This slide captures one way to think about the motility disorders. If it is unfamiliar, I strongly suggest you review the slide-set entitled ‘Motility disorders: Overview’ (N18) before proceeding.
In this slide-set, we’ll take a look at supranuclear syndromes.
Before discussing **supranuclear lesions**, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do *that*, we have to define the role of the **afferent** system. (Get comfy, this is gonna take a minute.)
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**Supranuclear**

Orbital

Neuromuscular junction

Extraocular muscle
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**Supranuclear**

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Motility Disorders: Supranuclear Syndromes
In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.

Motility Disorders: Supranuclear Syndromes

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Next, we will **drill down on the six supranuclear pathways/control systems. They will be presented in an order that makes sense (to me), but that should **not** be (mis)interpreted as reflecting some sort of intrinsic order of importance.**
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Motility Disorders: Supranuclear Syndromes

Supranuclear

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The motility disorders: supranuclear syndromes consist of six systems in the primate CNS that deal with these fixation-related issues.
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But let’s consider what it takes to accomplish these tasks. Scrutinizing an object requires steady bifixation—but not too steady, or the photoreceptors (PRs) will fatigue and the image will disappear. Further, the object might be moving, necessitating object-tracking. Further still, the primate’s head might be moving, necessitating object-tracking.

At long last, the payoff for all this backstory: The supranuclear pathways consist of systems in the primate CNS that deal with these fixation-related issues. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.

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Motility Disorders: Supranuclear Syndromes

Supranuclear

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Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, not microsaccadic) or visually disruptive slower refixation movements (nystagmus, not saccades).

The vergence system is responsible for maintaining fixation on an object that is moving toward or away from the eyes. There are a number of components to the vergence system; clinically, the two most important are fusional vergence (disparity between the retinas with respect to image location) and accommodative vergence (triggered by retinal image blur). Many forms of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasms of the near.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then it must ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In primates, vision has two purposes: 1) to detect objects of interest (eg, things you may want to eat, or may want to eat you), and 2) to scrutinize objects of interest (ie, to determine definitely whether it’s an eat-er vs an eat-ee). To accomplish these ends, the afferent visual system can be divided into two channels:

-- A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.
-- A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on outs.

Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), while also monitoring for other objects that may require scrutinization (peripheral channel). It follows from this that the efferent visual system has two jobs: 1) Keep both foveas pointing at the current object of regard; and 2) rapidly redirect both foveas to a new object when one is detected in the periphery.

But let’s consider what it takes to accomplish these tasks. Scrutinizing an object requires steady bifixation—but not too steady, or the photoreceptors (PRs) will fatigue and the image will disappear. Further, the object might be moving, meaning the efferent system has to precisely track it. Further still, the primate’s head might be moving, also necessitating object-tracking.

The VOR is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head in a given plane leads to an excitatory burst from the canal of the same plane on the side of the head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the opposite side. So, a rapid horizontal head turn to the right leads to an excitatory burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid right horizontal head turn will produce a rapid leftward rotation of the eyes.

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, not microsaccadic) or visually disruptive slower refixation movements (nystagmus, not saccades).

The vergence system is responsible for maintaining fixation on an object that is moving toward or away from the eyes, thus necessitating converging or diverging. There are a number of components to the vergence system; clinically, the two most important are fusional vergence (disparity between the retinas with respect to image location) and accommodative vergence (triggered by retinal image blur). Many forms of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
Before discussing \textit{supranuclear lesions}, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do \textit{that}, we have to define the role of the \textit{afferent} system. (Get comfy, this is gonna take a minute.)

The \textit{ocular fixation system} is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant steady, or the photoreceptors (PRs) will fatigue (and subsequent image loss) from occurring.

Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, fatigue), or fusional vergence insufficiency, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

But let's consider what it takes to accomplish these tasks. Scrutinizing an object requires steady bifixation—but not necessarily. In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image.

Infranuclear \textit{supranuclear pathways} are responsible for maintaining fixation on an object that is moving toward or away from the eyes, thus necessitating they converge or diverge. There are a number of components to the system has to precisely track it. Further still, the primate's \textit{vestibulo-ocular system} has two jobs: 1) Keep both foveas pointing at the current object of regard; and 2) rapidly redirect both foveas to a new object when one is detected in the periphery.

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the \textit{two words} or \textit{eponym} syndrome.

The \textit{ocular tilt reaction} is caused by a \textit{vertical deviation} accompanied by \textit{intorsion} of the hypertropic eye, with an \textit{intorsion} of the same plane on the opposite side of the body, and a \textit{Horner syndrome}. (Wallenberg syndrome is discussed in a separate section.)

Less common are disorders stemming from otolith issues, which have two classic manifestations: skew deviation and skew deviation. To accomplish these ends, the afferent visual system can be divided into two channels:

- \textit{Peripheral channel for detecting objects.} The peripheral channel is very sensitive to motion.
- \textit{Central channel for scrutinizing objects.} Anatomically, this channel consists of the foveas, which provide high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

The rapid right horizontal head turn will produce a rapid leftward rotation of the eyes. So a burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts of the same plane on the opposite side. So, a rapid horizontal head turn to the right leads to an excitatory burst from the canal toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head in a given plane.

Thus, lesions of a supranuclear pathway manifest as difficulties with either the vestibulo-ocular system (aka the \textit{vestibulo-ocular reflex, VOR}) or the \textit{optokinetic system} (aka \textit{optokinetic nystagmus, OKN}) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The head is still. It does this via continuous microsaccadic refixation movements, which produce a constant steady, or the photoreceptors (PRs) will fatigue too much from occurring.

Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, or accommodative esotropia, and accommodative vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.

In primates, vision has two purposes: 1) to rapidly redirect both foveas to a new object when one is detected in the periphery. 2) Refixation. Before discussing OKN systems in the primate CNS that deal with these fixation-related issues.

--A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

--A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

The afferent system has two jobs: Scrutinize an object of regard (central channel), which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.
Motility Disorders: Supranuclear Syndromes

Supranuclear

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The head is still. It does this via continuous accommodative esotropia and fusional vergence. If the head is not still, the afferent system usually does not maintain a precise head fixations. Thus, lesions of a supranuclear pathway manifest as difficulties with either the vergence system; clinically, the two most important are convergence insufficiency, and spasm of the near. Infranuclear motility disorders (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for maintaining an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN). The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the head, and a Wallenberg syndrome.

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is in motion. The vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) is responsible for maintaining fixation on an object that is moving toward or away from the eyes. A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the parafoveal macula on the retina, ie, the central channel, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth. But let’s consider what it takes to accomplish these tasks.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts of the same plane on the opposite side. So, a rapid horizontal head turn to the right leads to an excitatory head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head in a given plane.

Thus, lesions of a supranuclear pathway manifest as difficulties with either the vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) or the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN). The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome.

In primates, vision has two purposes: 1) to want to eat you, and 2) to take a minute. To accomplish these ends, the afferent visual system can be divided into two channels:

- A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.
- A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

The visual system has to precisely track it. Further still, the primate’s nuclear systems in the primate CNS that deal with these fixation-related issues consist of bifixation. Further still, the primate’s optokinetic system (aka optokinetic nystagmus) is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head will cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid right horizontal head turn will produce a leftward motion of the eyes. A burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus. That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus. Less common are disorders stemming from otolith issues, which have two classic manifestations:

- Ocular tilt reaction: Skew deviation accompanied by a head tilt.
- Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye.

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, maintenance or restoration of bifixation. But let's consider what it takes to accomplish these tasks.

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts of the same plane on the opposite side. So, a rapid horizontal head turn to the right leads to an excitatory head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head in a given plane.

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome.

The optokinetic system (aka optokinetic nystagmus) is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head will cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid right horizontal head turn will produce a leftward motion of the eyes. A burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts of the same plane on the opposite side. So, a rapid horizontal head turn to the right leads to an excitatory head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head in a given plane.

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The afferent system has two jobs: Scrutinize an object of regard (central channel), which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth. But let's consider what it takes to accomplish these tasks. In primates, vision has two purposes: 1) to detect objects, and 2) to want to eat you), and 2) to objects of interest (ie, to determine definitely whether it's an eat-er vs an eat-ee). To accomplish these ends, the afferent visual system can be divided into two channels:

- **A peripheral, low-resolution channel for detecting objects.** The peripheral channel is very sensitive to motion.
- **A central, high-resolution channel for scrutinizing objects.** Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), and the image will disappear. Further, the object might be moving, meaning the efferent system has to precisely track it. Further still, the primate's visual system has to precisely track it. Further still, the primate's vestibulo-ocular system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements (ie, saccadic, or visually disruptive slower refixation movements (nystagmus, not saccades)).

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then produce a constant torque to maintain this new direction. Thus, lesions of a supranuclear pathway manifest as difficulties with either the vergence system; clinically, the two most important are fusional vergence insufficiency, and accommodative esotropia, and spasm of the near. Other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

Infranuclear --Ocular tilt reaction : Skew deviation accompanied by a head tilt

--Skew deviation : A vertical deviation accompanied by intorsion of the hypertropic eye

Less common are disorders stemming from otolith issues, which have two classic manifestations: semicircular canal issues, usually manifesting with nystagmus, and otolith issues, which produce a constant microsaccadic refixation movements, either brief and rapid (VOR) or slower and sustained (OKN). Of these, semicircular canal issues are the most common, and may manifest with nystagmus. That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

The head's rotational movements can be divided into two categories: (1) Brief and rapid movements (VOR) or (2) Slower and sustained movements (OKN). VOR is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head. These bursts produce an excitatory burst from the canal that is opposite the direction of the movement. Thus, a rapid right horizontal head turn will produce a rapid leftward rotation of the eyes. The burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid head movement is responsible for maintaining fixation on an object that is moving toward or away from the eyes. The vestibulo-ocular system is responsible for maintaining fixation on a stationary object when the head is moving. The optokinetic system is responsible for maintaining fixation on an object that is moving toward or away from the foveas. The optokinetic system is responsible for maintaining fixation on a stationary object when the head is moving. The head's rotational movements can be divided into two categories: (1) Brief and rapid movements (VOR) or (2) Slower and sustained movements (OKN). VOR is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head toward which the rotation occurred, while simultaneously leading to an inhibitory burst from the canal of the same plane on the side of the head. These bursts produce an excitatory burst from the canal that is opposite the direction of the movement. Thus, a rapid right horizontal head turn will produce a rapid leftward rotation of the eyes. The burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid head movement is responsible for maintaining fixation on an object that is moving toward or away from the eyes. The vestibulo-ocular system is responsible for maintaining fixation on a stationary object when the head is moving. The optokinetic system is responsible for maintaining fixation on a stationary object when the head is moving.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In primates, vision has two purposes: 1) to detect objects of regard (central channel), which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth. But let's consider what it takes to accomplish these tasks.

The afferent system has two jobs: Scrutinize an object of regard (central channel), and scan the visual system. In order to rapidly refixate both foveas on a peripheral image, the efferent system must...
Motility Disorders: Supranuclear Syndromes

Supranuclear

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant microfixation pattern. The afferent system has two jobs: Scrutinize an object of regard (central channel), and when necessary, acquire new objects of regard. 

Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on one side of the body, and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

Thus, lesions of a supranuclear pathway manifest as difficulties with either the vestibulo-ocular reflex (aka the vestibulo-ocular reflex, VOR) or the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

Less common are disorders stemming from otolith issues, which have two classic manifestations: The ocular tilt reaction: Skew deviation accompanied by a head tilt; and skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye.

Supranuclear Syndromes

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Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

-- two words

-- three words

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant steady, or the photoreceptors (PRs) will fatigue too much.

The vestibulo-ocular system is responsible for maintaining fixation on an object that is moving toward or away from the eyes. It consists of the vestibulocochlear system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

The cause of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
Before discussing **supranuclear lesions**, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do *that*, we have to define the role of the *afferent* system. (Get comfy, this is gonna take a minute.)

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the *lateral medullary* or *Wallenberg syndrome*. Its VOR manifestation is *ataxia*; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a *Horner syndrome*. (Wallenberg syndrome is discussed in detail in slide-set *N3*.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with *nystagmus*.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

--Skew deviation
--Ocular tilt reaction

The **ocular fixation system** is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

**Supranuclear**

The **vestibulo-ocular system** is responsible for maintaining fixation on an object that is moving toward or away. The *vestibulo-ocular reflex* (aka the *vestibulo-ocular reflex, VOR*) and the *optokinetic system* (aka *optokinetic nystagmus, OKN*) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

Dysfunction of the VOR may manifest as difficulties with either the *directional preprogrammed system* or *vergence system*; clinically, the two most important are *fusional vergence* and *accommodative vergence*. The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

--Skew deviation
--Ocular tilt reaction

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.
Supranuclear

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:
--Skew deviation: A vert vs horiz deviation
--Ocular tilt reaction

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is stationary. This system is critical for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

The vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations.

Wallenberg syndrome is usually manifesting with nystagmus. At last, the payoff for all this backstory: The vestibular labyrinth, ie, the semicircular canals and otoliths, is controlled by the vestibular system; clinically, the two most important are vestibulo-ocular reflex, VOR and optokinetic system.

Supranuclear Syndromes

Motility Disorders: Supranuclear Syndromes

Supranuclear

The vestibulo-ocular system is responsible for maintaining a high-quality image of a stationary object when the head is stationary. This system is critical for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

Wallenberg syndrome is usually manifesting with nystagmus. At last, the payoff for all this backstory: The vestibular labyrinth, ie, the semicircular canals and otoliths, is controlled by the vestibular system; clinically, the two most important are vestibulo-ocular reflex, VOR and optokinetic system.

The supranuclear pathways consist of supranuclear lesions that deal with these fixation-related issues. Infranuclear motility disorders include convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

--Skew deviation: A vertical deviation

--Ocular tilt reaction

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements (saccadic) or visually disruptive slower refixation movements (nystagmus, not saccades).

Of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
The visual system has two jobs: Scrutinize an object of regard (central channel), and 2) to scrutinize objects of interest (ie, to determine definitely whether it's an eat-er vs want to eat you), and 2) to

- A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

- A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous refixation movements (microsaccades or saccades) and other mechanisms such as microvestigial saccades.

The vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

- VOR: Rapid, brief compensatory eye movements that counteract the movement of the head to keep the images stable on the fovea. It is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head produces bursts of eye movement in the opposite direction. The semicircular canals are sensitive to linear accelerations and canals are sensitive to angular accelerations.

- OKN: Slow, sustained eye movements that follow the movement of a patterned background. It is used to track moving objects and can help maintain fixation on a moving target.

The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

- Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye vs extorsion of the hypotropic eye

- Ocular tilt reaction

Supranuclear Syndromes

Motility Disorders: Supranuclear Syndromes

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In primates, vision has two purposes: 1) Keep both foveas pointing at the current object of regard; and 2) rapidly redirect both foveas to a new object when one is detected in the periphery.

It follows from this that the afferent system has two jobs: Scrutinize an object of regard (central channel), while also monitoring for other objects that may require scrutinization (peripheral channel). It consists of the retinal photoreceptors, which are responsible for converting light into neural signals, and the optic nerve, which carries these signals to the brain.

Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), and 2) to

The head is still. It does this via continuous refixation movements (ie, saccadic, or visually disruptive slower refixation movements (nystagmus, not saccades).
The head is still. It does this via continuous refixation movements (i.e., saccades, not nystagmus). Dysregulation of this system manifests as either visually disruptive refixation movements (i.e., saccadic, not nystagmus) or fatigue (and subsequent image loss) from occurring.

Shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PRs from the eyes, thus necessitating they converge or diverge. There are a number of components to the vergence system; clinically, the two most important are convergence insufficiency and divergent esotropia.

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, or maintenance of bifixation. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance of bifixation.

The inequivalence of image rotations—either brief and rapid (VOR) or slower and sustained (OKN)—is responsible for maintaining a high-quality image of a stationary object when the visual system has to precisely track it. Further still, the primate’s visual system has two jobs: 1) Keep both foveas pointing at the current object of regard; and 2) to detect objects of interest (i.e., to determine definitely whether it’s an eater vs. an eatee). To accomplish these ends, the afferent visual system can be divided into two channels: the peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

The afferent system has two jobs: Scrutinize an object of regard (central channel, MLF), while also monitoring for other objects that may require scrutinization (peripheral channel). It follows from this that the afferent system has to precisely track it. Further still, the primate’s visual system has two jobs: 1) Keep both foveas pointing at the current object of regard and 2) to detect objects of interest (i.e., to determine definitely whether it’s an eater vs. an eatee). To accomplish these ends, the afferent visual system can be divided into two channels: the peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

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The cause of VOR dysfunction can be a central lesion, usually in the brainstem. A classic example is the lateral medullary or Wallenberg syndrome. Its VOR manifestation is ataxia; other findings include a loss of pain and temperature sensation on one side of the face and the opposite side of the body, and a Horner syndrome. (Wallenberg syndrome is discussed in detail in slide-set N3.)

That said, the majority of VOR dysfunction cases stem from peripheral issues, i.e., disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations: --Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye --Ocular tilt reaction

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the visual system has to precisely track it. Further still, the primate’s visual system has two jobs: 1) Keep both foveas pointing at the current object of regard and 2) to detect objects of interest (i.e., to determine definitely whether it’s an eater vs. an eatee). To accomplish these ends, the afferent visual system can be divided into two channels: the peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

The vestibulo-ocular system is responsible for maintaining a high-quality image of a stationary object when the visual system has to precisely track it. Further still, the primate’s visual system has two jobs: 1) Keep both foveas pointing at the current object of regard and 2) to detect objects of interest (i.e., to determine definitely whether it’s an eater vs. an eatee). To accomplish these ends, the afferent visual system can be divided into two channels: the peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

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That said, the majority of VOR dysfunction cases stem from peripheral issues, ie, disorders of the semicircular canals or otoliths. Of these, semicircular canal issues are the most common, usually manifesting with nystagmus.

Less common are disorders stemming from otolith issues, which have two classic manifestations:

--Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye
--Ocular tilt reaction: accompanied by a

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous accommodation.

Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR acquisition of bifixation.

In primates, vision has two purposes: 1) to detect objects; and 2) to scrutinize objects (ie, to determine definitely whether it's an eater vs. an eat-ee). To accomplish these ends, the afferent visual system can be divided into two channels:

The central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.

The peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion.

In order to rapidly refixate both foveas on a peripheral image, the efferent system must reorient both eyes to the image. Movement of the eyes is caused by the vestibular labyrinth, ie, the semicircular canals and otoliths. Rapid rotation of the head will produce a burst from the canal of the same plane on the side of the head turn. So a burst from the right horizontal canal, and an inhibitory burst from the left horizontal canal. These bursts cause the eyes to rotate in the opposite direction at a rate proportional to the rate of the head turn. So a rapid right horizontal head turn will produce a rapid leftward rotation of the eyes.

Thus, lesions of a supranuclear pathway manifest as difficulties with either the vestibulo-ocular reflex (aka the vestibulo-ocular reflex, VOR) or the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

Less common are disorders stemming from otolith issues, which have two classic manifestations:

--Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye
--Ocular tilt reaction: accompanied by a
Motility Disorders: Supranuclear Syndromes

Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then it must ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.

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Less common are disorders stemming from otolith issues, which have two classic manifestations:
--Skew deviation: A vertical deviation accompanied by intorsion of the hypertropic eye
--Ocular tilt reaction: Skew deviation accompanied by a head tilt

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant vergence system; clinically, the two most important are fusional vergence and accommodative vergence.

VOR dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

But let's consider what it takes to accomplish these tasks. Scrutinizing an object requires steady bifixation—but not too steady, or the photoreceptors (PRs) will fatigue and the image will disappear. Further, the object might be moving, meaning the efferent system has to precisely track it. Further still, the primate's head might be moving, also necessitating object-tracking.

--A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on out.

At long last, the payoff for all this backstory: The supranuclear pathways consist of systems in the primate CNS that deal with these fixation-related issues. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.

The ocular fixation system is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, not microsaccadic) or visually disruptive slower refixation movements (nystagmus, not saccades).

The vergence system is responsible for maintaining fixation on an object that is moving toward or away from the eyes, thus necessitating they converge or diverge. There are a number of components to the vergence system; clinically, the two most important are fusional vergence (disparity between the retinas with respect to image location) and accommodative vergence (triggered by retinal image blur). Many forms of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.

The vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN).

In contrast, the OKN system is driven by images sweeping across the retina. Such a stimulus causes the eyes to pursue the image, then saccade in the opposite direction. (Note that, unlike the pursuit movements generated by the previously-discussed smooth-pursuit system, the pursuit movements produced by the OKN are involuntary.)

Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.

Motility Disorders: Supranuclear Syndromes

Supranuclear
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Dysregulation of this system manifests as either visually disruptive larger refixation movements (ie, saccadic, not microsaccadic) or visually disruptive slower refixation movements (nystagmus, not saccades).

The vergence system is responsible for maintaining fixation on an object that is moving toward or away from the eyes, thus necessitating they converge or diverge. There are a number of components to the vergence system; clinically, the two most important are fusional vergence (disparity between the retinas with respect to image location) and accommodative vergence (triggered by retinal image blur). Many forms of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.

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Subarachnoid systems in the primate CNS that deal with these fixation-related issues...
Motility Disorders: Supranuclear Syndromes

Supranuclear

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The vestibulo-ocular system (aka the vestibulo-ocular reflex, VOR) and the optokinetic system (aka optokinetic nystagmus, OKN) are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN). Many forms of vergence dysfunction can occur, including convergence insufficiency, divergence insufficiency, accommodative esotropia, and spasm of the near.
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A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on out.

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In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.

The **saccadic system** is responsible for rapidly shifting fixation from the current object of interest to a new one located in the visual periphery.

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**Supranuclear**

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At long last, the payoff for all this backstory: The **supranuclear pathways** consist of systems in the primate CNS that deal with these fixation-related issues.

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**six systems in the primate CNS that deal with these fixation-related issues**
In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.

**Supranuclear**

The **saccadic system** is responsible for rapidly shifting fixation from the current object of interest to a new one located in the visual periphery. These refixation movements are the result of a two-stage process. First there is the **pulse**, which is the burst of innervation that is needed to overcome inertia and get the eyes rapidly rotating into position to fixate the new object. The magnitude of the burst (and thus the speed of the rotation) is proportional to how far the eyes need to go, ie, the farther the rotational distance, the bigger the burst.

At long last, the payoff for all this backstory: The **supranuclear pathways** consist of six systems in the primate CNS that deal with these fixation-related issues. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.
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In primates, vision has two purposes: 1) to detect objects of interest (eg, things you may want to eat, or may want to eat you), and 2) to scrutinize objects of interest (ie, to determine definitely whether it’s an eat-er vs an eat-ee). To accomplish these ends, the afferent visual system can be divided into two channels:

--A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.
--A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on out.

Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), while also monitoring for other objects that may require scrutiny (peripheral channel). It follows that the efferent visual system has two jobs: 1) Keep both foveas pointing at the current object of regard; and 2) rapidly redirect both foveas to a new object when one is detected in the periphery.

But let’s consider what it takes to accomplish these tasks.

Scrutinizing an object requires steady bifixation—but not too steady, or the photoreceptors (PRs) will fatigue and the image will disappear. Further, the object might be moving, meaning the efferent system has to precisely track it. Further still, the primate’s head might be moving, necessitating object-tracking.

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Motility Disorders: Supranuclear Syndromes

The saccadic system is responsible for rapidly shifting fixation from the current object of interest to a new one located in the visual periphery. These refixation movements are the result of a two-stage process. First there is the pulse, which is the burst of innervation that is needed to overcome inertia and get the eyes rapidly rotating into position to fixate the new object. The magnitude of the burst (and thus the speed of the rotation) is proportional to how far the eyes need to go, ie, the farther the rotational distance, the bigger the burst.

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The supranuclear pathways consist of six systems in the primate CNS that deal with these fixation-related issues. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then ‘ramp down’ the amount of torque to the level needed to maintain gaze in this new direction.
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**Motility Disorders: Supranuclear Syndromes**

**Supranuclear**

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**Infranuclear**

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**Infranuclear**

The efferent system is responsible for rapidly shifting fixation from the current object of interest to a new one located in the visual periphery. These refixation movements are the result of a two-stage process. First there is the **pulse**, which is the burst of innervation that is needed to overcome inertia and get the eyes rapidly rotating into position to fixate the new object. The magnitude of the burst (and thus the speed of the rotation) is proportional to how far the eyes need to go, i.e., the farther the rotational distance, the bigger the burst. The next stage in the process is called the **step**, and involves the onset and maintenance of the tonic level of innervation needed to hold the eyes in their newly-reached position.

**Supranuclear pathways** consist of six systems in the primate CNS that deal with these fixation-related issues.

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**Six systems in the primate CNS that deal with these fixation-related issues**

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**Supranuclear**

In order to rapidly refixate both foveas on a peripheral image, the efferent system must first produce just enough torque to overcome inertia and rotate the eyes to this image, then 'ramp down' the amount of torque to the level needed to maintain gaze in this new direction.
Disorders of the saccadic system can take many forms: Saccadic intrusions (unwanted saccades pulling gaze off its intended target); alterations in saccade speed or accuracy.

At long last, the payoff for all this backstory: The supranuclear pathways consist of six systems in the primate CNS that deal with these fixation-related issues.

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Before discussing supranuclear lesions, we need to define the role of the efferent (ie, motor) component of the visual system. But before we do that, we have to define the role of the afferent system. (Get comfy, this is gonna take a minute.)

In primates, vision has two purposes: 1) to detect objects of interest (eg, things you may want to eat, or may want to eat you), and 2) to scrutinize objects of interest (ie, to determine definitely whether it’s an eat-er vs an eat-ee). To accomplish these ends, the afferent visual system can be divided into two channels:

- A central, high-resolution channel for scrutinizing objects. Anatomically, this channel consists of the foveas, which provides high-resolution vision allowing objects to be examined in sharp detail and stereoscopic depth.
- A peripheral, low-resolution channel for detecting objects. The peripheral channel is very sensitive to motion. Anatomically, this channel consists of the rest of the retina, ie, the parafoveal macula on out.

Reiterating for emphasis: The afferent system has two jobs: Scrutinize an object of regard (central channel), while also monitoring for other objects that may require scrutinization (peripheral channel). It follows from this that the efferent visual system has two jobs: 1) Keep both foveas pointing at the current object of regard; and 2) rapidly redirect both foveas to a new object when one is detected in the periphery.

But let’s consider what it takes to accomplish these tasks. Scrutinizing an object requires steady bifixation—but not too steady, or the photoreceptors (PRs) will fatigue and the image will disappear. Further, the object might be moving, meaning the efferent system has to precisely track it. Further still, the primate’s head might be moving, also necessitating object-tracking.

At long last, the payoff for all this backstory: The supranuclear pathways consist of systems in the primate CNS that deal with these fixation-related issues. Thus, lesions of a supranuclear pathway manifest as difficulties with either the maintenance or acquisition of bifixation.

Disorders of the saccadic system can take many forms: Saccadic intrusions (unwanted saccades pulling gaze off its intended target); alterations in saccade speed or accuracy. The inability to initiate saccades is the hallmark of four-word dz name, and its abb. Infants with appear to be blind for the first several months of life, because their inability to initiate saccades gives the impression they have no interest in visual stimuli.

The saccadic system is responsible for rapidly shifting fixation from the current object of interest to a new one located in the visual periphery. These refixation movements are the result of a two-stage process. First there is the pulse, which is the burst of innervation that is needed to overcome inertia and get the eyes rapidly rotating into position to fixate the new object. The magnitude of the burst (and thus the speed of the rotation) is proportional to how far the eyes need to go, ie, the farther the rotational distance, the bigger the burst. The next stage in the process is called the step, and involves the onset and maintenance of the tonic level of innervation needed to hold the eyes in their newly-reached position.

Disorders of the saccadic system can take many forms: Saccadic intrusions (unwanted saccades pulling gaze off its intended target); alterations in saccade speed or accuracy. The inability to initiate saccades is the hallmark of congenital ocular motor apraxia (COMA). Infants with COMA appear to be blind for the first several months of life, because their inability to initiate saccades gives the impression they have no interest in visual stimuli. However, once the infants gain control over head movements (around age 2 months), they learn to induce horizontal versions by turning their heads and thereby inducing a VOR response. So for example, when they want to see an object that has moved to their right, COMA infants will jerk their head to the left, causing a VOR-mediated eye-turn to the right.
Motility Disorders: Supranuclear Syndromes

Supranuclear

Disorders of the saccadic system can take many forms: Saccadic intrusions (unwanted saccades pulling gaze off its intended target); alterations in saccade speed or accuracy. The inability to initiate saccades is the hallmark of **congenital ocular motor apraxia (COMA)**. Infants with COMA appear to be blind for the first several months of life, because their inability to initiate saccades gives the impression they have no interest in visual stimuli.

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**six systems in the primate CNS that deal with these fixation-related issues**
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**Supranuclear**

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Thus, lesions of a supranuclear pathway manifest as difficulties with either the **maintenance** or **acquisition** of bifixation.

**Motility Disorders: Supranuclear Syndromes**

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**Motility Disorders: Supranuclear Syndromes**

**Supranuclear**

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Motility Disorders: Supranuclear Syndromes

Supranuclear TLDR

Summary slides coming up…
1) The **ocular fixation system** is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous *microsaccadic refixation movements*, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.
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2) The **smooth-pursuit system** is responsible for maintaining fixation on a moving object. When it is impaired, pursuit movements may either lag behind the object or jump ahead of it. **Of note, that this is the only supranuclear pathway that is activated voluntarily.**
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4) The **vestibulo-ocular reflex (VOR) system** and the 5) **optokinetic nystagmus (OKN) system** are responsible for holding an image steady during head rotations—either brief and rapid (VOR) or slower and sustained (OKN). The VOR is controlled by the vestibular labyrinth, ie, the semicircular canals and otoliths; the OKN system, by images sweeping across the retina.
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An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it?

Motility Disorders: **Supranuclear Syndromes**

**Supranuclear TLDR**
1) The **ocular fixation system** is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous *microsaccadic refixation movements*, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

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**Motility Disorders: Supranuclear Syndromes**

**Supranuclear TLDR**

An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it?

It is this: With four important exceptions, **supranuclear pts do not complain of diplopia**

Why don’t most pts with supranuclear disorders have diplopia?
1) The **ocular fixation system** is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous microsaccadic refixation movements, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

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An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it? It is this: With four important exceptions, **supranuclear pts do not complain of diplopia**.

Why don’t most pts with supranuclear disorders have diplopia? Because most supranuclear disorders affect both eyes in a symmetric fashion.
Motility Disorders: Supranuclear Syndromes

Supranuclear **TLDR**

1) The **ocular fixation system** is responsible for maintaining a high-quality image of a stationary object when the head is still. It does this via continuous **microsaccadic re-fixation movements**, which produce a constant shifting among the PRs regarding which are responsible for the retinal image. This shifting prevents PR fatigue (and subsequent image loss) from occurring.

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supranuclear pts do not complain of diplopia

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6) The **supranuclear TLDR**

Motility Disorders: **Supranuclear Syndromes**

**Supranuclear TLDR**

An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it? **supranuclear pts do not complain of diplopia**

What are some of the supranuclear disorders that present typically, ie, without diplopia?

--Gaze palsies, eg, Parinaud syndrome
--Congenital ocular motor apraxia (COMA)
--Progressive supranuclear palsy (PSP)
--Saccadic disorders

Why don’t most pts with supranuclear disorders have diplopia?

Because most supranuclear disorders affect both eyes in a **symmetric** fashion.
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**Motility Disorders: Supranuclear Syndromes**

**Supranuclear TLDR**

An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it?

It is this: With four important exceptions, supranuclear pts do not complain of diplopia because most supranuclear disorders affect both eyes in a symmetric fashion.

What are some of the supranuclear disorders that present typically, ie, without diplopia?

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- Gaze palsies, eg, **Parinaud syndrome**
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Each of these is addressed in detail in other slide-sets—check the ToC.
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**An important rule-of-thumb can be stated regarding supranuclear motility disorders and diplopia—what is it?**

It is this: With **four important exceptions**, supranuclear pts do not complain of diplopia.

**What are the four supranuclear disorders in which pts c/o diplopia?**
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It is this: With **four important exceptions**, supranuclear pts do not complain of diplopia.

- Skew deviation
- Divergence insufficiency
- Convergence insufficiency
- Convergence spasm

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**Motility Disorders: Supranuclear Syndromes**

**Supranuclear TLDR**

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