VESTIBULO-OCULAR REFLEXES AND OPTOKINETIC NYSTAGMUS

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THIS CHAPTER INCLUDES A REVIEW OF:

- Vestibular System (VOR)
- Optokinetic System
- Abnormal Vestibular and Optokinetic Function

INTRODUCTION

- Humans are dynamic beings, constantly presenting challenges to our visual systems to maintain good vision
- Any movement, whether it is jogging, walking, spinning, playing football, etc. requires one of two specialized systems to keep our gaze steady and clear, and also to ensure that the world does not shake and move with our body movement.
- **Optokinetic system** and the **Vestibular system** prevent such visual disturbances caused by movement
- These systems 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).

VESTIBULAR SYSTEM

	• The vestibulo-ocular reflex (VOR) is controlled by the vestibular system. It is a normal reflex that is responsible for the compensatory positioning assumed by the eyes when the head moves. • Conjugate movement— both eyes move together in the same direction as one another but in the opposite direction to the head movement. This allows the eyes to hold fixation on the target; i.e. if the head moves left, then the eyes move to the right. This is what happens during step 3 of the Park's 3-step test. • The function of the VOR is to stabilize the retinal image during a quick/short movement made by the head or body or indeed any movement of the visual world. • Consider what would happen to your vision if the VOR did not work well! • People with cerebellar disease may have abnormal VOR. For such people, the world is perceived to move and shake with every head movement • VOR generates the 'slow' phase of nystagmus • It is stimulated by things like your heel striking the pavement as you walk • VOR is stimulated only by 'acceleration', not by continued movement

TYPES OF VOR:	There are two types of Vestibulo-ocular reflexes: Dynamic and Static.
DYNAMIC VOR	The Dynamic VOR arises from the labyrinthine semi-circular canals in the inner ears • Generates an eye movement that is equal and opposite to the amount of head movement • Detects transient angular accelerations of the head. • As the head rotates in one direction, the endolymphic fluid is displaced in the opposite direction. The degree 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 with the velocity signal to obtain a head displacement signal. • As the fluid moves in the three canals, a stabilizing eye movement (VOR) is generated. This prevents the sensation of head tilt or diplopia when the head moves quickly
STATIC VOR:	The Static VOR arises from the maculae, saccule and utricle (otolith organs) • This reflex involves the maculae of the saccule as well as the utricle and otolith • Concerned with static tilts and transient linear accelerations of the head; i.e. it is effected by gravity. Tilting of the head will stimulate the static VOR. • A 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 vestibulo-ocular response) or the '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 are mentioned in Table 10.1. It illustrates that the orientation of the horizontal semi-circular canals approximates the pulling action of the medial rectus on the same side of the movement and the lateral rectus on the opposite side. Similar relationships can be observed for the superior and posterior semi-circular canals.

Table 10.1 Connections between semicircular canals and EOMS

Canal	IPSI	Contra
Horizontal	MR	LR
Superior	SR	IO
Posterior	SO	IR

VOR ADAPTATION	• The system can adapt to changes resulting from growth, age, neurological disease, and weightlessness. Astronauts use their VOR (and OKN) system to help them maintain fixation on their instrument panels in space. • VOR can be suppressed voluntarily → the cerebellar flocculus does this. Why is it important to be able to suppress VOR and in what kind of situations would you want to do this? • VOR can make 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. – Magnification changes when such people remove their spectacles. 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 i.e. the overall "summed" retinal-image motion from the two eyes will be distributed equally between the two eyes.

CLINICAL NOTE: It is likely that the two systems are at work while a patient adapts to the new Rx that induces aniseikonia: These two systems are VOR adaptation and VOR recalibration of visual-perceptual cues. Rotational magnification changes do not occur with contact lenses because contact lenses follow the eye rotations.

OPTOKINETIC SYSTEM

OPTOKINETIC SYSTEM	▪ Optokinetic nystagmus (OKN) is the eye movement controlled by the Optokinetic system ▪ Optokinetic system produces eye movements (OKN) that compensate for prolonged, sustained self-rotational head movements (esp. of low frequency like 0.1 HZ) ▪ Supported by the pursuit system ▪ OKN takes over from VOR during sustained movements, because the vestibular system 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 OKN is an involuntary jerk nystagmus induced by generalized movements of all of or a large part of the visual field. It has: – A slow-phase tracking response that attempts to stabilize the retinal image – A fast saccade to reset the eye back to the primary position. Clinically, we can induce OKN with an OKN drum.

SOME INTERESTING OKN FACTS	• A large field of motion, using a stationary target produces the best OKN • The initial 1 or 2 seconds of response are due to the activation of the pursuit system; then the OKN system 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.** This is too long a delay to be much help in preventing retinal slip from fast head movement. Therefore VOR stabilizes fixation with a quick response, and then OKN takes over as the motion continues. • Horizontal and vertical gain is similar, except that vertical gain is more quickly and moreinfluenced by target velocity increases and declines in terms of performance → 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

Newborns have asymmetric OKN, Nasal vs. Temporal, at 2-3 months of age. It is assumed that this is 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.

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

SOME INTERESTING OKN FACTS	OK, so we have these two systems, but how do they interact with each other and why is it important? Here is 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 perceiving diplopia → it also moves your eyes in the opposite direction of the head rotation (the right in this example). This is 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 it 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. • Now, the Optokinetic (OKN) system kicks in and takes over the operation as the primary gaze stabilizer until you stop moving. • This cooperation between VOR and OKN allows for the maintenance of stable, clear retinal images during head movements of any length of time. • Without these two systems working for us, we would have oscillopsia and blurry vision during most of the movements we make!!
WHAT HAPPENS WHEN YOUR HEAD STOPS MOVING?	• Your receptionist 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 to that in which you had been 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, it produces a postrotational nystagmus, called OKAN (Optokinetic after Nystagmus). OKAN occurs in the same direction as the OKN response in order to cancel out the postrotary vestibular nystagmus (VOR); it 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 levels of ability to dampen – The longer it takes to dampen VOR the more likely you will find induced nystagmus. This will cause more dizziness. Note: It is possible to voluntarily dampen VOR. • OKAN results from velocity storage, the phenomenon is thought to be due to an indirect central neurologic integrating circuit that is activated by the head movement and gradually stores this velocity information. When the movement stops, that circuit will discharge it.

Why is it important that you understand these systems?

It is important because as future clinicians you may come across patients who have problems with these systems, which can cause tremendous suffering for your patients. It the VOR fails due to disease, then vision while moving will be 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 if these systems did not function properly. It would make normal living very challenging. You could not do a lot of the things that most of us take for granted.

RECAP

During head movement:

- VOR (vestibular system) initiates to stabilize an image on the retina and compensate for brief head movements
- As the movement is continued, the vestibular system fades out and the Optokinetic system takes over to stabilize the 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 movement).
- 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 developing vertigo.

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

ABNORMAL VESTIBULAR AND OPTOKINETIC FUNCTION

A. ABNORMAL VESTIBULAR FUNCTION	**AMBLYOPIA** There are asymmetric and reduced vestibular responses in amblyopic eyes. You can test this using the caloric reflex test, which tests VOR and involves irrigating warm or cold water into the external auditory canal. It has been found that in people with strabismus and amblyopia, the caloric nystagmus has a much more variable amplitude and frequency when compared to that of people with normal BV. Some patients with congenital esotropia have reduced VOR during clinical testing. This means that many patients with amblyopia have vestibular-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.

Why is this important clinically?

It is important because it is another tool to help you to determine if that infant in your exam chair is going to develop 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.

A. ABNORMAL VESTIBULAR FUNCTION	**VESTIBULAR DISEASE** Acute, unilateral, peripheral disease can cause a transient imbalance in vestibular tonus (i.e. 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 amplified in darkness compared with in light. → Why would this be important clinically? Bilateral, acute, peripheral disease of the labyrinth can cause serious problems for the patient, especially oscillopsia and degraded vision, which develop due to the poor ability of the VOR to compensate. → Why will this cause oscillopsia and blurry vision? Central vestibular disorders can also cause a number of problems, 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:** An asymmetric response is normal ▪ **Lesions in the anterior and cortical visual pathways** will show a slow build-up 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. They will also show normal OKN but absence of OKAN in a bilateral case.

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