MOTION SICKNESS

ETIOLOGY
- excessive stimulation of vestibular apparatus
  - individual susceptibility varies greatly.
  - aggravating factors: visual stimuli (e.g. moving horizon), poor ventilation (fumes, smoke, carbon monoxide), emotional factors (e.g. fear, anxiety).
Specific forms:
1. Sea, air, car, train, swing sickness - motion (repetitive angular and linear acceleration and deceleration) is etiologic factor.
2. Space adaptation syndrome - weightlessness (zero gravity) is etiologic factor; adaptation occurs over several days; can recur with reentry (as gravity force increases again).

SYMPTOMS & SIGNS
- cyclic nausea & vomiting
  - may be preceded by yawning, hyperventilation, salvation, pallor, profuse cold sweating, blood pressure changes, somnolence, aerophobia, dizziness, headache, general discomfort, and fatigue.
  - with prolonged exposure, patient may adapt and gradually return to well-being; symptoms may recur if motion increases or recurs after short respite.
  - prolonged motion sickness (with vomiting) may lead to dehydratio, inanition, depression.

PREVENTION AND TREATMENT
- Prevention is easier than treatment!
1) position yourself where motion is least (e.g. in middle of ship close to water level, over wings in airplanes)
2) supine or semirecumbent position with head supported is best.
3) avoid visual fixation on waves or other moving objects, avoid reading.
4) keep axis of vision at 45° angle above horizon.
5) well-ventilated cabin is important, and going out on deck for breath of fresh air helps.
6) small amounts of fluids + simple food should be consumed frequently during extended travel; if air travel is short, food and fluids should be avoided (alcohol & dietary excesses before or during travel increase likelihood of motion sickness).
7) in space adaptation syndrome, movement (which aggravates symptoms) should be avoided.
8) PROPHYLACTIC DRUGS (1 hour before departure):
   - DIMENHYDRINATE, DIPHENDRINAMINE, MECLIZINE, CYCLIZINE 50 mg po, PROMETHAZINE 25 mg po - to sedate vestibular system;
   - SCOPOLAMINE 0.6 mg po /dermal patch applied 4 & before departure can deliver smaller doses - 0.5 mg over 3 days) - minimize vagal-mediated GS symptoms;
   - DIAPZAM 5-10 mg po - to sedate vestibular system.
   - dosages should be appropriately modified for prolonged travel.
   - if vomiting occurs, drug must be given rectally / parenterally.
9) prolonged vomiting - i/v fluids & electrolytes.

MÉNIÈRE DISEASE

ETIOPATHOPHYSIOLOGY
- generalized dilation of membranous labyrinth (endolymphatic hydrops).
- prevalence: 15% (≈ 50% patients have notable family history).
- seen at almost all ages; typical onset begins at early to middle adulthood.
- cause unknown.

MÉNIÈRE DISEASE is idiopathic
MÉNIÈRE SYNDROME is secondary to specific condition (e.g. endocrine abnormalities, trauma, electrolyte imbalance, autoimmune dysfunction, medications, infections [parasitic, syphilitic], hyperlipidemia).

- relative overproduction & redistribution of endolymph → increased hydrostatic pressure within endolymphatic system.
- physical distention by increased endolymphatic pressure leads to MECHANICAL DISTURBANCE of otolithic organs (→ nonrotational vestibular symptoms) and organ of Corti (→ hearing loss & tinnitus).

VERTIGO is caused by break in membrane separating perilymph (K-rich extracellular fluid) and endolymph (K-rich intracellular fluid) → CHEMICAL MISMATCH bathes vestibular nerve receptors, leading to depolarization blockade and transient function loss (sudden change in vestibular nerve firing rate creates acute vestibular imbalance); symptoms improve after membrane is repaired and vestibular potentials are restored.

unilateral (≤ 20% bilateral).

CLINICAL FEATURES
- Recurrent prostrating VERTIGO (usually spinning sensation, sometimes violent).
  - vertigo attacks appear suddenly; last for hours (up to 24 h); subside gradually.
  - attacks are associated with nausea & vomiting, significant Nystagmus.

DIRECTION of nystagmus is not reliable indicator of lesion site - irritative phase during attack (fast phases toward involved ear), may be followed by parietic phase (fast phases toward opposite ear).

- vertigo is so sudden in onset and so intense that patients find themselves on floor as if pushed by invisible force, even from sitting position (can cause devastating accidents and falls).
INNER EAR DISORDERS

- neither loss of consciousness nor other neurologic symptoms accompany fall.
- anxiety and panic reactions are generated by fear that vertigo may occur at any moment.
- both tests – Romberg test, Fukuda marching step test.
- 3. **TINNITUS** (usually low-tone roaring) - constant or intermittent; may be worse after, or during vertigo attack.
- **AURAL FULLNESS** (feeling of discomfort, fullness, pressure in affected ear) – may be as “warning” symptom.

- disease often presents with just 1 or 2 symptoms months to years before manifesting entire tetrad.
- attacks may occur in clusters and have long symptom-free remissions.
- some patients are completely asymptomatic between episodes; however, many notice progressive deterioration of hearing and balance function with each successive attack.

**DIAGNOSIS**

- **audiogram** (gray shaded area indicates normal range; left ear (X marks) is normal at all frequencies; right ear (circles) has decreased hearing on both air and bone for lower frequencies):
- **transtympanic electrocochleography (ECoG)** can detect distortion of nerve-containing membranes of inner ear (i.e. may suggest hydrops).
- **electrocochleography (ENG)** with movement and caloric stimulation - endolymphatic hydrops causes reduced vestibular response in affected ear.

N.B. late tertiary syphilis can present with identical symptoms – always do fluorescein treponema antibody (FTA-ABS) test!

**TREATMENT**

- **VESTIBULAR SUPPRESSANTS, ANTIEMETICS** are useful during attacks. see p. Ear34

**SPECIAL PROCEDURES**

a) **endolymphatic sac decompression (± shunting)** - removing petrous bone that encases endolymph reservoir; some surgeons insert drain or valve (from endolymphatic space to mastoid or subarachnoid space) to further reduce pressure.

b) **vestibular neuroectomy** relieves vertigo and usually preserves hearing.

c) **labirintectomy** - if vertigo is sufficiently disabling + hearing has degenerated to useless level; does not require entry into cranial cavity!

d) **trans tympanic** medication perfusion (e.g. GENTAMICIN through myringotomy) – chemical labirintectomy.

e) **MENNET device** - delivers pulses of pressure to inner ear via tympanostomy tube; no one knows exactly why this works, but some patients have symptomatic relief when device is used on daily basis.

N.B. avoid destructive procedures in bilateral Ménière disease - destruction of balance portion poses high risk of destroying hearing as well!

After destructive procedures, patients may initially feel worse than ever!!! - vestibular rehabilitation is helpful.
INNER EAR DISORDERS

**Prognosis**
- In general, condition tends to **spontaneously stabilize** over time (Ménière disease is said to “burn out” over time), but patients are left with poor balance and poor hearing.

**BENIGN PAROXYSMAL POSITIONAL VERTIGO**

The most common cause of true vertigo (∼20% vertigo patients)!
First described by Barany in 1921.

**Pathophysiology**

- Dislodged otoconia become trapped in **POSTERIOR SEMICIRCULAR CANAL**.
although posterior semicircular canal is most common site of lesion, patients rarely may have horizontal or superior canal variants.

**CUPULOLITHIASIS theory**
- granular basophilic masses composed of calcium carbonate (derived from otoliths) attached (fixed) to cupula of posterior semicircular canal.
- model is analogous to situation of heavy object attached to top of pole - extra weight makes pole unstable and thus harder to keep in neutral position - pole is easily prone to "clunk" from one side to other depending on direction it is tilted; once position is reached, weight of particles keeps cupula from springing back to neutral.

**CANA LITHIASIS theory**
- free-moving densities (CANA LITHIS) in posterior semicircular CANAL.
- while head is upright, particles sit at most gravity-dependent position; when head is tilted back supine, after momentary (inertial) lag, gravity pulls particles down arc - this causes endolymph to flow away from ampulla and causes cupula to be deflected.
- model can be compared to pebbles inside tire - as tire is rolled, pebbles are picked up momentarily and then tumble down with gravity.

**ETIOLOGY**
- a) spontaneous degeneration of utricular otolithic membranes
- b) head trauma (labyrinthine concussion)
- c) otitis media
- d) ear surgery
- e) occlusion of anterior vestibular artery.

Rarely observed in individuals < 35 years without history of antecedent head trauma.
- significant proportion of children with benign paroxysmal vertigo develop migraine headaches later in life.

**CLINICAL FEATURES**
Very violent VERTIGO lasting < 30 sec (vs. in Ménière disease – lasts hours).
- induced by certain head positions - lying on one ear, lying supine, rolling over in bed, tilting head backward.
- onset is typically sudden.
- severity covers wide spectrum; in extreme cases, slightest head movement may be associated with nausea and vomiting.
- usually subsides in several weeks or months (but may recur after months or years).
- no associated hearing loss or tinnitus!

**DIAGNOSIS**
Provocative pathognomonic tests – DIX-HALLPKE maneuver, NYLEN-BÁRANY maneuver:
- classic geotropic rotatory (± upbeating) nystagmus with latency, limited duration, fatigability is pathognomonic.
- sustained / nonfatiguing nystagmus may indicate CUPULOLITHIASIS rather than CANALITHIASIS.
- negative test is meaningless except to indicate that active canalithiasis is not present at that moment.
- the most accurate way to define rotational nystagmus - clockwise / counterclockwise from patient's point of view.
- rotational nystagmus also can be described as geotropic / ageotropic:
  - Geotropic means "toward earth" and refers to upper half of eye.
  - Ageotropic refers to opposite movement
- if head is turned to right, and eye rotation is clockwise from patient's point of view (top half turns to right and toward ground), then nystagmus is geotropic.
- if head is turned toward left, then geotropic nystagmus is counterclockwise rotation.

If DIX-HALLPKE maneuver is positive, other laboratory tests are not needed to make diagnosis!!!

**Positional nystagmus may also occur with CNS lesions**
- nystagmus lacks latency, fatigability, and severe subjective sensation;
- nystagmus may continue for as long as position is maintained;
- nystagmus may be vertical or change direction, and if rotary, it is likely to be perverted (i.e. not in anticipated direction).

**TREATMENT**
- avoid provocative positions.
- medications (such as diazepam or benzodiazepines) usually are not helpful!
- most effective first choice treatment is CANALITH REPOSITIONING - office maneuver that shifts particles out of semicircular canal and into vestibule, where particles do not cause symptoms.
- two modifications - EPLEY maneuver and SEMONT maneuver
- in experienced hands, success rate > 95%.
- if lasts for ≥ 1 year → divide nerve to posterior semicircular canal.

**VESTIBULAR NEURONITIS**
- thought to be viral (as Bell’s palsy of CN7);
- frequent epidemics among adolescents and young adults;
- usually follows febrile illness;
- histological finding of axonal degeneration in vestibular nerve.
- alternative etiology - compression by arterial loop in cerebellopontine angle.
- sudden onset of severe VERTIGO (lasts for 1-2 days); self-limited.
- after attack, patients may complain of unsteadiness for weeks as vestibular system gradually accommodates.
5. that develops o-
4. ...
3. ...
1. ...

SEROUS LABYRINTHITIS
- occurs in 1 of 5000 persons every year.
- Most commonly as sequela of postmeningitic labyrinthitis.
- mostly affected is inner ear disease).
- new disorganized bone obliterates spaces within bony labyrinth.
- Most commonly as sequel of postmeningitic purulent labyrinthitis!
- mostly affected is scala tympani of basal turn.
- profound deafness + loss of vestibular function.
- Occurrence of ossification virtually guarantees that hearing will not be restored.
- documentation of oto-neurogenic is possible with high-resolution CT (HRCT; high-resolution T2 MR may also be useful.
- treatment -- steroids + cochlear implants (timing is important determinant of successful cochlear implantation!!! see p. Ear32
- SUDDEN HEARING LOSS
- - unilateral severe SENSORY/RECEPTOR HEARING LOSS (> 30 dB over 3 contiguous pure-tone frequencies) that develops over five hours.
- occurs in 1 of 5000 persons every year.
- bilateral in only 1-2% cases.

ETIOLOGIC CATEGORIES
1. Idiopathic - most cases.
2. Labyrinthine viral infection
3. Labyrinthine vascular compromise (embolism, thrombosis, hemorrhage); cochlea is end organ with no collateralvasculature.
4. Intracochlear membrane ruptures
5. Immune-mediated inner ear disease - rapidly progressive, often fluctuating, bilateral hearing loss over weeks to months (generally, too slow to classify as sudden hearing loss); most important diagnostic finding is improvement in hearing observed with trial of immunosuppressants.

VASCULAR DISORDERS
- Viral infections can cause congenital (rubella, CMV) and acquired (mumps, measles) hearing loss.
- Vascular disorders (such as labyrinthine artery infarction) are generally impossible to exclude, and their diagnosis is suggested by identical symptom complex combined with vascular risk factors.

PURULENT LABYRINTHITIS
- secondary to bacterial infections:
  a) ACUTE OTITIS MEDIA (via oval and round windows or dehiscent horizontal semicircular canal [result of erosion by cholesterol]) -- typically unilateral.
  b) PURULENT MENINGITIS (via cochlear aqueduct or internal auditory canal) -- typically bilateral, and vestibular symptoms are present in 20% children with meningitis! Streptococcus pneumoniae, Neisseria meningitidis are agents most likely associated with hearing loss.
- clinical features:
  1) SEVERE VERTIGO and tinnitus.
  2) complete & permanent HEARING LOSS.
- complications:
  a) facial paralysis
  b) meningitis (can gain access to subarachnoid space through cochlear aqueduct).
  c) chronic otitis media and choledestoma
  d) labyrinthitis ossificans (decisions regarding cochlear implantation must be made early!)
  e) cochlea, vestibule, and semicircular canals enhance on T1-weighted MRI postcontrast images in acute labyrinthitis.

TREATMENT
- IV antibiotics appropriate for meningitis.
- steroids in meningogenic hearing loss are controversial.
- rarely - labyrinthectomy (to drain inner ear) or radical mastoidectomy.

SEROUS LABYRINTHITIS
- occurs when bacterial toxins & host inflammatory mediators (cytokines, enzymes, complement) cross round window membrane → labyrinthitis in absence of direct bacterial contamination.
- associated with acute / chronic middle ear disease (one of most common complications of otitis media!!!).
- audiogram reveals mixed hearing loss when middle ear effusion is present.
- vestibular symptoms are less common.
- treatment - eliminating infection + clearing middle ear effusion.

LABYRINTHITIS OSSIFICANS
- in various pathologies (e.g. Paget disease of bone, osteopetrosis, otosclerosis, temporal bone trauma, inflammatory and infectious conditions, obstruction of labyrinthine artery, autoimmune inner ear disease, new disorganized bone obliterates spaces within bony labyrinth.
- Most commonly as sequel of postmeningitic purulent labyrinthitis!
- mostly affected is scala tympani of basal turn.
- profound deafness + loss of vestibular function.
- Occurrence of ossification virtually guarantees that hearing will not be restored.
- documentation of oto-neurogenic is possible with high-resolution CT (HRCT; high-resolution T2 MR may also be useful.
- treatment -- steroids + cochlear implants (timing is important determinant of successful cochlear implantation!!! see p. Ear32

VIRAL LABYRINTHITIS
- most common form of labyrinthitis; rare in children.
- upper respiratory tract infection precedes onset in 50% cases.
- sudden unilateral loss of VESTIBULAR FUNCTION + HEARING (vs. vestibular neuritis)
- hearing loss may be presenting symptom in many patients.
  - mild to moderate (vs. purulent labyrinthitis -- profound hearing loss);
  - typically in high frequencies.
  - severe, often incapacitating, VERTIGO is characteristic (patient is often bedridden!)
  - spontaneous nystagmus towards unaffected side; diminished / absent caloric responses in affected ear.
- eventually resolves after several days to weeks; return of hearing usually mirrors return of vestibular function (unsteadiness and positional vertigo may persist for several months).
- recurrent attacks are rare (may be complicated with Ménière disease).
- treatment is similar to vestibular neuritis; short course of oral corticosteroids may be helpful.

Specific Forms
Herpes zoster oticus (e.g. Ramsay-Hunt syndrome) - auditory & vestibular symptoms develop in ~ 25% patients; see p. 256 (8) !! Viral infections can cause congenital (rubella, CMV) and acquired (mumps, measles) hearing loss.

- Vestibular symptoms are rare (may be confused with Ménière disease).
- treatment is similar to vestibular neuritis; short course of oral corticosteroids may be helpful.
Hearing loss from sustained exposure
coexisting systemic autoimmune disease occurs in 15-30% of patients (RA, ulcerative colitis, SLE, polyarteritis nodosa).
6. Tumors
( internal auditory canal, cerebellopontine angle).
• prognosis for some recovery of hearing is good (usually within 10-14 days).
• tinnitus & vertigo may be present initially (usually subsides in several days).

TREATMENT
1. Otologic emergency!
• in idiopathic cases no form of treatment is of proven value; try: 1) bed rest.
2) corticosteroids, appear rational!!!
3) vasodilators (PAPAVERINE, HISTAMINE, NICOTINE acid, PROCAINE, NAACL, CARBONI
[5% carbon dioxide] - improve blood supply to cochlea, reversing hypoxia.
4) rheologic agents (LOW MOLECULAR WEIGHT DEXTROSE, PENTOXYFLINE, ANTIAGGREGANTS) - better oxygen delivery might be achieved.
5) hyperbaric oxygen.
6) antiviral agents (ACYCLOVIR, AMANTADINE, FAMECYCLOVIR, VALACYCLOVIR).
7) duralectomy - used under assumption that some episodes are secondary to endolymphatic hydrops.
8) triiodohydroxy acid derivatives (e.g. DIAZEPAM, MEGILMINE - angiographic contrast agent) - affect stria vasculaies and assist in maintaining endolymphatic potential.

PERILYMPHATIC (S. LABYRINTHINE) FISTULA
• abnormal communication between perilymphatic space and middle ear
• perilymph leakage → secondary endolymphatic hydrops (as in Ménière disease).

ETIOLOGY
a) destruction of horizontal semicircular canal by CHOLESTEATOMA.
b) rupture of round / oval window membrane:
• in everyday activities (lifting, straining, coughing, sneezing) increases in CSF pressure can be transmitted to inner ear via patent cochlear aqueduct or through lamina cribrosa of internal auditory canal → precipitous increase in pressure within INNER EAR.
• pressure can increase rapidly within MIDDLE EAR as result of barometric pressure change, scuba diving, compression trauma of ear, Valsalva maneuver, pinched-nose sneezing.
• following stapedectomy
head injury!!!
• congenital anomalies

CLINICAL FEATURES
1. Sudden severe HEARING LOSS (90%), loud roaring TINNITUS (63%), AURAL FULLNESS (25%)
• patient may hear explosive sound in affected ear when fistula occurs.
2. Severe rotational incapacitating VERTIGO (> 80%)

DIAGNOSIS
Accurate diagnosis is difficult! - there are no reliable tests to determine presence of fistula!
Obtain gadolinium-enhanced MRI in all individuals with unilateral otologic symptoms!
Most important differential – Ménière disease!
1. FISTULA TEST (via tymanometry with electroxystagmography) - application of positive pressure to tympanic membrane produces nystagmus (nystagmus direction depends on fistula site).
2. Dynamic platform posturography: pressure is applied to external auditory canal; increase or decrease in pressure is transmitted to tympanic membrane middle ear space and, if fistula is present, to inner ear → abnormal sway is generated by these pressure changes, positive results give high likelihood of having fistula.
3. Elevated SP/AP ratio on electrocochleography (ECoG)
4. Middle ear endoscopy
5. EXPLORATORY TYMPSANGTOMY - definitive diagnosis!
• some surgeons elect surgical exploration even when likelihood of fistula is low.
• other surgeons use fairly rigid criteria.

TREATMENT
Medical therapy: sedation, bed rest, elevation of head of bed, stool softeners, avoidance of Valsalva maneuver.

Surgical treatment
Fistula grafting is definitive treatment (highly effective for vestibular symptomatology, but its effect on hearing loss is less predictable).
• round and oval window niche must be carefully observed for accumulation of clear fluid.
• β2-transferrin is specific for human aqueous humor, CSF, and perilimph; time required to perform tests prevents its intraoperative use, preservative irrigation of middle ear via mycetology can obtain dilute perilymph that could be tested by Western blot analysis for presence of β2-transferrin.
• grafting is performed by removing mucosa of round and oval window area; autogenous grafts (fascia or periichondium) are placed directly over leak (if no actual leak is identified, footplate and round window are grafted prophylactically).
• postoperatively avoid increasing ICP (heavy lifting, straining, placing head in dependent position).

NOISE-INDUCED HEARING LOSS
Any intense noise may damage inner ear (loss of hair cells in organ of Corti - sensory hearing loss).

a) OCCUPATIONAL NOISE-INDUCED HEARING LOSS – if noise is present in workplace.

b) SOCIOAUSIS - if noise is associated with recreational (nonoccupational) activities (mostly firearm noise); produce minor losses because exposure times are short.
• 1.8% American males have handicapping NHB.
• PRESCRIPTIONS is due to physiologic aging process ± lifetime of socioacusis.

PATHOPHYSIOLOGY
Hearing loss from sustained exposure
• high frequencies are much more damaging than low frequencies at same physical intensity levels - most sound level meters are equipped with filter that de-emphasizes frequencies to which human ear is less sensitive (filter is called A filter, and measurements are reported as dBA).
INNER EAR DISORDERS

pure continuous stimuli are more damaging than interrupted stimuli.

any noise > 85 dB is damaging (OSHA has determined that exposure to < 85 dBA continuously for an 8-hour workday is unlikely to cause harm).

changes in organ of Corti range: distorted stereocilia of inner and outer hair cells + complete absence of organ of Corti and rupture of Reissner membrane.

– OUTER HAIR CELLS are more susceptible (than inner hair cells).

– temporary threshold shifts (recover almost completely within 24 hours once noxious stimulus is removed) are due to:
  1) MECHANICAL trauma → decreased stiffness of stereocilia (floppy stereocilia respond poorly).
  2) METABOLIC exhaustion (exhaustion of recovery, but if prolonged may cause cell death) → permanent threshold shifts are associated with fusion of adjacent stereocilia and loss of stereocilia.

– repeated temporary threshold shift slowly converts to permanent threshold shift

– death of sensory cell leads to Wallerian degeneration → loss of primary auditory nerve fibers.

Acoustic trauma - one-time brief exposures followed by immediate permanent hearing loss.

– sound stimuli generally exceed 140 dBA and are often < 0.2 seconds duration.

– 2 types: impulse noise (result of explosion) and impact noise (results from collision; usually metal on metal).

– MECHANICAL tearing of membranes and physical disruption of cell walls with mixing of perilymph and endolymph; acoustic trauma can result in disruption of tympanic membrane and ossicular injury.

CLINICAL FEATURES

– persons vary greatly in susceptibility (≥ 10 years of exposure is generally required for significant hearing loss to occur).

– high-frequency tinnitus accompanies hearing loss.

– symmetrical bilateral loss (permanent threshold shift) first occurs at 4 kHz (characteristic notch centered around 4000 Hz on audiogram) = gradually occurs in higher frequencies as exposure continues, and eventually in lower frequencies.

– N.B. loss is less at 8 kHz than at 4 kHz (vs. in most sensorineural hearing losses).

– conversation on telephone is unimpaired (telephones do not use frequencies > 3 kHz).

– as severity of hearing loss increases, rate of progression decreases (provided injurious stimulus remains constant).

– hearing loss does not progress if exposure is eliminated.

– no method of treatment is available, and no recovery is expected once permanent threshold shift has occurred.

– if hearing loss interferes with communication, hearing aid is helpful.

PREVENTION

1) limiting length of exposure

2) reducing noise at its source

3) ear protectors

– when exposure is continuous, injury is consequence of total amount of energy to which cochlear tissues are exposed (e.g. if sound energy is doubled [i.e. increase 5 dB], risk of injury can be kept constant if exposure time is cut roughly in half).

– if noise levels cannot be brought down to PEL, hearing protection must be provided.

– duration of 16 hours, 85 dBA

– duration of 6 hours, 90 dBA

– duration of 2 hours, 100 dBA

– if noise levels cannot be brought down to PEL, hearing protection must be provided.

Fig. Safe weekly exposure times to noise


Worker Compensation

Pure-time threshold curve in noise-induced hearing loss. A notch is typically seen at 2-4 kHz, meaning that the hearing threshold is better at 5 kHz than at 4 kHz. The hearing thresholds at an "early" stage are shown in light red, and the later configuration with a flatter notch is shown in darker red.


Relationship between noise exposure measured in dBA and the exposure time that can cause measurable hearing loss in less than 5% of normal-hearing subjects. Because the sound level is measured logarithmically, an increase of 1 dBA corresponds to a 50% reduction of exposure time.


Worker Compensation
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EAR
DISORDERS

American Academy of Otolaryngology (committee on hearing & equilibrium) has established computational method to determine hearing handicap:
- it recognizes that hearing thresholds of 500, 1000, 2000, and 3000 Hz are critical for understanding human conversation, and it uses only those frequencies in calculation of hearing handicap.
- method also recognizes that overall hearing acuity is disproportionately determined by acuity of better-hearing ear (calculation heavily favors better-hearing over poorer-hearing ear).
- method recognizes 25 dB threshold or better to be normal.
- computation is performed as follows:
  - compute sum of hearing threshold levels at 500, 1000, 2000, and 3000 Hz and divide by 4; subtract 25 from average; multiply result by 1.5 to produce percent monaural hearing impairment.
  - if monaural percent figure is same for both ears, that figure expresses percent handicap; if percent monaural hearing impairment is different between ears, apply formula (5 x % of better ear) + (1 x % poorer ear) divided by 6 = percent hearing handicap.

PRESBYCUSIS - gradual bilateral sensorineural hearing loss that occurs in people as they age.
- exact cause is still not known today.
- related in part to lifetime noise exposure, arteriosclerosis, diabetes.
- histologic change occurs throughout auditory system - from hair cells to auditory cortex:
  1) thickening and secondary stiffening of basilar membrane
  2) gradual cumulative epithelial atrophy (loss of hair cells)
  3) atrophy of stria vasculare
  4) atrophy of neurons in cochlea and central neural pathways.

CLINICAL FEATURES
- begins after age 20; becomes significant only in > 65 (25-30% people 65-74 years are impaired; for people aged ≥ 75 years - 40-50%).
- first affects highest frequencies (18-20 kHz); associated with difficulty in speech discrimination and central auditory processing of information (esp. when background noise is present) – patients complain that others mumble.
- gradually affects lower frequencies (begins to affect 4-8 kHz range by age 55-65).
- variation - some persons are severely handicapped by age 60, and some are essentially untouched at age 90.

TREATMENT
1. Speech reading (lipreading), use of nonauditory clues.
2. Hearing aid with more gain in higher frequencies.
3. Some patients (with relatively intact spiral ganglia and central pathways) benefit from cochlear implants.

DRUG-INDUCED OTOTOXICITY (≥ TOXIC LABYRINTHITIS)
1) high-frequency sensorineural hearing loss (above 8000 Hz upper limit of standard audiogram!!!) and high-pitched tinnitus.
2) positional nystagmus, dysequilibrium and oscillopsia.

- drugs affect both auditory and vestibular portions of inner ear, but particularly toxic to organ of Corti (cochleotoxic).
- many agents produce hair cell damage.
- bilaterally symmetrical, but also can be asymmetrical!
- time of onset is unpredictable; – marked hearing loss can occur even after single dose; – hearing loss may not manifest until several months after therapy completion.
- hearing loss is usually irreversible with most agents.

1. Aminoglycosides - ototoxicity is likely multifactorial (most promising mechanism involves iron chelation leading to production of free-radical complex).
   - streptomycin, gentamicin, tobramycin are primarily vestibulotoxic.
   - amikacin, neomycin, kanamycin, are primarily cochleotoxic
   N.B. neomycin is most cochleotoxic antibiotic!
   N.B. safest aminoglycoside is netilmicin!
2. Other antibiotics
   vancomycin causes reversible ototoxicity.
3. **Salicylates** (in high doses) - reversible tinnitus (!!) and hearing loss (cochleotoxic).

4. **Quinine and its synthetic substitutes** - can cause reversible hearing loss (cochleotoxic).

5. **Loop diuretics** (ethacrynic acid, furosemide, bumetanide) – probably affect stria vasculosa; reversible cochleotoxicity.

6. **Antineoplastic agents**, esp. platinum-based compounds (cisplatin > carboplatin) - irreversible severe hearing loss (cochleotoxic).

**PRECAUTIONS**

Avoid ototoxic drugs in:

1. **renal failure** - nearly all are eliminated through kidneys – monitor [drug], adjust dosage.
2. do not use as topical medication for ear when tympanic membrane is perforated (can be absorbed into inner ear fluids).
3. **pregnancy**
4. **elderly persons**
5. **persons with preexisting hearing loss**.

- throughout therapy, perform baseline hearing evaluations (to document preexisting hearing loss) followed by periodic testing.
- avoid noise exposure (incl. high hearing aid output) for 6 months after therapy completion.

**SUPERFICIAL SIDEROSIS**

- uncommon disorder caused by chronic slow or repeated bleeding into subarachnoid space
- **cardinal features** (occur in approximately 90% of cases):
  1) deafness
  2) cerebellar ataxia
- **diagnosis**
  - MR / T2 hypointensity enveloping surface of brainstem, cerebellum, and sometimes cortical fissures.
  - lumbar puncture - bloody / xanthochromic CSF
  - bleeding site goes unidentified in half the cases.
- **treatment** – CSF leak repair (if one is present)
- **prognosis** - progression to a bed-bound state may occur over decades in up to one-fourth of these patients.

Axial T2-weighted (A through D) and proton density (E) brain MRI - signal hypointensity due to hemosiderin deposition around the medulla and cerebellar vermis (A), along the cerebral convexities (B), around the midbrain (C), around the pons and along the cerebellum folia (D), and along the interhemispheric and sylvian fissures (E). Sagittal T1-weighted (F) and sagittal T2-weighted (G, H) spine MRI in Cases 7 (F, H) and 15 (G), showing hemosiderin deposition along the cervical cord (F), thoracic cord (G), and the roots of the cauda equina (H). Axial T2 (I, J) spinal MRI in Cases 15 (I) and 7 (J), showing hemosiderin deposition around the cord (I) and along the cauda, resulting in nerve root clumping suggestive of arachnoiditis (J). Also presented is a CT showing a rim of hyperdensity due to hemosiderin around the brainstem in Case 7 (K).

**BIBLIOGRAPHY** for ch. “Otology” – follow this [LINK >>]

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