

VS III: Ocular Motility and Binocular Vision
Spring 2007

Vestibulo-Ocular Reflexes
And
Optokinetic Nystagmus

Reading Chapter 5, Basic Eye Movements for the Clinician

I. Introduction

- Dynamic beings, constantly presenting challenges to our visual systems to maintain good vision
- Any movement, whether it be jogging, walking, spinning in a swivel chair, playing basketball, etc. requires one of two specialized systems to keep our gaze steady and clear
 - ...also to ensure that the world doesn't shake and move with our body movement.
- Optokinetic system and the Vestibular system prevent such visual disturbances caused by movement
 - allow us to do whatever movement we wish and still see clearly
 - if faulty, then with every head movement there would also be movement of the environment (a.k.a. oscillopsia).

II. Vestibular System (VOR)

- Main movement is the Vestibulo-ocular reflex (VOR)→ like Doll's Head movement
- Conjugate movement—both eyes move together in the same direction opposite the direction that the head moves in order to keep the eyes on the target→ if head moves left, then eyes move to the right. This is what was at work during step 3 of the Park's 3 step test.
- Function= stabilizes the retinal image during and quick/short head or body movement and movement of the visual world
 - What would happen to your vision if VOR didn't work well?
- Folks with cerebellar disease may have abnormal VOR→ so the world moves and shakes with every head movement
- VOR generates the "slow" phase of nystagmus
- Stimulated by things like your heel striking the pavement as you walk
- VOR is stimulated only by "acceleration," not by continued movement

A. Two Types of VOR: Dynamic and Static

Dynamic VOR= from the labyrinthine semicircular canals in the inner ears

- Generated eye movement that is equal and opposite to the amount of head movement
- used to sense transient angular accelerations of the head.
- As head rotates one direction, the endolymphic fluid is displaced in the opposite direction→ amount of endolymphic motion is proportional to the head velocity.
- This movement displaces the cuppula and the hair cells and stimulates a head-velocity signal.
- The vestibular neural network integrates that velocity signal to get a head displacement signal.

- as the fluid moves in the three canals a stabilizing eye movement (VOR) is generated so that you don't feel like you're tilting/seeing double in space when moving your head quickly

Static VOR= from maculae, saccule, utricle (otolith organs)

- The maculae of the saccule, and the utricle and otolith involved
- concerned with static tilts of the head and transient linear accelerations of the head; effected by gravity→ e.g. stimulate this if you tilt your head
- heavy otolith sits on top of the macula in the inner ear
- any shift in the otolith provides information regarding the tilt/angle of head
- resulting eye movement occurs with a 35-msec delay and is called TVOR, translational vestibuloocular response (also called "ocular head tilt reaction").

Besides maintaining stable retinal images during brief head movements, the vestibular system also

- Maintains overall body posture
- Equilibrium
- Muscle tone

Vestibular Pathway has a "3 neuron reflex arc:"

Hair cell sensory endings→ Primary vestibular nerve→→Vestibular Nuclei→

secondary vestibular neurons (via MLF)→ Oculomotor neurons→ EOM's

Connections between semicircular canals and EOMS:

<u>Canal</u>	<u>IPSI</u>	<u>CONTRA</u>
Horizontal	MR	LR
Superior	SR	IO
Posterior	SO	IR

B. More interesting facts about the vestibular system:

NBEO♦VOR latency is 16msec- this is the fastest time of all eye movements

♦VOR gain is defined as the ratio of the amplitude of eye rotation to the amplitude of head rotation

→Means that you get a certain amount of eye movement generated by a certain amount of head movement/rotation.

♦ Gain for horizontal head movement is slightly greater than that for vertical head movement. Therefore, compensatory eye movements are slightly better for horizontal than for vertical head movements.

♦ VOR gain best for nearpoint →more effective/efficient to rotate the eye than the head

****in what situation is this clinically significant?****

♦VOR gain decreases with age

♦With small levels of rotation of the head, fixation disparity (accuracy of vergence) remains good, due to Panum's Fusional area.

****What does this mean from a clinical standpoint?

♦VOR is not evident in congenitally blind folks. In late onset blindness, it is present, but reduced in ability. VOR is learned.

C. VOR adaptation

- System can adapt to changes resulting from growth, age, neurological disease, and weightlessness.
 - Astronauts use their VOR (and OKN) system to help them to maintain fixation on their instrument panels in space.
- VOR can be suppressed voluntarily → the cerebellar flocculus does this
Why is this important to be able to suppress VOR and in what kind of a situation would you want to do this?
- VOR also has variable adaptation to OPTICAL induced magnification changes:
 1. Left-right reversal prism: adaptation is slow and incomplete
 2. Telescopic spectacles in Low Vision: rapid adaptation
 3. Magnification changes in spectacle correction: myopes require LESS VOR-compensation for a given angular head rotation than do hyperopes → due to minification vs. magnification. With changes in magnification as these folks take off their glasses, VOR will readapt within 30 minutes to prevent the world from moving during every head movement.
 4. Anisometropia/aniseikonia: with moderate aniseikonia, VOR gain will adapt to an intermediate level between the two eyes (e.g., the overall “summed” retinal-image motion from the two eyes will be distributed equally between the two eyes).

Clinical note: Two systems probably work when patients “get used to” their new Rx with optically induced aniseikonia: VOR adaptation and recalibration of visual-perceptual cues, such as retinal disparity vs. other depth cues. Rotational mag changes don't occur with CL's. **Why?**

III. Optokinetic System

- Main movement is Optokinetic nystagmus (OKN)
- Optokinetic system produces eye movements (OKN) that compensate for prolonged, sustained self-rotational head movements (esp. of low freq., like 0.1 HZ)
- Gets help from the pursuit system
- OKN takes over from VOR during sustained movements, because vestibular cannot handle longer movements
 - as the vestibular system falters → the Optokinetic system takes over to stabilize images on retina
- Visual input can indirectly modify both VOR and OKN : you will see this in lab....

OKN (Optokinetic Nystagmus) is an involuntary jerk nystagmus induced by generalized movements of all or a large part of the visual field. It has a slow-phase tracking response that attempts to stabilize the retinal image and a fast saccade to reset the eye back to the primary position.

Clinically, we induce it with the OKN drum.

A. Some interesting OKN facts:

- Large field of motion produces the best OKN with a stationary target
- The initial 1 or 2 seconds of response are due to activation of the pursuit system; then the OKN dominates the rest of the response

- The peripheral retina dominates the response. Decreased central VA will reduce the OKN response by 20-30%
 - In what clinical situation will this fact about the peripheral retina cause an impact on OKN response? What symptoms will the patient experience in this case?
- **Latency is 140 msec.** Starts too late to be much help in preventing retinal slip from fast head movement. VOR stabilizes things in the quick responses, then OKN takes over as the motion continues.
- Horizontal and vertical gain is similar, except that vertical is more influenced by target velocity increases and declines its performance faster--> what does this mean for your OKN drum testing?
- OKN vertical gain upwards is greater than downwards
- Torsional OKN is slow and irregular
- Scotopic OKN is slightly lower than photopic OKN gain
- Full field OKN with a large drum surrounding the patient produces a sensation of self-rotation in the opposite direction-> think of driving through a rotating tunnel at Universal Studios (or in space?)

- Newborns have asymmetric OKN, N vs. T, at 2-3 months of age. This is assumed to be related to maturation of the neural pathways and development of BV paths. By 6-9 months, the two directions should be symmetrical.... Or the patient is at risk for strabismus or amblyopia.

A. Optokinetic Pathway

Retina → visual cortex → dorsal terminal nucleus of the optic tract → nucleus of the optic tract → inferior olive → cerebellum → vestibular nuclei → oculomotor nucleus

OK, so we have these two systems, but how do they interact with each other and why is it important?? Here's a scenario:

- You are in clinic waiting for your patient to arrive and decide to take a spin on a rotating exam stool.
- You start to spin slowly on the stool to the left.
- Your labyrinthine canals are stimulated with the "acceleration" movement made by your head as you begin to spin:
- Vestibular VOR system kicks in to stabilize your gaze so that you don't fall over and throw up from being dizzy and seeing diplopically → it also moves your eyes in the opposite direction of head rotation (the right in the example).
 - Important because if the images on the retina are not stable, then they start to slide around and that can cause blur or diplopia → at the very least this would make you feel dizzy and nauseated
- The vestibular VOR is a phasic/fast response, not designed to last very long, so...

- Fades out and eventually stops altogether, but you're having so much fun that you decide to continue your spin for a few more moments.
- → So, the Optokinetic (OKN) system kicks in and takes over the operation as primary gaze stabilizer until you stop moving.
- This cooperation between VOR and OKN allows stable, clear retinal images during head movements of any length of time.
- Without these two working for us, we would have oscillopsia and blurry vision during most any movement we make!!

What happens when your head stops moving?

- Your checkout doctor comes into the room and tells you that your patient is here (and also wonders why you're spinning on that stool!)
- You stop your motion.
- Vestibular system (VOR) is stimulated once again (because it is stimulated with any quick head movement)
- If VOR kept going, then you would again have a few seconds of movement in the opposite direction that you're moving → why is this bad if VOR kept working in this situation?
- BUT, the Optokinetic system continues to work for a few seconds after movement stops
 - Produces a postrotational nystagmus, called OKAN (Optokinetic After Nystagmus) that is in the same direction as the OKN response in order to cancel out the postrotary vestibular nystagmus (VOR).
 - Minimizes vertigo because the vestibular and OKN forces will be cancelled out.
 - OKAN won't completely suppress the postrotary nystagmus **IMMEDIATELY**
 - Takes a little while to dampen it down.
 - Different people have a different ability to dampen
 - Longer it takes to dampen VOR- induced nystagmus, the more dizzy people tend to be on circus rides, roller coasters, etc. (also remember that you can voluntarily dampen VOR).
- OKAN results from velocity storage,
 - phenomenon thought to be due to an indirect central neurologic integrating circuit that is activated by the head movement and gradually stores this velocity info. When the movement stops, that circuit will discharge it. It's kinda like a capacitor in electronics (this is also how turn signals and blinking lights occur).

So what's the big deal with these systems??

It is a big deal, because there can be problems with the systems that can cause tremendous suffering for your patients. If the VOR fails due to disease, then vision while moving is impaired. VOR acts like a balancing mechanism while you walk: if it doesn't work, you won't see people clearly unless you STOP to look at them, otherwise they are blurry or double. Imagine trying to drive, jog, walk, go up and down stairs, and so on. It would make normal living pretty tough, for you couldn't do a lot of the things that most of us take for granted.

So.... to recap:

During head movement:

- VOR (vestibular system) initiates to stabilize and compensate for brief head movements
- As the movement is continued, the vestibular system fades out and the Optokinetic system takes over to stabilize gaze (with help from the pursuit system). This results in OKN (Optokinetic nystagmus).
- The cooperation between the two systems allows the vision to stay clear and stable during head movements.

As the head movement stops (and the fluid in the ears is still sloshing around):

- The vestibular system kicks in again (because there is a fast change in movement) and stimulates postrotary nystagmus (fast phase opposite direction of head mvt).
- The Optokinetic system keeps on working for a few seconds after head movement has stopped, and generates OKAN to counteract the effects of the vestibular system's nystagmus.
- This stops the person from feeling vertigo.

In reality, the two systems work together in one cooperative system. However, for our purposes, we can consider them as separate entities.

IV. Abnormal vestibular and Optokinetic function

A. Abnormal vestibular function

- Amblyopia:

There are asymmetric and reduced vestibular responses in amblyopic eyes. You can test this with calorics (remember COWS?). It has been found that in folks with strabismus and amblyopia, the caloric nystagmus has a much more variable amplitude and frequency when compared to that of folks with normal BV.

Some patients with congenital ET have reduced VOR during clinical testing → means that many patients with amblyopia have vestibularly-related balance problems in the dark.

Finally, in patients with strabismus, there is an asymmetric VOR adaptation between the two sides. The VOR gain increases more after adaptation to nasal field motion than after temporal field motion. This ties in with what we talked about before about the asymmetry in OKN in kids with strabismus.

OK, so why is this important clinically??

Its important because it is another tool to help you to determine if that infant in your exam chair is going to get strabismus or amblyopia; it will help you to give a more informed answer to those parents who are concerned about possible strabismus. It will also help you to give appropriate diagnosis and treatment to stroke patients who are dizzy from a vestibular imbalance.

- Vestibular disease

-Acute unilateral peripheral disease can cause a transient imbalance in vestibular tonus (e.g., baseline tonic innervation) between the right/left vestibular nuclei.

This will give rise to spontaneous nystagmus, with a slow phase directed towards the side of the lesion. It is also greater in darkness than in light.

→ why would this be important clinically?

-Bilateral acute peripheral disease of the labyrinth can cause serious problems for the patient, esp. oscillopsia and degraded vision, because of the poor ability of the VOR to compensate.

→ why will this cause oscillopsia and blurry vision?

-Central vestibular disorders can cause lots of problems as well, such as spontaneous nystagmus. Patients may also complain of a “tilting” of their world.

B. Abnormal optokinetic function

- Amblyopia:

 - Usually has reduced OKN

 - Asymmetric OKN

- Nystagmus:

 - Reduced OKN in congenital nystagmus

- Neurological disease:

 - Newborns- asymmetric response (normal)

 - Lesions in the anterior and cortical visual pathways-
Will show slow buildup of OKN responses and asymmetries

 - Unilateral and bilateral labyrinthine disorders-

 - Will show increased slow phase velocity towards the side of the lesion and bi-directional reduction of OKAN in a unilateral case, and will show normal OKN but absence of OKAN in a bilateral case.