GAZE STABILIZATION SYSTEMS

Vestibular Ocular Reflex (VOR)

Purpose of VOR

Chief function is to stabilize gaze during locomotion. Acuity declines if slip exceeds 3-5 deg/sec. Ex: Head bobbing and heel strike during running.

Orient eyes to gravity during sustained head tilt.

Measurement of VOR

Usually search coil or EOG.

Measured experimentally with moving chair to avoid neck proprioception, usually either with sustained rotation or sinusoidal oscillation.

Measured clinically with **Caloric test** for vestibular function.

Patient is tilted back 60 degrees to make lateral canal vertical.

Water is injected in ear canal, causing convection currents in lateral semicircular canal.

Cold **O**pposite **W**arm **S**ame (jerk direction)

Dynamics of the VOR

Phases of VOR

Slow phases cancel the head rotation to keep the gaze steady. Most of VOR in natural situations is slow phase.

Fast phases are saccades which recenter the eye if slow phase carries it too far over. They will usually carry the eye past primary position to anticipate continued motion.

Comatose patients can show slow phase without fast; eye travels to an extreme and stays there.

Latency (time from stimulus onset to response onset) is very short (16 msec, compared to about 75 msec for visually driven movement).

Frequency response with sinusoidal oscillation: VOR **gain** (eye velocity / head velocity) falls off for frequencies below 0.01 Hz. The upper limit is not precisely known, due to measurement limitations. VOR **phase lag** (delay of eye relative to head) is zero above about 0.1 Hz. Where gain is 1.0 and phase lag is 0.0, the VOR is perfectly compensating for head movement.

Persistance in the dark: VOR persists for several seconds during sustained rotation in the dark, then eventually decays to zero. We describe this with a **time constant** (time taken for velocity to fall to 37% of initial value). The time constant for VOR is about 15 seconds. The time constant for the movement of endolymph is about 6 seconds. The difference represents a central mechanism referred to as **velocity storage**.

After-nystagmus

Motion continues in semicircular canals.

Velocity storage mechanism also contributes

When OKN and VOR are both active (rotation in the light), there is little or no afternystagmus due to cancellation by aftereffects of VOR and OKN.

Suppression of VOR

Sometimes you don't want your eyes to compensate for head rotation, as for example during pursuit.

Pursuit system can produce cancelling signal.

Attention on stationary world after rotation can discharge velocity storage.

Attention and arousal level in dark also affects gain of VOR. Being alert and trying to look forward enhances response.

Adaptability of VOR

Gain of VOR is calibrated by visual feedback. Ex: Switching from spectacles to contacts.

VOR can even be reversed after wearing special goggles that invert the image left for right.

Cerebellum mediates gain of VOR based on visual input through accessory optic pathway.

Anatomy of Vestibular System (review)

Semicircular canals

Lateral (Horizontal) canals

Tilt up 30 deg.

Excited by movement toward ampulla

Vertical Canals

Oriented 45 degrees off midline.

Left Anterior (aka Superior) is approximately coplanar with Right Posterior, and *vice versa*

Excited by movement away from ampulla, inhibited by movement toward ampulla.

Otoliths ("ear rocks")

Saccule is parasaggital, detects vertical acceleration.

Utricle is horizontal, detects head roll, head pitch and lateral acceleration.

Ocular counter-roll mediated by utricle.

Pairing of canals and extraocular muscles.

These pairings represent just the primary connections.

Anterior -> ipsi SR, contra IO

Posterior -> contra IR, ipsi SO

Lateral -> contra MR, ipsi LR

Three Neuron pathway for VOR

Bipolar cells in Canals via VIIIth Nerve to Vestibular Nuclei.

Premotor cells in Vestibular Nuclei to motoneurons in appropriate Cranial Nerve nuclei.

Motoneurons to muscles.

Push-pull nature of vestibular system.

For each canal there are both excitatory connections to agonist muscle and inhibitory connections to antagonist muscle.

Canal pairs have mutual inhibition through connections across midline between subnuclei of Vestibular Nucleus.

Lesions to Vestibular System

Most injuries reduce the signal from a canal, nerve or nucleus, but rarely one may have an irritative condition which increases it.

Reducing a signal from one canal is responded to as if the opposing canal were being stimulated (push-pull system).

Eyes drift toward side with lesion, jerk away.

Five functions of Vestibular Nucleus.

Binocular yoking of muscle pairs.

Reciprocal innervation of antagonist pairs.

Neural integration to convert velocity signals to position signals. This integrator also operates for OKN and pursuit as well as VOR.

Velocity Storage for VOR and OKN.

Gain adjustment for VOR adaptability.

Opto-Kinetic Nystagmus (OKN)

Purpose of OKN

Also called the Opto-Kinetic Reflex (OKR), or Parade Nystagmus, or Railway Nystagmus, it serves to stabilize images on the retina.

Primarily functions as a supplement to VOR during locomotion. The world is mostly stable, so most retinal image motion is generated by self motion.

Also stabilizes images on the retina when a large object moves across the visual field.

Measurement of OKN

Can be measured with virtually any kind of eye tracker.

In clinic, a small handheld drum is sometimes used. This is not ideal, because it tends to stimulate intentional following movements which mask the reflex.

Experimentally, a full field stimulus is usually used, such as a drum surrounding the subject or a large screen projection.

Because it is a reflex, OKN can be used to measure acuity in non-verbal subjects, such as infants.

Dynamics of OKN

On Sustained rotation of a stimulus, there is a **slow phase** following movement interrupted by **fast phase** returns to primary gaze. Velocity of OKN builds up over several seconds. After build up, if the lights are turned off it continues in the dark for several seconds, a phenomenon called **Opto-Kinetic After Nystagmus (OKAN).** OKAN is in the same direction as OKN and thus tends to cancel the Vestibular After-Nystagmus.

Latency is about 10 times longer than VOR, (e.g. as much as 150 msec). Perhaps 1/3 to 1/2 of this delay is in retinal processing alone.

Gain is the ratio of **Slow Phase Velocity** to Stimulus Velocity. **Amplitude** is the extent of each slow phase excursion. **Frequency** is the rate at which the gaze is reset by the fast phase. These depend on instruction, because voluntary Pursuit and Saccades can contribute to OKN.

Stare OKN (aka Passive or Involuntary OKN)

Subject is instructed to stare straight ahead and ignore the moving field.

Frequency is characteristically about 3 Hz. Amplitude tends to be small, around 1-2 deg because gaze resets often. Gain is about 0.5 even for lower velocities, meaning that the image is slipping on the retina.

Look OKN (aka Active or Voluntary OKN)

Subject is instructed to look at the moving targets.

Amplitude is 5-10 deg, as subject tracks over a significant range. Frequency is about 1 Hz. since gaze resets less often. Gain is near 1.0 for low velocities, meaning there's almost no slip of image on retina.

Suppression of OKN

Since OKN is a reflex, it has to be suppressed in order to follow a small moving target. This is accomplished by the Smooth Pursuit System. This also occurs if a fixation target is supplied in an OKN drum.

Pathway for OKN generation

Subcortical Pathway

Mediated by the Accessory Optic System, and the Nucleus of the Optic Tract (NOT).

Visual information from the contralateral eye only stimulates these pretectal nuclei, which in turn stimulate the Inferior Olive -> the Cerebellum -> the Vestibular Nucleus. The OKN pathway thus converges with the VOR pathway.

Horizontal OKN is mediated through the NOT and DTN (Dorsal Terminal Nucleus). Nuclei on the left side of the brainstem generate slow phases to the left in both eyes, based on visual information from the right eye only.

Vertical OKN is mediated through a different nucleus, the Lateral Terminal Nucleus.

Cortical Pathway

Mediated by the Geniculo-Striate pathway.

Visual information from both eyes is relayed back to the nuclei of the accessory optic system.

Asymmetry of OKN

When only one eye is viewing, the subcortical pathway shows an asymmetry in which OKN is much stronger for stimulus motion toward the nose.

In humans the Subcortical pathway develops first, so that infants show OKN asymmetry until about age 2 mo. In normal adults, the cortical

pathway completely dominates the subcortical and no asymmetry is evident.

Strabismic Amblyopes will often show an OKN asymmetry, however, possibly due to inadequately developed binocular cortical processing. These individuals may also show a latent nystagmus in which slow phase drifts toward the nose of a viewing eye occur only when the other eye is covered or deprived of clear vision. One prominent explanation for latent nystagmus is activity in the subcortical pathway for generating OKN.