Brien Holden Vision Institute

SACCADES

AUTHOR

Thomas Salmon: Northeastern State University, USA

PEER REVIEWER

Scott Steinman: Southern California College of Optometry, USA

THIS CHAPTER INCLUDES A REVIEW OF:

- Measurable Characteristics of Saccades
- Saccadic Alterations
- Models of Saccades
- Factors for Saccadic Changes
- Orders of Saccades
- Components of Saccades
- Neuroanatomic Control of and Signal Processing for Saccadic Eye Movements
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INTRODUCTION

WHAT IS A SACCADE?

A saccade is a 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.

It is an accurate, high velocity, (non ballistic?) eye movement used to bring objects of interest on the fovea. It is present during reading, scanning, and observation. Saccades are usually small in size, less than 15 degrees.

TYPES OF SACCADES	Three types of Saccades: 1. Saccadic refixations (this lecture) 2. Microsaccades (last lecture) 3. Saccadic oscillations and nystagmus (future lecture)

COMPONENTS OF A SACCADE	**Step Innervation** holds the eye in place and position against the returning forces of the eye and orbit. This is known as a tonic force The **pulse step controller** not only excites the agonist muscle but also inhibits the antagonist muscle.

MEASURABLE CHARACTERISTICS OF SACCADES

- Latency
- Velocity
- Amplitude

LATENCY	Latency refers to the time delay that occurs from the onset of the initial movement of the target to the onset of the saccadic eye movement exerted to foveate the displaced target. This latency ranges from 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. This is an important consideration when testing and training. **COMPONENTS OF LATENCY** **Afferent neurosensory delay:** This is the time it takes for the neural transmission to travel from the retina to the visual cortex, to the high-level brain centres involved in making decisions regarding saccadic movement. This delay lasts 50 msec. **Efferent delay** This is the time it takes for the neural transmission to travel from other higher levels to the lower level processing centres within the midbrain. This delay lasts 30 msec. **Computational delay** This delay is noncognitive and lasts 50 msec. **Decision-making processing delay** **This delay is due to the time it takes** for the brain to decide if and where to change gaze. This decision-making involves the higher level processing and it takes at least 50 msec.
VELOCITY	The upper limit for saccadic velocity is thought to be 750 degrees per second (some readings will say 800 to 1000 msec). The velocity cannot be altered voluntarily. It is interesting to note that the relationship between the size of the saccade (amplitude) and the velocity is linear. The larger the saccade, the faster is the velocity. This relationship is called the main sequence. Not only is there a main sequence between the size and velocity but also with saccade duration, peak acceleration and deceleration. So the larger amplitude saccades produce the highest velocities, the largest peak accelerations and decelerations and are sustained for the longest duration. The main sequence is a reflection of the pulse component of the pulse step controller signal for saccades. So when the biomechanics of the system are normal, the central system has a direct effect on speed, duration, peek acceleration and deceleration. The formula to calculate the duration is as follows: **Duration = 2.2 X the amplitude + 21 msec** **CAUSES OF REDUCED VELOCITY** With slowed velocity, suspect drug ingestion. Anticonvulsants, sedatives and antidepressants are the most common causes of slowed velocity. Abnormally fast saccades can be traced to error in calibration of equipment. It is important to note that there are limits in saccadic speed due to the main sequence. This would be an unexpected problem.
AMPLITUDE	Remember that the size of a saccade is usually less than 15 degrees. Errors in amplitude give rise to the classification system; hypometric and hypermetric. 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 an 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. The higher central control also plays an important role.

SACCADIC ALTERATIONS

SACCADIC SUPPRESSION	A saccadic suppression is an attempt to reduce the effects of smeared vision. It is the 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 the result of 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 completely counteract the effects of smeared vision however. **Saccadic Omission and masking** involves another alteration that further reduces smearing.
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 has no smearing. ▪ If a flash is on during the entire saccade of a 50 to 70 msec saccade, everything appears smeared. It is the illumination that allows smearing to occur. ▪ If the flash is shown before the saccade, or after the saccade as well as during the saccade, no smearing is reported. Acuity is measured as 20/1000 or worse if a flash is on during the entire saccade. Masking occurs due to the presence of an immediate or succeeding visual fixation and visual stimulation. Masking therefore occurs independently of eye movements. Neurons in the striate cortex and superior colliculus are responsible for the saccadic omission and suppression.

MODELS OF SACCADES

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 msecs. The 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 THE 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	This is an 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.

FACTORS AFFECTING SACCADIC CHANGES

THE AGEING SACCADE	▪ Latency increases by 1-2 msec a year ▪ Peak Velocity decreases by 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 SACCADES	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. During a vertical saccade, up gaze leads to divergence, while down gaze leads to convergence.
SHORT TERM SACCADIC ADAPTATION	This is a normal self-correcting dynamic change in effective calibration of saccades and is probably due to the cerebellum ▪ This adaptation reduces the probability of inaccurate saccades by monitoring the system error. This is referred to as the 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 a saccade is faster, 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 at learning how to give an appropriate initial signal to make a saccade and then learning how to make adjustments after it has initiated 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 the target moves 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

CLASSIFICATION BASED ON GENERATION FACTOR	Two ways to classify: **Volitional Saccades** Saccades made electively as part of purposeful behaviour 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. **Anti-saccades:** 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** • Saccades generated to new stimuli. • May be a visual, auditory or tactile stimuli • Stimuli are unexpected. 1. **Spontaneous Saccades:** A seemingly random saccade that occurs when the subject is not required to perform any particular behavioural task 2. **Quick Phase:** Saccades during the quick phases of nystagmus are generated during vestibular or optokinetic stimulation or as automatic resetting movement in the presence of motion. The worse the system, the lower is the level of saccades
CLASSIFICATION BASED ON BASIC EYE MOVEMENTS	Saccades are classified as normometric or dysmetric: **Normometric:** It is 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:** It is a single or multiple-step movement without appropriate gain. There are two types of dysmetric saccades: hypometric and hypermetric. Hypometric saccades are too small and tend to undershoot the target, while hypermetric saccades are too large and tend to overshoot it.
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 centre that it is the eye that has moved and not the world. This keeps the perception of stability alive.
POST SACCADIC DRIFT	After a horizontal saccade, some post-saccadic drift occurs. It is both disconjugate (vergence) and conjugate (version) ▪ The conjugate element is in the same direction as the saccade ▪ The disconjugate element is convergent to help correct for the 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 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 the final neural signal.
2. **Lower level control:** It includes the structures involved in the actual generation of the pulse-step controller signal to the oculomotor neurons.

Remember that when a saccade is generated, it sends a neural signal called the efference copy to other higher-level brain centres. This neural signal is motor-based information 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, an error in saccade occurs to the left and similarly left lobe lesions affect 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 signals. 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	Burst neurons are responsible for: ▪ **Horizontal saccades:** For these particular saccades, the burst neurons are located in the paramedian pontine reticular formation (PPRF) or pons ▪ **Vertical and torsional saccades:** For these particular saccades, the burst neurons are located in the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) Three kinds of burst cells are located in the pons in the PPRF: 1. **Short-lead excitatory burst neurons:** They begin high frequency firing just before and during a saccade. They produce a pulse of neural activity, which correlates with the peak velocity and amplitude of a saccade. 2. **Inhibitory burst neurons:** They are located next to the abducens nucleus for the horizontal saccades. These neurons send axons across the midline to the opposite abducens nucleus to inhibit the contralateral motorneurons during ipsilateral saccades. These axons also go 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:** They exhibit firing rates that are irregular and of low frequency. The activity occurs prior to the saccade. These excitatory burst neurons (EBN) are involved in the 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 – In cases where there is loss of saccades to the right, consider the presence of a right lesion to the PPRF.

PAUSE NEURONS	▪ Located in the nucleus raphe interpositus of the midbrain ▪ Fire continuously except just before and during a saccade. ▪ They inhibit the Excitatory Burst Neuron (EBN) during saccadic free periods preventing unwanted saccades.
SEQUENCE OF EVENTS	▪ Pause cells receive information that a saccade is being planned from higher-level centres such as the superior colliculus and the frontal eye fields and perhaps also the LLBN (Long-lead burst neurons). 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 in the same eye. The antagonist may receive an inverse innervational change. The antagonist muscle stops working 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.

ABNORMAL SACCADES

Types of abnormal saccades:

SLOWED DYNAMICS	A slowed dynamics saccade is where the peak velocity is below normal limits. Saccade duration is also prolonged, due to failure to develop or a disturbance in the normal pulse step neural controller signal.	Seen in: ▪ MS ▪ Parkinson's ▪ Alzheimer's ▪ Frontal lobe lesions ▪ AIDS ▪ Thyroid ophthalmyopathy ▪ Myasthenia gravis - good signal, fatigue during saccade ▪ EOM palsy or paresis ▪ Drug toxicity ▪ Advanced Age
INACCURATE AMPLITUDE	Dysmetric saccades occur as either hypermetric or hypometric, due to gained fluctuations and biases in the cerebellum.	Seen In: ▪ MS ▪ Parkinson's ▪ Alzheimer's ▪ Frontal lobe lesion ▪ Amblyopia ▪ Parietal lobe lesion ▪ Cerebellar disease ▪ Hemianoposia

DELAYED INITIATION	This refers to an increase in latency, due to signal processing and decision-making. It may also be exacerbated in diseases like MS where there is demyelinization.	Seen In: ▪ MS ▪ Parkinson's ▪ Alzheimer's ▪ Frontal lobe lesions ▪ Amblyopia ▪ Parietal or parieto-occipital lobe lesions ▪ Unilateral hemispheric cerebral lesions

MOST COMMON 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 – absence or loss of control of voluntary muscle movements ▪ Bradykinesia – slow movement ▪ 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 that causes delay or disruption of neural transmission **Visual problems** ▪ Diplopia ▪ Blurred vision ▪ INO, BINO – (Bilateral) Internuclear Ophthalmoplegia ▪ 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 **Saccadic abnormalities** ▪ Dysmetria ▪ Variable waveforms ▪ Increased duration without decreased peak velocity, due to fatigue ▪ Latency is normal ▪ Tensilon (edrophonium chloride*) results in hypermetric saccades – *Edrophonium chloride is a competitive antagonist of skeletal muscle relaxants and is a diagnostic agent in myasthenia gravis

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** ▪ 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

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