Tinnitus, Vertigo, Earache

**TUNITUS**

- subjective sensation of noise in ear (constant nonmusical tone described as ringing, whistling, hissing, humming, roaring; may involve more complex sounds that vary over time).

**VERTIGO (DIZZINESS)**

- abnormal sensation of movement (usually rotary). Subjective vertigo – patient feels as if environment is moving in relation to him.

**SUBJECTIVE TINNITUS**
- audible only to affected individual.
  - mechanism: causing tinnitus is obscure.
  - as symptom may occur in nearly all ear disorders (85% patients presenting with ear-related symptoms report experiencing tinnitus); most commonly:
    1. disorders of cochlea / CNS, often associated with hearing loss
    2. transient episodes occur in most individuals and are not associated with disease (e.g. after exposure to loud concert).
    3. side effect of NSAIDs (e.g. 3000 mg of aspirin may produce tinnitus in some persons), loop diuretics, aminoglycosides.

**OBJECTIVE TINNITUS**
- audible to anyone in addition to affected individual.
  - relatively rare.
  - caused by something creating unusually loud disturbance about ear.
  - primary etiologies
    - A. Muscular tinnitus: degenerative diseases of head and neck (incl. amyotrophic lateral sclerosis) - loss of neuromuscular control over muscles in ear – repetitive flutter / myosinus of stapedius / tensor tympani muscles;
    - H: lysis of tensor / stapedius muscles via tympanotomy - uniformly successful, performed bilaterally (this is one of few cases in otology where operating on both sides at same time makes sense).
    - B. Vascular tinnitus:
      - a) aberrant or abnormal (ectatic, tortuous) carotid artery – tinnitus can be auscultated with each heartbeat;
      - b) abnormal jugular bulb and jugular vein (venous hum). H: many operations described for treatment of venous hum and carotid arterial tinnitus, all of these operations initially met with success but limited long-term control of symptom.

**SYMPTOMATIC TREATMENT**
- treatment is directed toward CAUSE ELIMINATION.
  - Be sure to clean ear canal of wax (frequent cause of tinnitus).
  - ability to tolerate tinnitus varies among patients.

**TREATMENT**
- treatment is frequently therapy that is helpful to one person is not helpful to next! - many have adopted philosophical outlook that tinnitus is chronic or psychologic disease and is treated with and not cured.
  - 1) correcting associated hearing loss (e.g. hearing aid) usually suppresses tinnitus.
  - 2) many patients find relief by playing background music to mask tinnitus and may go to sleep with radio playing.
  - 3) some benefit from TINNITUS MASKER (worn like hearing aid) - presents sound more pleasant than tinnitus (deliver constant low level white noise).
  - 4) electrical stimulation of inner ear (e.g. cochlear implant), occasionally reduces tinnitus but is appropriate only for profoundly deaf.
  - 5) antidepressants (esp. NORCYPYLLINE, Paxil)
  - 6) NASICS
  - 7) biofeedback (goal is to decrease stress and anxiety that may be contributing to tinnitus).
  - 8) support groups, American Tinnitus Association (PO Box 5, Portland, OR, 97207).
  - 9) extract of GINKGO BILOBA (goal is to decrease stress and anxiety that may be contributing to tinnitus).
  - 10) homeopathic therapy, acupuncture (strong placebo effect)
  - 11) section of cochlear nerve (effective in only 25%)

**EAR34**

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DIAGNOSTIC ALGORITHM

4.3.1

CENTRAL VERTIGO

- Vertigo is more severe (most severe with CNS transaction – ablative vertigo) associated with nausea & vomiting (esp. if onset is acute), aggravated by positional changes.
- Vertigo may be PAROXYSMAL.
- Tinnitus, hearing loss are common.
- Diseases of semicircular canal neurons → rotational vertigo.
- Diseases of utricle / sacule → sensations of tilting or listing (as on boat).
- Often accompanied by jerk nystagmus.
  - Unidirectional horizontal or rotary (never vertical).
  - Slow phase toward lesion side.
  - Can be inhibited by ocular fixation.
  - Fatigues over time.
- Symmetric bilateral lesions → same gaze directions; nystagmus is nonfatigable, nystagmus associated with other findings of CNS dysfunction.
- Vertigo is usually symmetric bilateral lesions often accompanied by tinnitus, hearing loss.
- Vertigo may be positional.

PHYSIOLOGIC VERTIGO

- Caused by abnormal stimulation of vestibular apparatus; accompanying signs & symptoms:
  - 1) Nystagmus
  - 2) Difficulty in balance - post-panting, ataxic gait & falling
  - 3) Autonomic responses (nausea & vomiting, sweating, hypotension, bradycardia).
- Vertigo is aggravated by movement and improved by remaining stationary.
- Lesion side (equivalent to direction of experimental endolymph movement) is side to which:
  - Slow component of nystagmus moves;
  - Post-panting and falling occurs;
  - Hallucination of environment movement is felt (hallucination of subject movement is in opposite direction).

PHYSIOLOGIC VERTIGO

a) Brain is confronted with mismatch among three stabilizing sensory systems (vestibular system, visual system, somatosensory system) – e.g. carsickness, height vertigo, vertical vertigo (motion picture chase scenes, incorrect spectacles, acute diplopia).
- Vestibular system is subjected to unfamiliar head movements to which it has never adapted (such as in motion sickness).
- Unusual head/neck position (such as extreme extension when painting ceiling).

CNS compensation rapidly counteracts vertigo!

PERIPHERAL VERTIGO - disease of labyrinth / vestibular nerve.

- Vertigo is more severe, not positional related.
- Vertigo is constant.
- Associated with other findings of CNS dysfunction.
- Nystagmus may be present in vertical, or multiple directions of gaze (may change direction in different gaze directions); nystagmus is not fatigueable, not inhibited by ocular fixation (visual fixation even may enhance nystagmus).
- Differentiating between central and peripheral vertigo (via oculomotor examination) – features of central vertigo:
  1) Spontaneous nystagmus that cannot be suppressed with visual fixation.
  2) Nystagmus that changes direction with gaze.
  3) Purely vertical, horizontal, or torsional nystagmus.
  4) Saccadic disocoria (overshoot / undershoot).
- Most common causes:
  - Cerebrovascular disease (e.g. involving vertebrobasilar circulation, which supplies labyrinth, lateral pontomedullary region containing vestibular nuclei, and cerebellum); e.g. Wallenberg syndrome.
  - Vertebrobasilar migraine.
  - Traumas (temporal lobe, cerebellum, brainstem, cerebellopontine angle).
  - Vertical nystagmus that changes direction of experiment endolymph movement (equivalent to vertebrobasilar migraine).
  - Associated with hearing loss, vertigo.
  - Head trauma (± labyrinth concussion).
- Psychogenic vertigo – nystagmus is absent during episode!

DIAGNOSIS

1. Determine meaning of “dizziness”, PROVOCATIVE TESTS (to provoke symptoms that patient can recognize as his own complaint):
   - a) Cephalic ischemia - orthostatic hypotension with Valsalva maneuver (decreases cerebral blood flow), hypertension.
   - b) Vestibular dysfunction - rapid rotation and abrupt cessation of movement in swivel chair; vigorous head shaking in horizontal plane (patient with Frenzel test).

2. Vestibular function should be evaluated see p. D.4b >>
   - Vestibular evaluation may reveal causal paresis, unilateral absence of sensitivity, directional preponderance, relative exaggeration of nystagmic response in one direction (e.g. acoustic neuromas frequently cause canal paresis or no response on side w neoplasm).

3. Minimum comprehensive audiologic assessment

4. MRI with gadolinium

DIAGNOSTIC ALGORITHM

Exertional vertigo, vestibular neuritis, labyrinthitis, vascular disorders

Yes

Sudden Onset?

Yes

Is it episodic?

No

Yes

Is positional?

No

Acoustic neurinoma, other cerebellopontine angle tumor

Yes

Associated with past fainting?

No

Benign positional vertigo, cervical osteophytes

No

Vertebrobasilar insufficiency, sleep apnea, hyperlipidemia, migraines

Yes

Yes

Psychosomatosis, hypertension, hyperthyroidism, hysteria, glycosuria, lymphoma, blood dyscrasia

No
**TREATMENT**

1) **BED REST**: if vertigo persists beyond few days, most authorities advise ambulation (in attempt to induce central compensatory mechanisms) despite short-term discomfiture to patient.

2) **FLUID REPLACEMENT** for intractable vomiting.

3) **VESTIBULAR SUPPRESANTS**:
   a) **antihistamines** (e.g. *DIPHENHYDRAMINE*, *MECLIZINE*, *CYCLIZINE*) - decrease excitability of labyrinth and block conduction in vestibular-cerebellar pathways - sedate vestibular system.
   b) **benzodiazepines** (e.g. *LORAZEPAM*, *DIAZEPAM*, *ALPRAZOLAM*) - facilitate inhibitory GABA neurotransmission; particularly effective in relieving distress of severe vertigo by sedating vestibular system. 
   c) **anticholinergics** (e.g. oral *SCOPOLAMINE* and *ATROPINE* in OTC preparations, transdermal *SCOPOLAMINE*, oral *GLYCOPYRROLATE*) - work centrally by suppressing conduction in vestibular cerebellar pathways - minimize vagal-mediated GI symptoms.
   d) **barbiturates** (e.g. *PENTOBARBITAL*) - to provide general sedation.
   e) **monoaminergics** (*EPHEDRINE*) - treat vertigo, possibly through modulating sympathetic system.

4) **ANTIEMETICS**: *DROPERIDOL*, *MECLIZINE*, *METOCLOPRAMIDE*, *ONDANSETRON*, *PROCHLORPERAZINE*, *PROMETHAZINE*, *TRIMETHOBENZAMIDE*, *THIETHYLPERAZINE*.

5) **VESTIBULAR REHABILITATION** - specific form of physical therapy that takes advantage of brain plasticity to increase sensitivity and restore symmetry.
   - designed as therapist-directed patient-motivated home-based exercise protocol.
   - optimal candidates have:
     1) stable vestibular deficits
     2) symptoms that are provoked by specific activities or stimuli
     3) intact cognitive, cerebellar, visual, and proprioceptive systems.

6) **HERBS**: *ginkgo biloba* (must be highly purified extract: 24% ginkgo flavonoids / ginkgo glycosides), *ginseng*, *blessed thistle*, *hawthorne*, *gotukola*, *VERTIGOHEEL* or *COCCULUS COMPOSITUM*.

**EARACHE (OTALGIA)**

A. **Trauma, infection, obstruction, neoplasms in external or middle ear**
   - even mild inflammation in ear canal produces severe pain
   - most common cause of earache in children - acute otitis media.

B. **Referred to ear from remote disease processes** (via CNS, 7, 9, 10 - all subserve sensation in external & middle ear): nose, para nasal sinuses, nasopharynx, teeth, gingiva, temporomandibular joint, mandible, parotid glands, tongue, palate tonsils, pharynx, larynx, trachea, esophagus.
   - most often, carcinoma of nasopharynx.

**BIBLIOGRAPHY** for ch. “Otology” → follow this LINK >>

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