

## Lecture 21 -- Vestibular System – M. Goldberg

### I. The vestibular labyrinth senses

- A. Head angular acceleration (semicircular canals)
- B. Head linear acceleration (saccul and utricle)
  - 1. Translational motion.
  - 2. Gravity (and, by extension, head tilt).

### II. The vestibular organ lies in the temporal bone

III. Each vestibular organ has a sensor for head acceleration, driven by hair cells similar to those in the cochlea. In the cochlea, vibration induced by sound deforms the hair cells; in the labyrinth, pressure of endolymph induced by acceleration deforms the hair cells. Deformation of the stereocilia towards the kinocilium causes hyperpolarization.

IV. In the semicircular canals the sensing organ is the ampulla. When the head moves in the plane of a canal endolymph lags, causing pressure against the hair cells. This pressure is proportional to head acceleration.

V. The three semicircular canals lie in 3 orthogonal planes. The semicircular canals are functionally paired and sense rotation:

- A. Horizontal canals: sense rotation in the horizontal plane
- B. Left anterior and right posterior canals (LARP) sense rotation in the vertical plane skewed 45° anteriorly to the left.
- C. Right anterior and left posterior canals (RALP) sense rotation in the vertical plane skewed 45° anteriorly to the right.

VI. The semicircular canals are functionally paired. The canals lie in roughly the same planes as the extraocular muscles:

- A. Horizontal canals: lateral and medial recti.
- B. LARP: left vertical recti, right obliques.
- C. RALP: right vertical recti, left obliques.
- D. Each canal excites a pair of muscles and inhibits a pair of muscles in its plane. Its partner excites the muscles it inhibits, and vice-versa.

V. The otolith organs sense linear acceleration.

- A. Hair cells lie in the macula.
- B. The saccul senses acceleration in the sagittal vertical plane: up and down (so it senses gravity) and forward and backward. Mnemonic: Saccul - Sagittal
- C. The utricle senses acceleration in the horizontal plane:

VI. The signals in the vestibular nerve: Although the cupula senses acceleration, the canal signal in the vestibular nerve is a tonic signal, deviations from which are proportional to head velocity. The macular (otolith) afferents have a tonic signal, deviations from which are proportional to acceleration.

VII. There are 3 major vestibular reflexes

- A. Vestibulo-ocular reflex – keep the eyes still in space when the head moves.
- B. Vestibulo-colic reflex – keeps the head still in space – or on a level plane when you walk.
- C. Vestibular-spinal reflex – adjusts posture for rapid changes in position.

VIII. The rotational VOR keeps the eye still when the head rotates, and uses the canal signal.

IX. The translational VOR keeps the eyes still when the head moves laterally and uses the otolith signal (for example when you are looking out of the window of the A train and trying to read the name of the station past which you are traveling). Gain of the translational VOR is dependent on viewing distance: during translation a far object moves less on the retina than a near object. The rotational VOR is not dependent upon viewing distance. Most head movement evokes a combination of the rotational (canal) and translation (otolith) VOR's.

X. The VOR is plastic

- A. It can be suppressed when you don't want it, for example if you are looking at something that is moving with you.
- B. Its gain can change.
  1. How do you know if the VOR is doing a good job?. There is no motion on the retina when the head moves.
  2. If a muscle is weakened, a given central signal will be inadequate, and the world will move on the retina.
  3. This can be mimicked by spectacles that increase retinal slip.
  4. In either case, the brain adjusts the VOR signal so the retinal slip is eliminated.
  5. The cerebellum is necessary for both suppression of the VOR and for slip-induced gain change.

XI. Vestibular nystagmus is a combination of slow phase eye movements evoked by the VOR, and quick phase resetting eye movements.

XII. The vestibular system is imperfect.

- A. The cupula habituates in 5 seconds. The brainstem and cerebellum extend this time to roughly 25 seconds, after which there is no further response to constant head acceleration.
- B. The vestibular system is a poor transducer of very slow ( $<0.1\text{Hz}$ ) rotation.
- C. The visual system compensates for the inadequacies of the vestibular signal by providing a description of the retinal motion evoked by the head movement.
- D. The optokinetic response is mediated by neurons in the accessory optic system in the pretectum, and the motion-sensitive areas in the cortex (MT and MST).
- E. The vestibular nucleus combines visual and vestibular signals

XII. Visual-vestibular conflict: Full-field stimulation is an effective stimulus for the vestibular nucleus. The neurons can't tell the difference, nor can you! Ordinarily the head movement implied by the visual and vestibular signals are equal. Motion sickness – nausea and vomiting – occurs when the visual and vestibular signals are unequal.

XIII. Sequelae of peripheral vestibular dysfunction

- A. Vertigo and nystagmus: The vestibular system has a tonic signal, changes of which are interpreted as head motion. Anything that deranges that signal causes vertigo, a perception of head motion when the head is still. This may be associated with visuovestibular conflict, nausea, and vomiting.
- B. Head tilt.
- C. Difficulty compensating for perturbations of head position – functional imbalance.
- D. Difficulty with path integration.

XIV. Peripheral causes of vertigo and nystagmus

- A. Benign positional vertigo: debris from the otoconia in the utricle float into the posterior canal, causing interference with cupula function, brought out by motion in the plane of the affected posterior canal. This can be treated by the Epley maneuver, that rotates the head to float the debris away.
- B. Acute viral labyrinthitis.
- C. Alcohol – alcohol is lighter than blood, so the hair cells float in the endolymph.

XV. Central causes of vertigo and nystagmus.

- A. Vestibular nucleus and other brainstem lesions.
- B. Cerebellar lesions.
- C. Peripherally caused nystagmus is worse with the eyes closed, because the normal cerebellum can use vision to suppress the nystagmus.

XVI. Cortical vestibular function. The vestibular nuclei project to the ventral thalamus (VP/VL) and thence to area 2v. A number of cortical areas have vestibular responses, but cortical vestibular processing is poorly understood. The human analog lies in the parietoinsular cortex.

XVII. Perceptual aspects of vestibular function:

- A. Awareness of self-motion.
- B. Vertical orientation.
- C. Patients with lesions of parietoinsular cortex have difficulty perceiving the vertical: they think vertical lines tilt towards the side of the lesion.