

VS III, Ocular Motility and Binocular Vision
Spring 2007

Reading: Eye Movement Basics for the Clinician
Chapter 3

What is a Saccade: accurate, high velocity, (non ballistic?) eye movements used to foveate objects of interest. Present during reading, scanning, observation. Usually small in size, less than 15 degrees.

Three types of Saccades, Saccadic refixations (this lecture), microsaccades (last lecture) and saccadic oscillations and nystagmus(future lecture)

Parts of a saccade:

Movement response to a high frequency pulse that sets up the movement to overcome the resistance of the globe and orbit bringing the eye to its new position.

The eye is held in place by the **step** innervation which keeps the eye in position against the returning forces of the eye and orbit. A tonic force

Pulse step controller not only excites the agonist muscle but also inhibits the antagonist muscle.

Measurable characteristics of Saccades:

Latency

Velocity

Amplitude

- **Latency:** Time of onset from the initial movement of the target until the onset of the saccadic eye movement to foveate this displaced target. This latency is from 180 to 200 msec, with a standard deviation of 30 msec. The latency is not affected so much by physical traits of the target, such as size and luminance, but by soft variables such as patient motivation, attention, and target predictability.
Remember this for testing and training

Components of Latency;

Afferent neurosensory delay for the neural transmission to travel from the retina to the visual cortex to the high level centers of the brain involved in making decisions as to saccadic movement. 50 msec

Efferent delay of 30 msec as the neural transmission goes from other higher levels to the lower level processing within the midbrain.

Computational delay of 50 msec that is noncognitive.

Decision making processing delay of at least 50 msec where the brain decides if and where to change gaze involving higher level processing.

- **Velocity of Saccades:** The measured velocity for 20 degree saccade is shown in this chart for a variety of measurements. The upper limit for saccadic velocity is thought to be 750 degrees a second. (some readings will say 800 to 1000 msec) The velocity can not be altered voluntarily .Of interest is the relationship between the size of the saccade (amplitude) and the velocity. The larger the saccade, the faster the velocity. This relationship of increased amplitude being linearly related to increased velocity is called the main sequence. Not only is there a main sequence between velocity but also saccade duration, peak acceleration and deceleration. So the larger amplitude saccades produce the highest velocities, the largest peak accelerations and decelerations and the longest duration. The main sequence is a reflection of the pulse component of the pulse step controller signal for saccades. So when biomechanics of the system are normal the central system has a direct effect on speed, duration, peak acceleration and deceleration. There is a formula to calculate the duration ($2.2 \times \text{the amplitude} + 21 \text{ msec.}$)

Causes of reduced velocity:

With slowed velocity, first suspect drug ingestion. Anticonvulsants, sedatives and antidepressants are the most common.

Abnormally fast saccades can be traced to error in calibration of equipment, remember there are limits in saccadic speed due to the main sequence. This would be an unexpected problem

- **Amplitude of Saccades:** Remember that saccades are usually less than 15 degrees. Errors in amplitude give rise to the classification system or hypometric and hypermetric found in our text.

For horizontal saccades, the movement in the abducting eye tends to be larger, faster and shorter in duration. The two eyes are not completely conjugate during saccades, but close.

For Vertical saccades the eyes are more conjugate, although in upward saccade they diverge horizontally and converge in downwards saccades. This fits into the normal demand on the eyes to diverge in up gaze and converge in down gaze and when prescribing prism we may get some divergence with BD prism and Convergence with BU prism.

The Incredible Saccadic machine: not all saccadic facts are related to the physical machine of muscles and but also the higher central control.

- Saccadic suppression;

Elevation of the visual threshold during a high velocity saccade. This occurs before, during and after a saccade. The amount of suppression is dependent on target and background characteristics. This suppression is central neural inhibition.

- Before Saccade: related to future saccadic planning
- During Saccade: keeps the world clear and normal
- After Saccade: prevents the effects of retinal image motion from affecting vision.

Suppression alone is not strong enough to account for lack of smeared vision so:

- Saccadic omission and masking:

Saccadic omission involves visual masking. This is when a target is obscured by a preceding or succeeding visual stimulus. A high contrast target with lots of contours is most effective in masking. Masking is the primary factor that contributes to the absence of smear.

Testing of masking:

If a flash is shown just for 1 to 5ms of a 50 to 70 msec saccade, the subject had no smearing.

If a flash is on during the entire saccade of 50 to 70 msec, every thing appeared smeared. This illumination allowed smearing to occur

If the flash was shown before the saccade, or after the saccade as well as during the saccade, no gray out was reported.

During the saccade, acuity measured as 20/1000 or worse. Masking occurred due to the presence or an immediate or succeeding visual fixation and visual stimulation . Masking occurs independently of the eye movements.

Neurons in the striate cortex and superior colliculus are responsible for the saccadic omission and suppression.

- **Saccades are a sampled data system model**

Retinal error is sampled via fixation by a motor impulse modulator at 200msec intervals. This interval is the refractory period. Sampling occurs with the onset of any target movement, as long as no saccade occurred in the previous 200 msec. Target changes that occur between samples are not sampled until the next sampling period. This information generates a corrective saccade. Position errors of less than 0.3 degrees are not corrected as they fall in the dead zone.

- **Modifications of sampled data system,**

Relative refractory period: depending on the timing of a second target displacement, the initial motor command and response can be modified. When the second movement is close to the original in time, the eye movement ignores the initial displacement, as the time between movements is increased to the 200msec refractory period, the movement reflects the first target position.

Under every day conditions, the sampled data system rules, when erroneous programming occurs, a second rapidly programmed saccade is executed to correct the initial saccade.

- **Dead Zone of Saccadic Eye Movements:**

Engineering concept referring to the threshold region. Stimulus change can be noticed, but is too small to warrant a response. The saccadic dead zone is approximately ± 0.25 to 0.30 degrees. This keeps the object in the foveal area. But with time due to slow drift, the retinal image gets to the edge of the dead zone and movement occurs.

The aging Saccade;

- Latency increases 1-2msec a year
- Peak Velocity decreases 1 deg/sec/year
- Saccadic gain, accuracy and anticipation do not change.
- Changes in higher level processing and neural transmission are responsible.

Vergence Change in Saccade,

The abducting eye generally has a greater amplitude, peak velocity and shorter duration than the adducting eye. This results in fixation disparity at the final point of fixation, leading to a corrective movement of a conjugate drift and convergence. This occurs in both horizontal and vertical saccades. With vertical saccades, up gaze leads to divergence, down gaze to convergence.

Short term Saccadic Adaptation:

- Normal self correcting dynamic change in effective calibration of saccades, probably due to the cerebellum
- Reduces the probability of inaccurate saccades by monitoring the system error. Signal to response difference
- Adaptation can occur in as few as 70 saccades. The time course of adaptation is exponential.
- The system response to decrease is faster and easier and better than the response to increase. A decrease reflects a reduction in gain, while an increase is a specific end point adjustment. The system is better in learning how to give an appropriate initial signal to make a saccade, then in learning how to adjust after it has made the saccade.
- The individual is unaware of this adaptation.

Prediction of Saccades:

- If given a predictable target, learning will occur.
- A saccade is said to be predictive when the return times range from 200 msec before to 150 msec after the target moves.
- Predictable saccades are generally hypometric.
- Within 5 cycles of repetitive target motion, prediction occurs.
- Age is not a factor.

Order of Saccades:

Two ways to classify:

Volitional Saccades: Saccades made electively as part of purposeful behavior

1. Predictive, anticipatory: Saccades generated in anticipation of a target appearing at a particular location
2. To remembered target: Saccades generated to a remembered location,
3. Antisaccades: saccades generated in the opposite direction to the object's appearance after instructed to do so.
4. To command: Saccades generated on cue

Reflexive Saccades:

1. Saccades generated to new stimuli.
2. May be a visual, auditory or tactile
3. Stimuli are unexpected.

Spontaneous Saccades: Seemingly random saccades that occur when the subject is not required to perform any particular behavioral task

Quick phase; Saccades during the quick phases of nystagmus. Generated during vestibular or Optokinetic stimulation or as automatic resetting movement in the presence of motion.

The worse the system the lower the level of saccades.

In Basic eye Movements for the Clinician: saccades are classified as normometric or dysmetric:

Normometric: a single accurate movement with the appropriate gain and dynamics. The neural controller signal has a single and precisely matched pulse-step combination. This is a perfect eye movement.:

Dysmetric: single or multiple-step movements without appropriate gain. There are two types of dysmetric saccades: hypometric and hypermetric. Hypometric are too small, undershoots and hypermetric are too large, overshooting the target.

Efference Copy: When saccades occur a neural signal called an efference copy is generated that not only directs the eye to move but also sends message to the higher level brain center that the eye has moved not the world. This keeps the perception of stability alive.

Post Saccadic drift:

After a horizontal saccade some postsaccadic drift occurs. It is both disconjugate (vergence) and conjugate (version)

- The conjugate element is in the same direction as the saccade
- The disconjugate is convergent to help correct for divergence that happened during the saccade.
- Glissade is another term for the drift.
- It is due to a mismatch between the size of the pulse and the tonic step.
- Fatigue increases Glissades.

Dynamic Overshoot:

- Dynamic overshoot is a small saccade in the opposite direction after a saccade.
- This is due to transient reversal in the central saccadic overshoot

Vision therapy can greatly improve the patient's accuracy of saccades

Neuroanatomic control of and signal processing for saccadic eye movements:

Two main controls for Saccades:

1. Higher level control: The primary structures involved in selecting the target, localizing and calculating the desired change in eye position, as well as shaping of the final neural signal.
2. Lower level control processes, including the structures involved in the actual generation of the pulse-step controller signal to the oculomotor neurons.

Also remember that when a saccade is generated, it sends a neural signal called the efference copy which is motor based information to other higher level brain centers to inform the brain that the world has not shifted.

Primary Higher Level control:

Lesions on one side of the frontal lobe lead to saccade error to the opposite gaze area. When the lesion is in the right frontal lobe error in saccade to the left, and left lobe controls saccades to the right.

The next lower level process involves the generation of the pulse-step neural controller signal. Two types of saccadic neurons in the brain, Burst cells and Pause cells. These are located in the brain stem tegmentum and generate the immediate, premotor and saccadic velocity signal:

After a saccade is generated, the eye is held in position by a tonic step command that is generated by the brainstem neural integrator.

The individual pulse and step components combine at the oculomotor neurons to become the pulse step controller signal that goes to the EOM's to produce a saccade.

Burst Neurons : for horizontal saccades are located in the paramedian pontine reticular formation, (PPRF) or pons.

For Vertical and torsional saccades in the rostral interstitial nucleus of the medial longitudinal fasciculus.

Three kinds of burst cells: located in the pons in the PPRF

1. Short- lead excitatory burst neurons: begin high frequency firing just before and during a saccade. They produce the pulse of neural activity correlated with the peak velocity and amplitude of a saccade.
2. Inhibitory burst neurons: Located next to the abducens nucleus for the horizontal saccades. Sends axons across the midline to the opposite Abducens nucleus to inhibit the contra motoneurons during ipsi saccades Also goes into the vestibular nuclei, nucleus prepositus, and parts of the PPRF. For vertical or torsional saccades, the inhibitory cells may be located in the riMLF just like the excitatory burst cells.
3. Long- lead excitatory burst neurons: exhibit firing rates that are of low frequency and irregular and activity occurs prior to the saccade. Involved with synchronization of overall premotor saccadic pulse generation.
 - Stimulation of the PPRF will generate an ipsilateral saccade
 - PPRF=burst cells=saccade.
 - Unilateral lesions in the PPRF abolish the ability to generate ipsilateral saccades,
 - Loss of saccades to the right, think right lesion to the PPRF.

Pause Neurons:

- located in nucleus raphe interpositus of the midbrain,
- fire continuously except just before and during a saccade.
- They inhibit the EBN during saccadic free periods preventing unwanted saccades.

Sequence of events:

- Pause cells receive information from higher level centers such as the superior colliculus and the frontal eye fields and perhaps also the LLBN that a saccade is being planned. These signals inhibit the pause cells.
- The EBNs are now free to fire. The EBN signal is the pulse component of the pulse-step saccadic neural signal.
- The pulse signal bifurcates: it goes to the oculomotor neurons as well as to the neural integrator. The neural integrator for horizontal saccades is located in the nucleus prepositus hypoglossi and in the medial vestibular nucleus, for vertical saccades it is in the interstitial nucleus of Cajal.
- The neural integrator converts this eye velocity-coded information into eye position coded information. The pulse becomes a step.
- The individual pulse and step components combine at the oculomotor neurons to become the pulse step controller signal that is transmitted to the appropriate oculomotor nerve and then to the extraocular muscles to produce a saccade.

- What happens to the antagonist muscle during this process? We know about the yoked agonist in the other eye, but not the antagonist on the same eye. The antagonist may receive an inverse innervational change. Stops work during the saccade via an inhibition of innervation called an off-pulse. At the end of the movement the antagonist then goes to its new tonic innervation called the off – step.

Types of abnormal saccades:

- Slowed dynamics:
Peak velocity is below normal limits. Saccade duration is also prolonged.
Failure to develop or a disturbance in the normal pulse step neural controller signal.

Seen in:

MS

Parkinson's

Alzheimer's

AIDS

Thyroid ophthalmopathy
Myasthenia gravis...good signal, fatigue during saccade
EOM palsy or paresis
Frontal lobe lesions
Drug toxicity
Advanced Age

- Inaccurate amplitude: Dysmetric saccades occur either hyper or hypo. Due to gain fluctuations and biases in the cerebellum.

Seen In:

Amblyopia
MS
Parkinson's
Cerebellar disease
Hemianopsia
Alzheimer's
Frontal lobe lesion
Parietal lobe lesion

- Delayed initiation: Increase in latency. Due to signal processing and decision making. May be also involved in diseases like MS where there is demyelination.

Seen In:

Amblyopia
MS
Parkinson's
Alzheimer's
Frontal lobe lesions
Parietal or parietoccipital lobe lesions
Unilateral hemispheric cerebral lesions

Top Conditions with dysmetria:

Parkinson's disease; Disorder of the extrapyramidal brain nuclei caused by a dopamine deficiency that results in impaired neural inhibition.

Signs; Rigidity
Akinesia, bradykinesia and tremor

For eye movements there is a degeneration of dopaminergic neurons in the substantia nigra, a structure in the brain that is involved in the saccadic pathway and projects into the superior colliculus.

Hypometria

Increased latency

Reduced peak velocity

Errors more common in voluntary tracking than in reflexive ones

Multiple Sclerosis:

Progressive degeneration of white matter of the brain and spinal cord

Causes delay or disruption of neural transmission

Visual problems:

Diplopia

Blurred vision

INO, BINO

Nystagmus

Optic neuritis

General muscle problems:

Weakness

Spasticity

Tremor

Hyper-reflexia

Ataxia

Saccadic abnormalities

Increased Latency

Dysmetria

Decreased peak velocity

Increased duration, especially on adduction due to the high prevalence for MLF problems. In conduction

Heat makes the symptoms worse... shoes, hot weather, exercise

Myasthenia Gravis

Autoimmune disease

Acetylcholine released from the presynaptic membrane is not as effective.

Antibodies are bonded to postsynaptic ACH receptor motor endplates.

Produces intermittent conduction blockages of neural signals

Abnormalities

Dysmetria

Variable waveforms

Increased duration without decreased peak velocity, due to fatigue

Latency is normal

Tensilon results in hypermetric saccades

AIDS

Cellular immune disorder resulting from HIV(human immunodeficiency retrovirus)

Severe recurrent infections and neoplasms

Eye movement problems may be the first sign of frank neurological involvement.

Hard to see clinically

Saccadic problems include

Hypometria

Decreased peak velocity

Increased duration

Normal latency

Slowed saccades reflect a defect in burst cells of the PPRF not of cortical regions in which such neurologic involvement is absent,