

Raghunath Tiruvaipati

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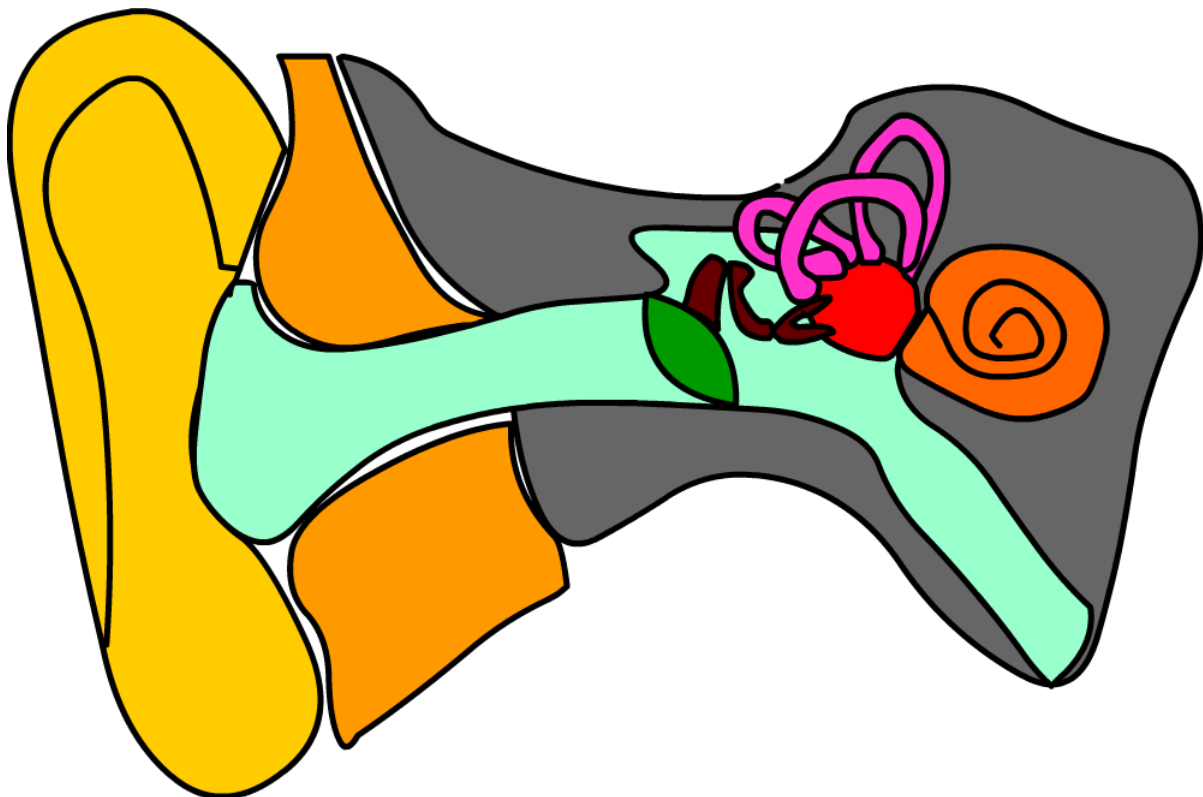
Vestibular System & Eye Moments

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RAGHUNATH
TIRUVAIPATI
@MXPLANK.COM

Vestibular System and Eye Movements

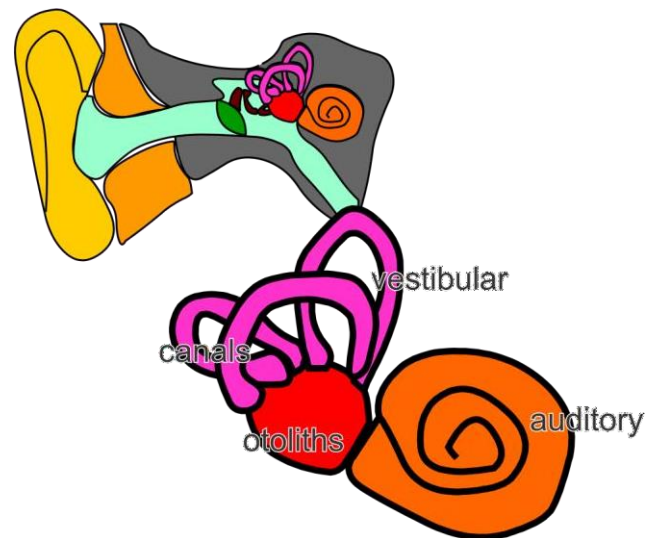


Contents

Vestibular System and Eye Movements	1
The sense of balance originates in the labyrinth.....	3
The anatomy of the otoliths	4
The otoliths sense	5
The functional anatomy of the semicircular canals	6
What is the vestibulo-ocular reflex (VOR)?	7
The generation of saccadic eye movements	8
The eyes are rotated by 6 extra ocular muscles.....	9
Vergence eye movements.....	10
Pursuit eye movements.....	10
What is nystagmus?	11
Why we get dizzy.....	12
For practice problems.....	13

The sense of balance originates in the labyrinth.

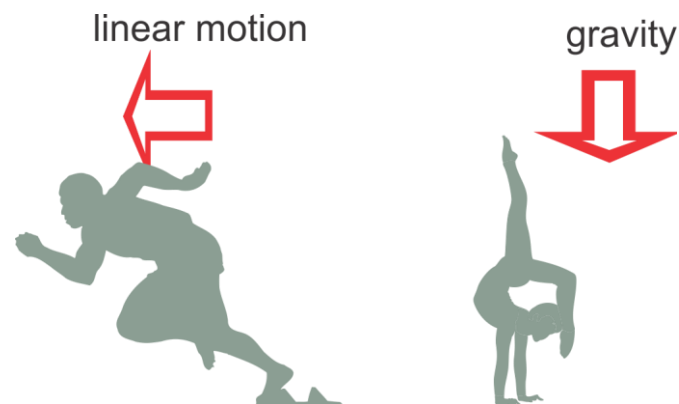
The labyrinth is a system of tunnels in the skull that contains the sensors for the auditory and vestibular systems. The **vestibular** system is responsible for one's sense of balance. The vestibular system has two parts: the otoliths and the semicircular canals. Each has different functions.



The **otoliths** have two functions:

1. They sense the head's **linear** motion (e.g. moving forward or to the side).

2. They are also able to sense the head's position relative to **gravity**. These are the organs that tell us which way is down.



angular motion



The canals detect the head's rotation (turning motion).

The anatomy of the otoliths

The otoliths are two spheres called the **utricle** and the **sacculle**.

In each, a portion of the inside surface of these spheres is covered with **hair cells** similar to those in the auditory system.

The hairs of these cells have a slanted "crew cut". The thickest and longest of hairs is called the **kinocilium**.

The hairs project into a gel-like substance. Calcium carbonate crystals, **ear stones**, are embedded in this gel. These stones are important because it is their weight that bends the hair cells.

Steps for converting motion into neural activity

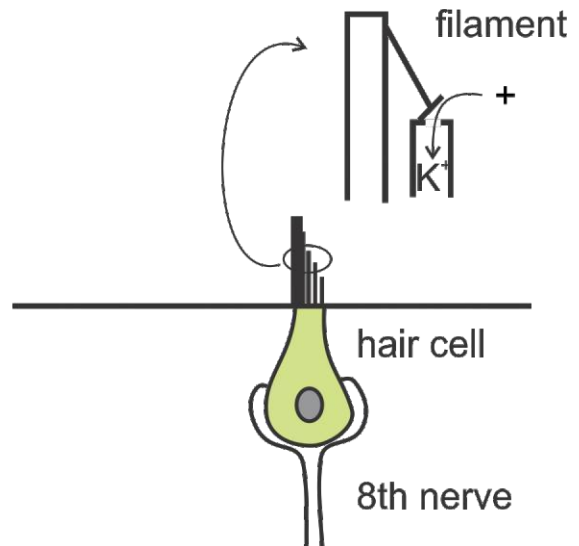
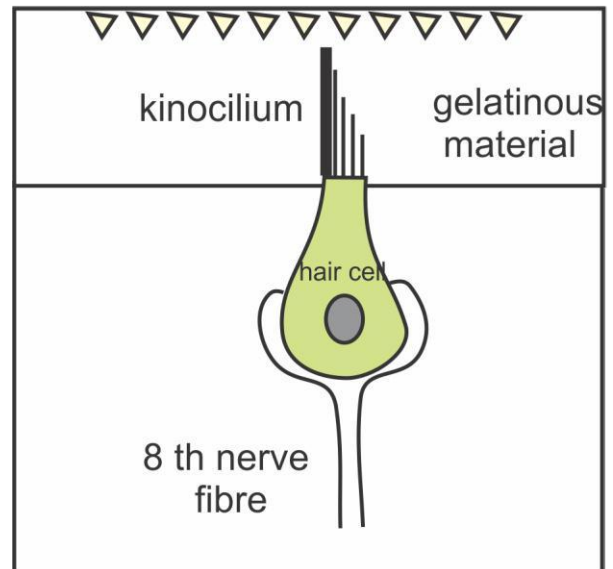
1) Motion or gravity act on the mass of the crystals. which, through the gel, bend the hairs.

2) The filament between adjacent hairs opens ion channels. A positive charge forces K^+ into the hair cell.

3) The hair cell depolarizes, releasing neurotransmitters.

4) There is an increase in the frequency of action potentials in the 8th nerve afferent.

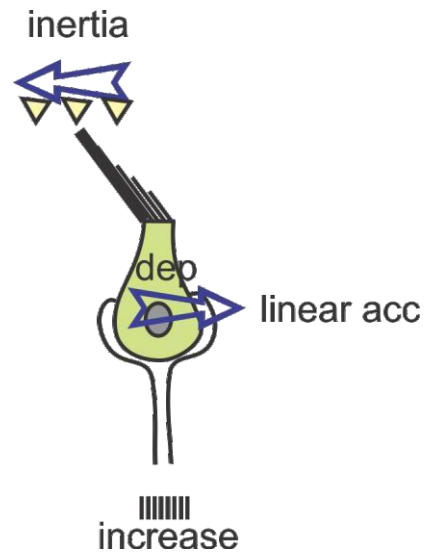
calcium carbonate crystals (otoliths)



The otoliths sense:

1. linear motion (e.g. moving forward or to the side).

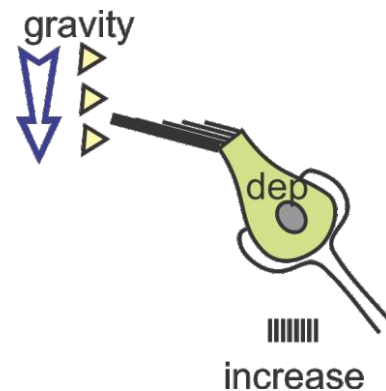
When the head moves, inertia tends to keep the crystals stationary and this bends the hair cells in the opposite direction.



2. They sense the head's position relative to gravity.

Like linear motion, gravity "pulls" on the crystals.

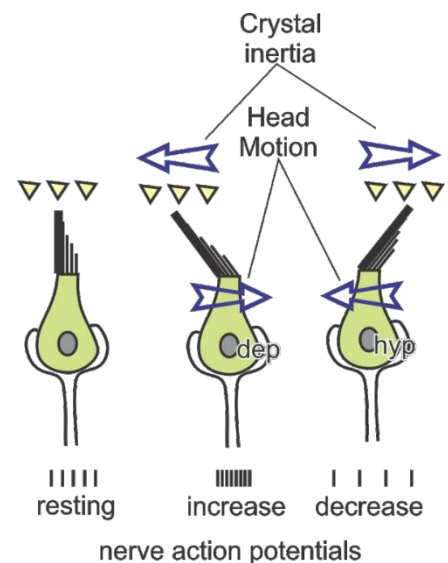
When head position changes, the direction of this gravitational "pull" changes, telling you that your head has tilted.



When the hairs are undisturbed, the vestibular afferents have a baseline firing rate of about 100 action potentials (AP) per second.

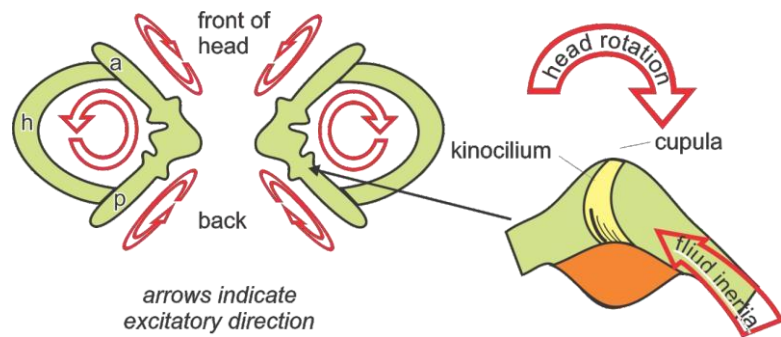
Bending the hairs towards the kinocilium depolarizes the cell, inducing an increase in AP frequency in the 8th nerve.

Bending away from the kinocilium causes hyperpolarization and reduced AP frequency.



The functional anatomy of the semicircular canals

There are three canals in each side. One is approximately **horizontal** (h), and the other two, the **anterior** (a) and **posterior** (p), are aligned vertically and are about perpendicular to each other. Within the canals are fluid-filled semicircular ducts which open at both ends onto the otoliths. Each canal has a swelling. Within this swelling a pliable membrane called the **cupula** seals the canal. Embedded in the cupula are hair cells.



How do the canals detect angular rotation of the head?

When the head starts to turn, the fluid lags behind because of inertia, pushing on and distorting the cupula. Bending the hairs towards the kinocilium causes an increased excitation of the hair cell. Bending the hairs away from the kinocilium causes less excitation of the hair cell.

How do the canals compute the direction of head rotation?

Each of the 6 canals is best activated by rotating the head in the plane of the canal. See arrows in the image. As you might expect, the horizontal canals are best activated when the head rotates in the horizontal plane. But notice that the right anterior canal is best activated when the head tips in the direction of the yellow arrow (not the nose down or the right ear down but a combination of both). The canals are arranged in pairs. Each canal has a partner on the other side of the head. When one partner is maximally excited, the other is maximally inhibited. When the head rotates rightward, excitation occurs in the right horizontal canal and inhibition in the left. Surprisingly, the partner of an anterior canal is the posterior canal on the other side. Both lie in the same plane.



What is the vestibulo-ocular reflex (VOR)?

The otoliths and canals activate many reflexes. These connect to your legs, trunk and arms for postural support. A key reflex is the vestibular-ocular reflex (VOR).

The function of the vestibulo-ocular reflex is to stabilize the image on the retina during rotations of the head. When the head rotates with a certain speed and direction, the VOR rotates the eyes with the same speed but in the opposite direction. The combination of head and eye rotation, the eye's rotation in space, is ideally near zero. Without the VOR, your eye would see a smeared image every time you moved your head. This is because the eye is like a camera with a slow shutter speed.

The neural mechanism for a horizontal VOR

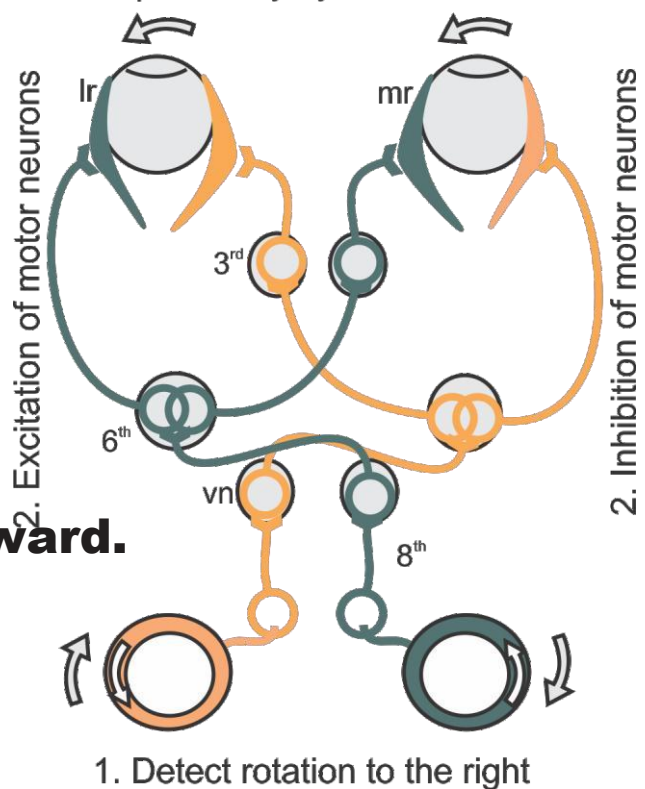
This sequence of events occurs when the **head rotates rightward**.

- The **right horizontal canal** hair cells depolarize.
- The right vestibular nucleus (VN) activity increases,
- as does that of motoneurons in the **left abducens** (6th) nucleus.
- The **left lateral rectus** (lr) muscle contracts.
- Left abducens nucleus also activates motoneurons in the **right oculomotor** (3rd) nucleus, which contracts the **right medial rectus** muscle.
- **Both eyes rotate leftward.**

Neurons on other side do the opposite.

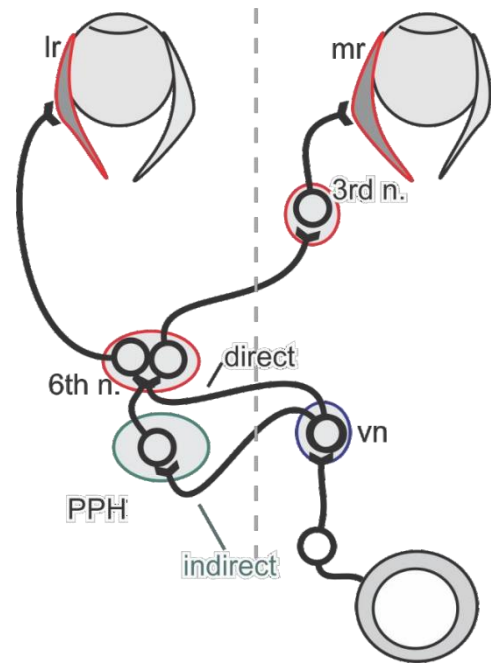
- Neurons in the left vestibular nucleus become less active.
- This in turn causes the right lateral and left medial rectus muscles to relax.
- This helps the eyes rotate leftward.

3. Compensatory eye rotation to the left



The direct path, by itself, is not enough. Why?

During a head turn to the right, the eyes turn to the left (via the **direct** path of the VOR). When the head stops turning, the eyes should stop turning and remain pointing to the left. However, the eyes will drift back to the center because muscles need a large maintained activation to keep the eye turned left. This additional tonic input comes from the **n. prepositus hypoglossi** (PPH) in the **indirect** path. This nucleus converts the short lasting (**phasic**) vestibular input into a long lasting (**tonic**) signal. This nucleus acts as a form of short-term memory, which remembers **how far** the head has turned.



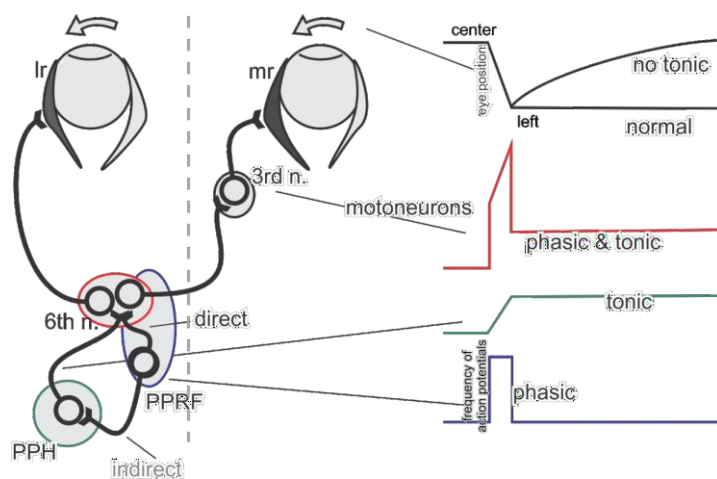
The generation of saccadic eye movements

As we saw in chapter 1, only the fovea of the retina sees in detail. Saccades redirect foveas to objects of interest, e.g. the words in this sentence.

Vision is impaired during these movements. To minimize this time, saccades are very fast (faster than any other movement). These high velocities are generated by a phasic burst of action potentials to the muscles (up to 1000 action potentials per second).

This burst of activity originates in the **paramedian pontine reticular formation** (PPRF), near the nucleus of the sixth cranial nerve (6th nucleus.). The PPRF on the left generates saccades to the left in both eyes. As in the VOR, there are two paths:

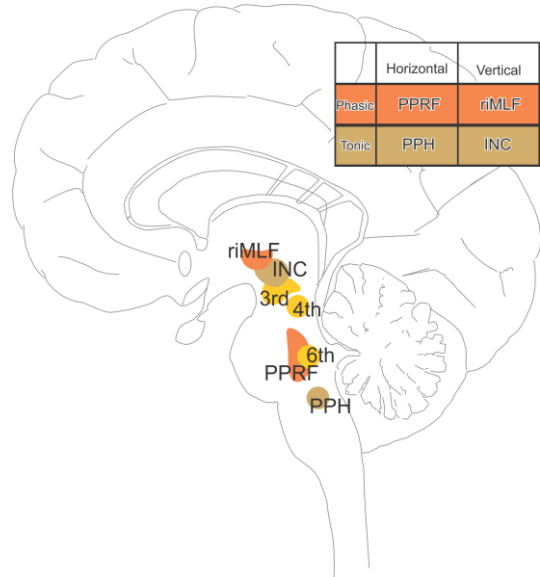
- 1) a direct path which mediates the phasic command to move the eyes.
- 2) an indirect path via PPH which generates the tonic command to hold the eyes in an eccentric



position.

How are vertical saccades generated?

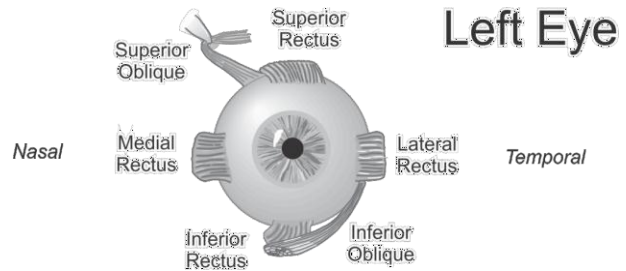
As in horizontal saccades, you need phasic activity to turn the eye and tone to hold it in its new position. The phasic activity is generated by **the rostral interstitial n. of the MLF (riMLF)** near 3rd and 4th nucleus. The tone is generated by the adjacent **interstitial n. of Cajal (INC)**. The pulse and step combine on the motoneurons of the 4 vertical muscles in the 3rd and 4th nucleus.

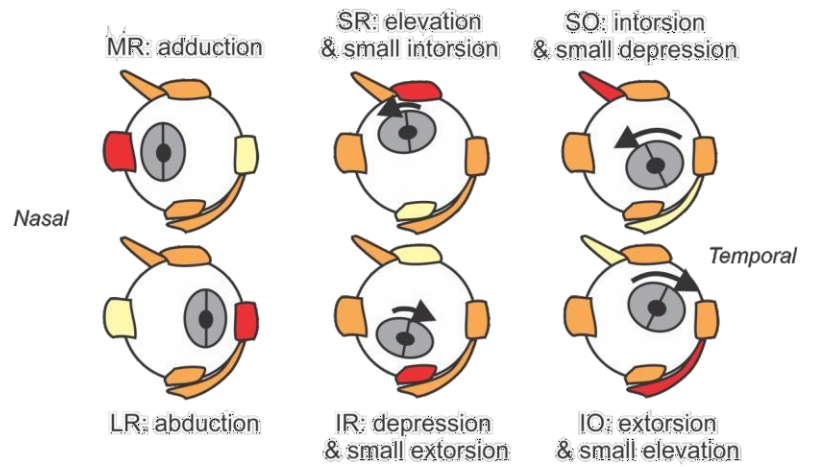


The eyes are rotated by 6 extra ocular muscles.

They act as 3 agonist/antagonist pairs. When your eye points forward, 60% of its motoneurons are active. To look elsewhere, one of each muscle pair contracts and the other relaxes.

You need 3 pairs to allow rotations in all three directions: horizontal, vertical and torsional.



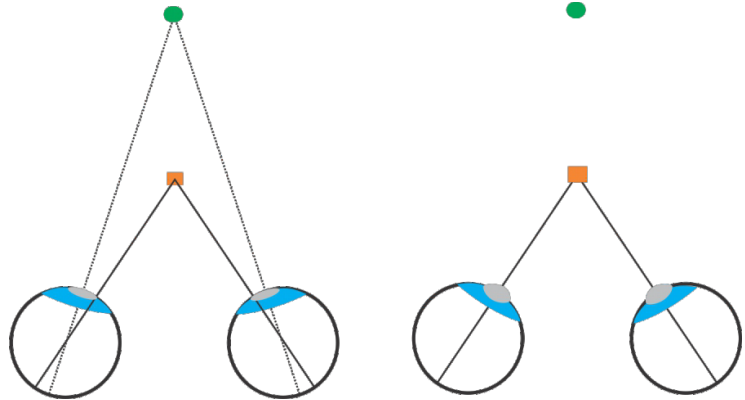


Vergence eye movements

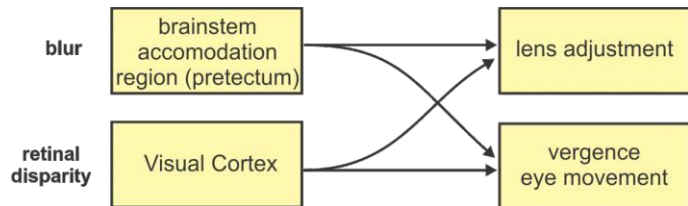
These are the slowest to develop during childhood. They prevent double vision (diplopia) and mask strabismus. They rotate the eyes in opposite directions (disconjugate). These movements are linked to accommodation reflex (the reflex that changes the eye's lens properties).

On viewing a near object the eyes (*the near response*)

- 1) converge to eliminate retinal disparity (seeing double),
- 2) accommodate (lens becomes more round) to eliminate the retinal blur and
- 3) their pupils constricts to reduce the blur.

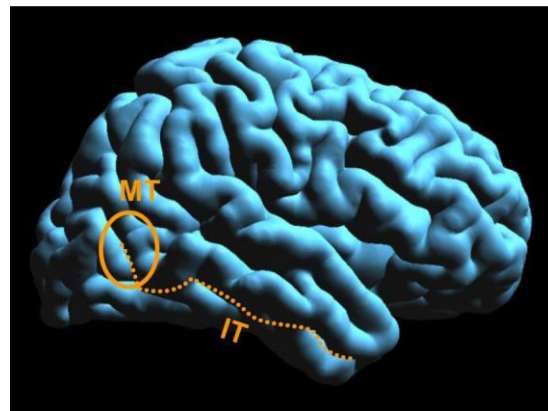
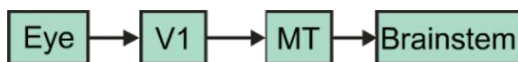


Either blur or retinal disparity will generate vergence. The strongest response is elicited when the image is blurred and there is retinal disparity.



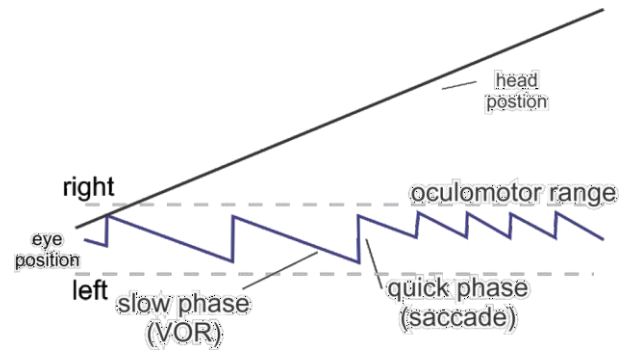
Pursuit eye movements

When an object moves, the image is kept still on the fovea by means of a pursuit eye movement (e.g. tracking a moving ball or your finger). The sequence of structures that are used to generate pursuit eye movements is:



What is nystagmus?

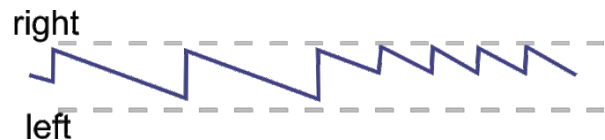
Nystagmus is a rhythmic back and forth movement of the eyes. Usually the movement in one direction is fast and slow in the opposite direction. Nystagmus is seen normally when you turn your head round and round. The **VOR** generates the **slow phase**, which keeps your eye on a target. When the eye approaches the maximum that the eye can turn (the oculomotor range), a **saccade (quick phase)** is generated in the opposite direction to a new target. The saccade can also occur earlier, before the eye approaches the oculomotor range, and this would increase the frequency of the nystagmus.



Three main types of nystagmus are seen with lesions.

1) Imbalance in the VOR

This looks like normal nystagmus. But here the head is still. The drive is not from head motion but from an imbalance in the VOR.

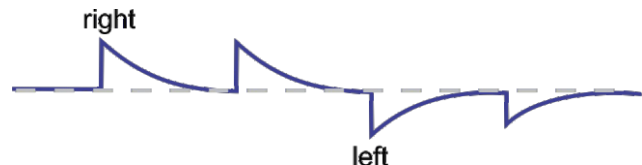


Recall that normally vestibular afferents have a tonic drive. The drive from the right side is cancelled by that from the left. Unilateral lesions (vestibular canal or afferent nerve) disrupt this balance. Patients sense that they are turning in the direction opposite to the lesion.

Which horizontal canal may be lesioned here? The slow phase is to the left. This would normally occur when you were turning to the right. But now the patient is stationary. Thus the imbalance is generating too much activity from the right side. This would occur if the patient had a lesion of the left horizontal canal.

2) Tonic activity in nPPH is too small

This is caused by a lesion of the nPPH (tone generator). After a saccade to the left or right, tonic activity in PPH is insufficient to keep the eye turned and it drifts back to the center. The patient then saccades again and the process is repeated. In contrast to the previous case here slow phase i) shows an exponential (not linear) drift to a position of rest (often at the centre). ii) its direction switches when the patient looks in the opposite direction. The direction, speed and waveform of nystagmus are important in determining the cause of nystagmus.



3) Benign Paroxysmal Positional Vertigo (BPPV)

BPPV lasts for a few minutes and is induced by a change of head position. Calcium crystals dislodge from the utricle and migrate to a semicircular canal. Crystals push on the cupula when the head position changes. Vertigo is a type of dizziness, a feeling of motion when one is stationary. In BPPV dizziness has a rotational component (a feeling of spinning). It is induced by

a change in position with respect to gravity. This nystagmus is paroxysmal (sudden reoccurring attacks).

Why we get dizzy

During normal head rotations, the eye rotates opposite to the head, thus canceling the motion of the head. This tends to stabilize the image of the world on the retina.

Vestibular input without vision

During very prolonged head rotations with the eyes closed, the elasticity of the cupula gradually restores it to its upright position. The drive to the VOR stops (falsely telling the brain that one is stationary). If at this point you open your eyes, you see the world moving and you feel dizzy.

Visual sense of motion without vestibular drive

Visual input on its own can drive the VOR (the **optokinetic response**). This visual input can elicit a false perception of motion. For example, a false sense of motion often occurs when looking out a car window and an adjacent car starts to move.

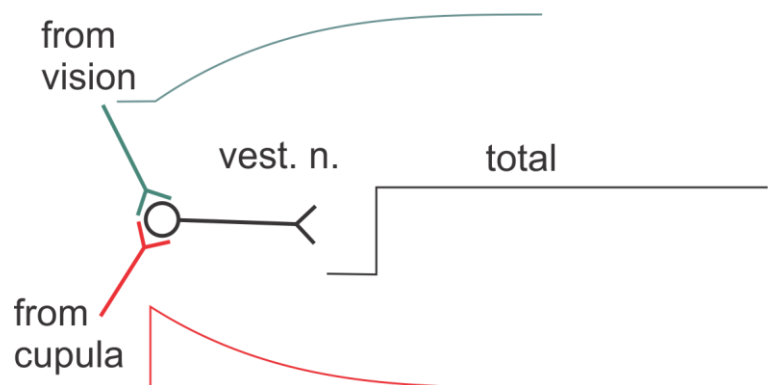
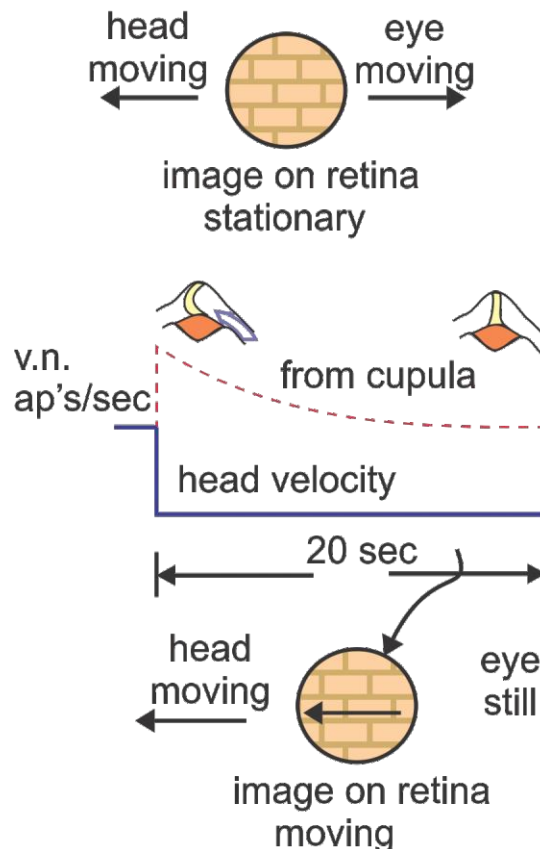
Combined visual and vestibular input

During a prolonged head rotation in the light, both the signal from the cupula and visual input reaches the vestibular nuclei. Visual input builds up as the signal from the cupula dies away, thus compensating for the loss of cupula drive.

Motion sickness occurs when the two signals are in conflict. Suppose you are inside the cabin of a boat during a storm. Your vestibular afferents are telling you that you are moving. Because you and the cabin are moving together, the visual system senses that you are not moving. To avoid motion sickness the best bet is to go out on the deck and look at the horizon.

One theory for the feeling of nausea is that the brain interprets this conflict as poisoning and responds by eliciting

Normal VOR response



Raghunath Tiruvaipati

vomiting to clear the poison.