neuropathy?
  - Normal OAEs
  - No ABR

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Elevated Bilirubin (hyperbilirubinemia; jaundice)

- Excessive amount of bilirubin in the blood.
- Any factor that causes:
  - excessive breakdown of red blood cells
  - abnormal metabolism of bilirubin by the liver
- Most Common Cause = Rh Incompatibility
  - Erythroblastosis fetalis
  - Antibodies from Rh- mother attack Rh+ protein in RBC's of child
  - Causes immature RBC's (erythroblasts)
    anemia
    hyperbilirubinemia

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Treatment

- Phototherapy - light converts bilirubin to a water soluble form that can be excreted by the kidneys.
- Exchange blood transfusion if phototherapy fails

52

Kernicterus

- Neurological syndrome associated with bilirubin deposits in the CNS.
- Hearing loss:
  - Bilateral, sensorineural, high frequency
  - Possible auditory neuropathy?
- Highest multiple handicap rate of all congenital etiologies (71.1%)
- Most brain damage of any etiology

53

Gram Birthweight < 1,500

- Methods of estimating gestational age are unreliable.
- Consequently, prematurity is now defined in terms of birthweight:
  - Low BW < 2000 gms. (4.4 lbs)
  - Very low BW < 1500 gms. (3.3 lbs)
- Hearing loss probably associated with hypoxia/anoxia

54

Very Low Birthweight

- Highest MI rate of all congenital etiologies
  - 16%
  - 2.2% rate in general population
- 14-44% die as neonates
  - More are surviving as result of improved medical care

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Outer Ear Malformations

- Microtia
  - Auricle
  - Grades
    - I = small, but well formed
    - II = malformed
    - III = remnant
- Atresia
  - EAC
  - Grades
    - I = lesion to EAC alone
    - II = EAC lesion, bony TM, and malformed middle ear
    - III = EAC, TM and middle ear are absent

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Grade II Microtia + Atresia

57

Outer Ear Malformations

- Associated anomalies:
  - 12-47% have cochlear pathology
  - Tortuous VII N.

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Treacher-Collins Syndrome (Mandibulofacial Distosis)

1900

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Treacher-Collins Syndrome

- Etiology
  - Dominant
  - Arrested development of structures
    - primarily from the first branchial arch
    - probably also involving the second arch
  - Occurs between the 5th and 9th weeks of gestation

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Treacher-Collins Syndrome

- Signs and symptoms
  - Deformities of facial bone structure
  - Malformed middle and outer ear
  - Notch in lower eyelid
  - Cleft lip & palate
  - Anomalies of bones in extremities
- Hearing loss
  - Auricle, EAC, and middle ear -- malformed or totally absent
  - Usually a maximum conductive loss
  - Occasionally, inner ear is affected as well

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Audiological Treatment

- Great hearing aid users!!
- Microtia = one of few instances where BC aids are indicated

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Otological Treatment

- Microtia
  - Plastic surgery
  - Plastic prosthesis
- Atresia
  - Bilateral atresia - almost always try to open one ear canal with surgery
  - When hearing is OK, risks probably outweigh the potential benefits of surgery:
    - High risk of "iatrogenic" facial paresis
    - Very difficult and dangerous if middle ear cavity is absent

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External Otitis

Inflammation of the outer ear

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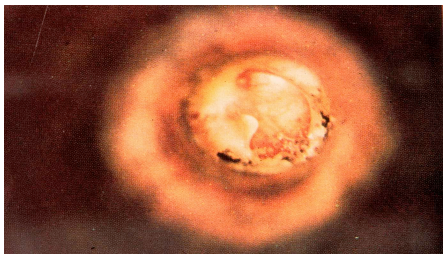
Outer Ear Fungus Infection



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External Otitis

- Rare, unless protective lining is damaged by some agent:
  - Moisture
    - Maceration (softening due to soaking) after swimming
    - Prolonged exposure to elevated temperature and humidity.
  - Trauma

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Symptoms

- Swelling (edema)
- Redness (erythema)
- Ear pain (otalgia)
- Drainage (otorrhea)
- Skin eruptions
- Polyps (fleshy masses)
- Conductive hearing loss? Depends on patency of ear canal.

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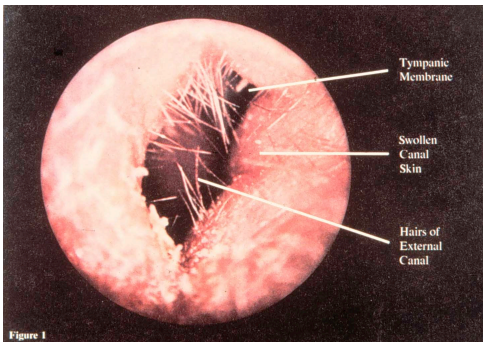

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Cerumenosis

- Impacted ear wax in EAC
- Symptoms
  - Reports of dizziness and tinnitus
  - Conductive hearing loss
    - Degree depends on extent of occlusion
    - 40 dB maximum loss with total closure

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Foreign Objects

"Don't stick anything smaller than your elbow in your ear"

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Foreign Objects

- Pieces of food (corn, beans, rice, etc.)
- Pebbles
- Insects
- Etc.

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Collapsed Canals

- When ear canals close due to pressure from the earphone headband during audiometric testing
- Most common in young children and geriatrics
- Tip Offs
  - Conductive loss (especially at high frequencies) with no history of middle ear disease
  - Variability in threshold responses

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Solutions

- Inserts
- Place phones up and back
- Hand held earphone

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Otitis Media

Inflammation of the middle ear

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Otitis Media Incidence

- Primarily a childhood disease (< age 8)
  - As many as 10% have frequent episodes
- High incidence populations:
  - Cleft palate (≥ 50%)
  - Downs syndrome
  - Native Americans
    - 2/3 have otorrhea before age 1
    - 90% by age 2
  - Allergy (62% of allergic preschoolers)

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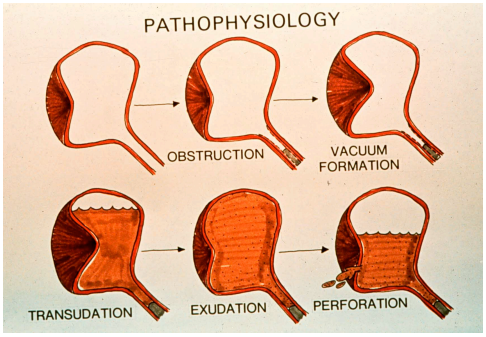

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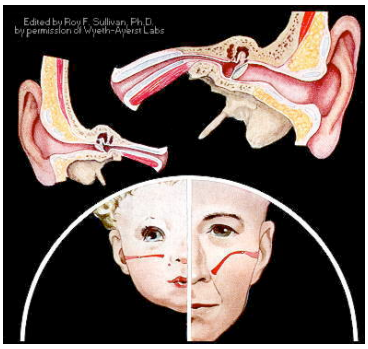

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Eustachian Tube Physiology

- **Normally closed – passive**
- **Opens 1000/day**
- **Functions to ventilate & drain middle ear**

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Eustachian Tube Maturation

- **At birth**
  - **13 mm long**
  - **10º angle**
- **Adult (reached by age 7)**
  - **35 mm long**
  - **45º angle**
- **Reason for increased vulnerability of young children to ME infection.**

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Acute Otitis Media

- Recent, usually abrupt, onset of signs and symptoms
- Effusion indicated by :
  - Bulging tympanic membrane
  - Limited or absent TM mobility
  - Air-fluid level behind the tympanic membrane
  - Otorrhea
- Signs or symptoms of inflammation
  - Distinct erythema of the tympanic membrane or
  - Distinct otalgia interferes with normal activity or sleep

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Acute OM - Early

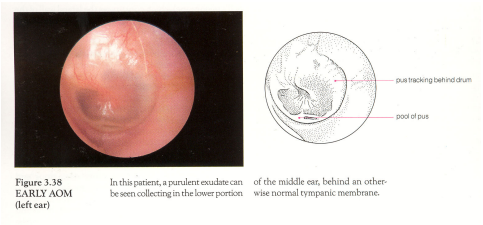

Figure 3.38 EARLY AOM (left ear) In this patient, a purulent exudate can be seen collecting in the lower portion of the middle ear, behind an otherwise normal tympanic membrane.

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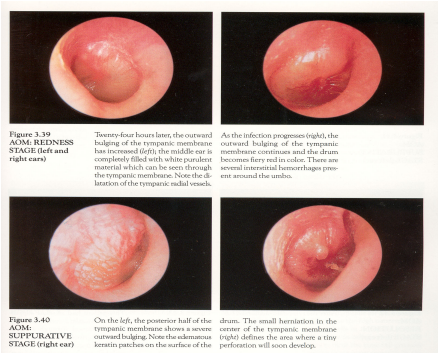

Figure 3.39 AOM: REDNESS STAGE (left and right ears) Twenty-four hours later, the outward bulging of the tympanic membrane has increased (*left*); the middle ear is completely filled with white purulent material which can be seen through the tympanic membrane. Note the dilatation of the tympanic radial vessels. As the infection progresses (*right*), the outward bulging of the tympanic membrane continues and the drum becomes fiery red in color. There are several interstitial hemorrhages present around the umbo.

Figure 3.40 AOM: SUPPURATIVE STAGE (right ear) On the *left*, the posterior half of the tympanic membrane shows a severe outward bulging. Note the edematous keratin patches on the surface of the drum. The small herniation in the center of the tympanic membrane (*right*) defines the area where a tiny perforation will soon develop.

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Otitis Media with Effusion

- Effusion
- No signs or symptoms of acute infection

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Serous OM

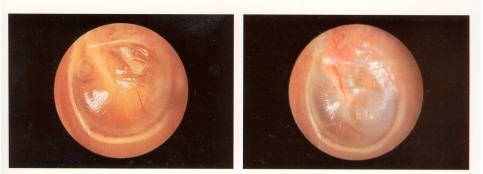

Figure 3.83 SEROUS OTITIS MEDIA: REAERATION (left ear)

On the *left*, the tympanic membrane is colored a golden-yellow and retracted onto the long process of the incus and the head of the stapes, which are abnormally prominent. *Right*, after Valsalva's maneuver, the patient was able to reaerate her middle ear.

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Serous OM

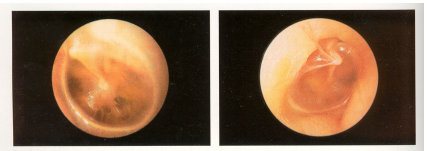

Figure 3.82 TYMPANIC MEMBRANE DISCOLORATION IN SOM (left and right ears)

Serous otitis media is characterized by the presence of a thin, watery, transparent, golden-yellow or straw-colored fluid within the middle ear. The tympanic membrane on the *left* shows an orange discoloration from the fluid; however, the transparency of the fluid allows the medial wall of the middle ear to be visualized. The tympanic membrane shown on the *right* has a golden-yellow color. The marked inward retraction of the tympanic membrane and the chalky white color and abnormal prominence of the short process of the malleus indicate that there is a substantial level of negative pressure in this middle ear.

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Secretory OM

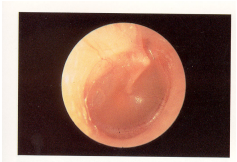

Figure 3.59 MUCOID OTITIS MEDIA: TYMPANIC MEMBRANE (right ear)

The tympanic membrane is slightly retracted and has a slight yellowish tinge, contrasting with the chalky white color of the malleus handle. The opalescence of the mucoid fluid within the middle ear prevents the medial wall of the middle ear from being clearly seen.

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Treatment: Acute Otitis Media

AAP (2004)

- < 6 mo.
  - Antibiotics
- 6 mo. to 2 yrs.
  - Antibiotics if Dx is certain, or illness = severe
  - "Observation option" = defer antibiotic Rx 2-3 days
    - If Dx is uncertain and illness is not severe

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Treatment: Acute Otitis Media

AAP (2004)

- ≥ 2 yrs.
  - Severe illness = antibiotics
  - Observation option if
    - Non-severe illness
    - Uncertain Dx

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Treatment: OME

- "Watchful waiting" for 3 months
  - 75 – 90 % resolve spontaneously
- Still OME at 3 months – test hearing
  - If significant hearing impaiment exists:
    - Speech-language eval
    - PE tubes

AAFP, AOHNS, AAP (2004)

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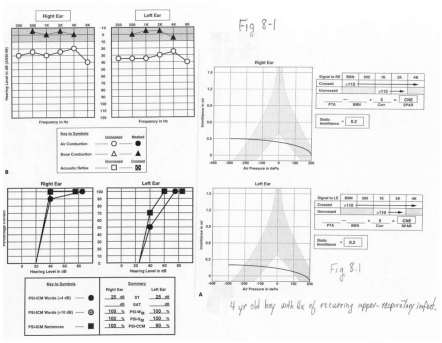
Otitis Media



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"Tympanogenic" Disease

- Four routes of infection spread:
  - Tegmental wall = fractured or eroded, or open infantal suture » meningitis.
  - Posterior wall » mastoiditis.
  - Jugular wall » systemic disease.
  - Opening of medial wall (via semicircular canal, windows, or other stucture) » labyrinthitis.

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Myringotomy

- Incision in the anterior-inferior quadrant of the eardrum, usually with aspiration of fluid from middle ear.
- Purpose:
  - drain fluid from middle ear
- Heals in 4 - 5 days

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Myringotomy

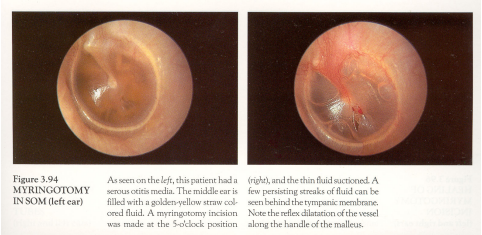

Figure 3.94 MYRINGOTOMY IN SOM (left ear) As seen on the *left*, this patient had a serous otitis media. The middle ear is filled with a golden-yellow straw colored fluid. A myringotomy incision was made at the 5-o'clock position (*right*), and the thin fluid suctioned. A few persisting streaks of fluid can be seen behind the tympanic membrane. Note the reflex dilatation of the vessel along the handle of the malleus.

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Tympanostomy Tubes

Pressure-Equalization (P.E.) tubes

Polyethylene tubes placed through the eardrum to keep a myringotomy incision open.

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Tympanostomy Tubes

- Purpose
  - Ventilate middle ear to compensate for eustachian tube dysfunction
    - Prevents build-up of negative pressure
  - Effective in restoring normal hearing
- Usually stay in place 6-8 months.
- If >1 year -- refer to otologist for recheck!

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PE Tubes

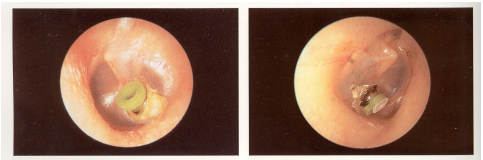

Figure 3.101 EXTRUDING VENTILATION TUBES (left and right ears) On the *left*, a large collar of accumulated keratin debris has almost lifted the medial flange of this green Silastic short Armstrong tube out of the tympanic membrane. *Right*, The medial flange of the green Silastic Castelli membrane tube has been lifted out of the drum by a collar of keratin and serous crust. The tube is now lying directly upon the surface of the drum.

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PE Tubes

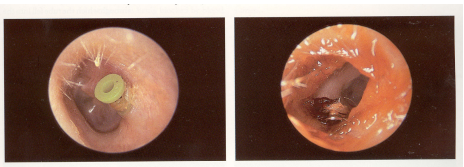

Figure 3.103 EXTRUDED VENTILATION TUBES IN EXTERNAL CANAL (left ear) The green Silastic tube seen on the *left* has been carried completely off the surface of the drum and onto the deep canal skin. When a stainless steel Reuter bobbin reaches the superficial canal skin (as seen on the *right*) it is lifted off the underlying skin by the canal hairs. Frequently, it is trapped for a period of time by the cerumen secreted in this part of the external canal.

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