Saccade Testing:
A Comprehensive Clinical Review

Australian Dizziness Clinics | **www.AustralianDizzinessClinics.com**
Version 3.0 | April 2026
Section 3A — Oculomotor Assessment | Vestibular Function Testing Series

How to Use This Review

This document is the companion clinical literature review to the saccadic function and VNG saccade assessment video series on the ADC education hub at www.australiandizzinessclinics.com. It is designed for vestibular physicians, audiologists, and neurologists building expertise in laboratory vestibular function testing.

The review follows clinical testing sequence: from theoretical foundations and neural substrates through methodology, normative values, interpretation frameworks, and clinical application. Callout boxes throughout identify clinically high-yield points and evidence-based pearls.

Callout box guide:

 Clinical Insight: *Clinically relevant observations derived directly from the basic science — the bridge between laboratory findings and patient management.*

 Clinical Pearl: *High-yield, memorable clinical points — the key facts that separate a competent clinician from an expert in vestibular function testing.*

 Key Point: *Foundational concepts and summary statements that anchor the clinical framework. Master these to interpret the full testing battery.*

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Clinical Neuro-Otology of Saccadic Eye Movements

A Comprehensive Review of Physiology, Assessment, and Pathological Significance

1. Introduction:

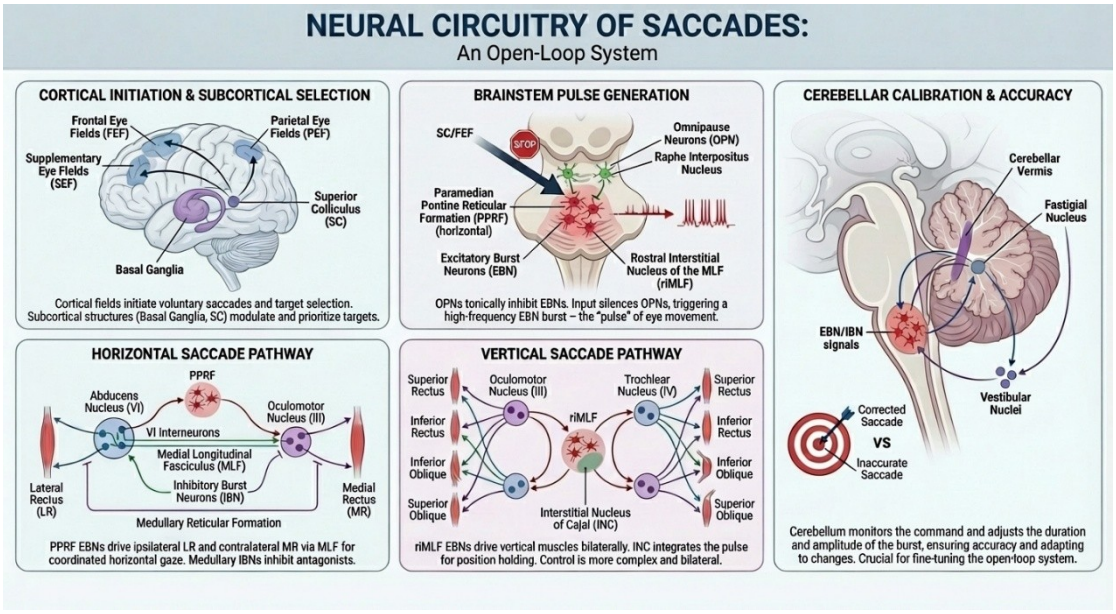
For the vestibular physician, saccades – the quick, conjugate eye movements that rapidly redirect gaze – are more than just the means to scan the environment; they are a powerful window into the integrity of the brain's oculomotor network. Alongside smooth pursuit and the vestibulo-ocular reflex (VOR), saccades form a cornerstone of the ocular motor exam [1]. Evolutionarily, saccadic eye movements provide the mechanism for ballistic refocusing of visual attention: in a fraction of a second, they reposition the fovea onto new targets of interest, enabling high-acuity vision of the object at the centre of gaze. These high-velocity movements (often reaching several hundred degrees per second) are essentially pre-programmed – once initiated, a saccade cannot be altered mid-flight – distinguishing them from the continuous feedback-driven nature of smooth pursuit.

Clinically, the behaviour of saccades offers invaluable diagnostic information, especially in distinguishing central from peripheral vestibular disorders. Whereas peripheral vestibulopathies (inner ear or VIII nerve lesions) generally spare the saccadic system, central pathologies often perturb it in characteristic ways. For example, a patient with acute vestibular syndrome (AVS)

due to vestibular neuritis will usually generate normal saccades with normal speed and accuracy. In contrast, a patient with a brainstem or cerebellar stroke may exhibit slow or dysmetric saccades (abnormally reduced velocity or overshoot/undershoot of targets), a red flag finding highly suggestive of central involvement. Thus, saccades complement the head impulse and nystagmus tests (as in HINTS+) by providing a direct assay of brainstem and cerebellar function in dizzy patients. Beyond the acute setting, saccadic examination can reveal subtle signs of neurodegenerative disease (e.g. parkinsonian syndromes, ataxias) and medication effects long before other clinical symptoms emerge.

This report provides an exhaustive review of the saccadic eye movement system. We will discuss the anatomical pathways and neurophysiology underlying saccade generation, outline bedside and laboratory methods for assessing saccadic function, and explore the spectrum of saccadic abnormalities – from slowed or dysmetric saccades to intrusions like square-wave jerks and opsoclonus – and their clinical implications. Emphasis is placed on practical clinical utility: understanding how specific saccadic signs (e.g. a unilateral adduction lag or a pattern of hypermetric saccades) can pinpoint lesion localization or differentiate a benign vestibular disorder from a dangerous stroke. By the end, the vestibular physician will appreciate saccades not merely as rapid eye movements, but as a sensitive barometer of central ocular motor pathways that can greatly enhance diagnostic accuracy in neuro-otology.

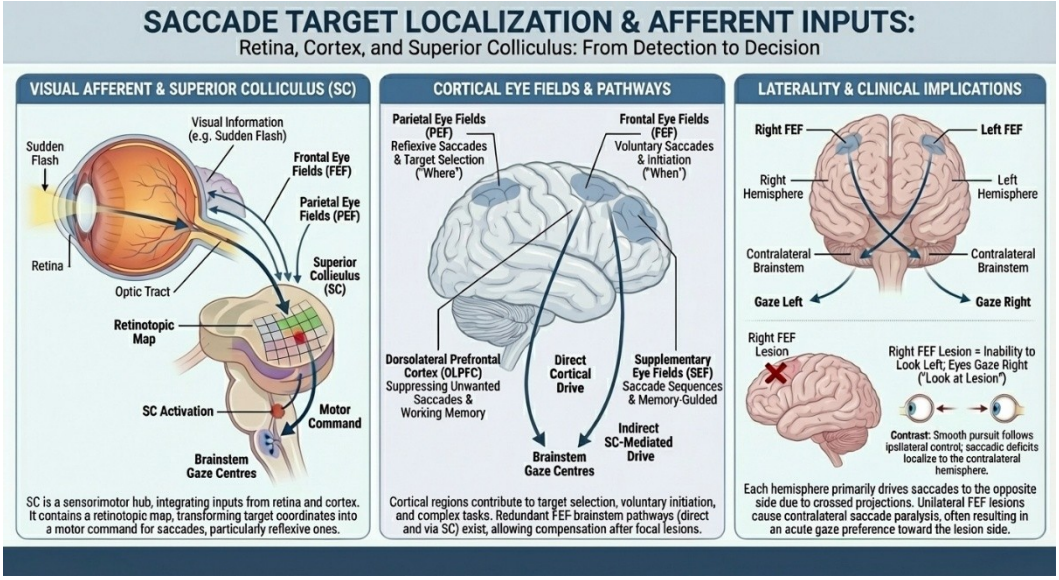
2. Neurophysiology and Anatomical Substrates of Saccades



Overview: The neural circuitry for saccades is a complex, high-speed system that transforms sensory information about target location into a precisely timed burst of motor activity to move the eyes. It involves a **widespread network: cortical eye fields for decision and initiation, subcortical structures for target selection, brainstem burst neuron centres for generating the pulse of eye movement, and the cerebellum for calibration and accuracy.** Unlike smooth pursuit, which operates as a continuous feedback loop, saccades are generated in an open-loop fashion – they require an initial "ballistic" command that then relies on cerebellar fine-tuning to hit the target accurately. Below we detail the afferent and efferent pathways of this system, highlighting key structures **(frontal and parietal eye fields, superior colliculus, basal ganglia, brainstem burst and omnipause neurons, and cerebellum)** and noting distinctions between horizontal and vertical saccadic control.

2.1. Target Localization and Afferent Inputs: Retina to Superior Colliculus and Cortical Eye Fields

Every saccade begins with the detection of a target or the decision to shift gaze. Visual afferents play a primary role in reflexive saccades: retinal input about a salient target (e.g. a sudden flash in the periphery) travels via the optic tract to the superior colliculus (SC) – a layered structure in the dorsal midbrain that serves as a master map for saccades [2]. The SC contains a retinotopic map of the visual field; activation of a particular locus in the SC drives a saccade to the corresponding region of space. Notably, the SC receives converging inputs from multiple sources (retina, visual cortex, parietal cortex, frontal eye field), integrating sensory information and higher-order signals (like attention or intention) to decide if and where to move the eyes [2, 2]. In essence, the SC is a key sensorimotor hub for saccades: it transforms visual (or auditory/somatosensory) target coordinates into a motor command.



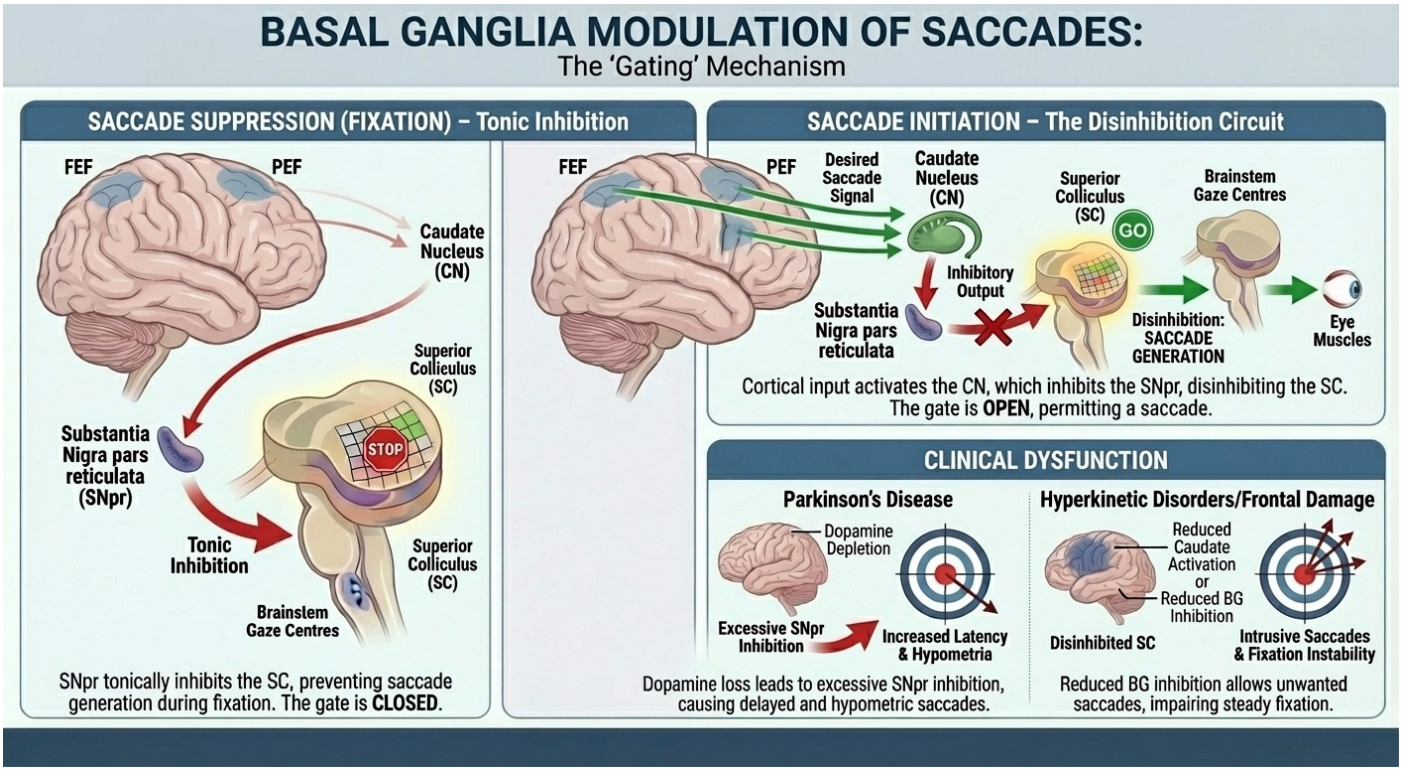
In parallel to the SC, cortical regions contribute to target selection and voluntary saccade initiation. The **Parietal Eye Field (PEF),** located in the posterior parietal cortex (Brodmann areas 7 and 39/40), is crucial for visually guided, reflexive saccades and attention-driven target selection [1]. It helps decide the "where" of a saccade by signalling which object in the visual scene is most salient. The **Frontal Eye Field (FEF)** in the posterior middle frontal gyrus (Brodmann area 8) is responsible for the "when" – the initiation of voluntary saccades and more cognitively driven eye movements [1]. The FEF can trigger saccades independent of a visual stimulus (e.g. on command or to remembered targets) and also adjusts saccadic velocity. Importantly, the FEF projects both directly to the contralateral brainstem gaze centres and indirectly via the superior colliculus [1, 1]. The FEF-SC relationship provides dual pathways for saccade generation: a direct cortical drive and a SC-mediated drive. This redundancy is why a single lesion in one pathway may not completely abolish saccades – for example, a focal FEF lesion might be partially compensated by intact SC circuits, and vice versa [3].

Laterality: A fundamental rule for saccadic control is that each cerebral hemisphere primarily drives **saccades toward the opposite side.** The right FEF directs gaze to the left, and the left FEF directs gaze to the right [1]. This is achieved by crossed projections: FEF neurons descend and decussate to activate contralateral pontine burst neurons (for horizontal saccades) and bilateral midbrain burst neurons (for vertical saccades) [1, 1]. Clinically, this contralateral control means a unilateral frontal lobe lesion (e.g. a right FEF stroke) can cause an inability to voluntarily look to the left, often manifesting as a transient gaze preference toward the side of the lesion (the classic "eyes look at the lesion" sign in an acute frontal stroke). This contrasts with the smooth pursuit system, which follows an "ipsilateral" control rule. The vestibular physician can leverage this difference: smooth pursuit asymmetry localizes to the ipsilateral hemisphere, whereas saccadic deficits localize to the contralateral hemisphere or brainstem side, a nuance that aids accurate lesion localization.Beyond the FEF and PEF, other cortical areas contribute

to saccades. The **Supplementary Eye Field (SEF)** on the medial frontal lobe is involved in planning sequences of saccades and memory-guided eye movements. The **Dorsolateral Prefrontal Cortex (DLPFC)** plays a role in suppressing unwanted saccades (e.g. in the antisaccade task) and in working memory for spatial targets. Lesions in these higher-order regions do not produce an obvious paralysis of saccades, but rather more subtle deficits: increased saccadic distractibility (inability to suppress reflexive glances to irrelevant stimuli) or difficulty with complex saccade tasks (like antisaccades or remembering a target location).

2.2. Basal Ganglia Modulation: Gating of Saccades

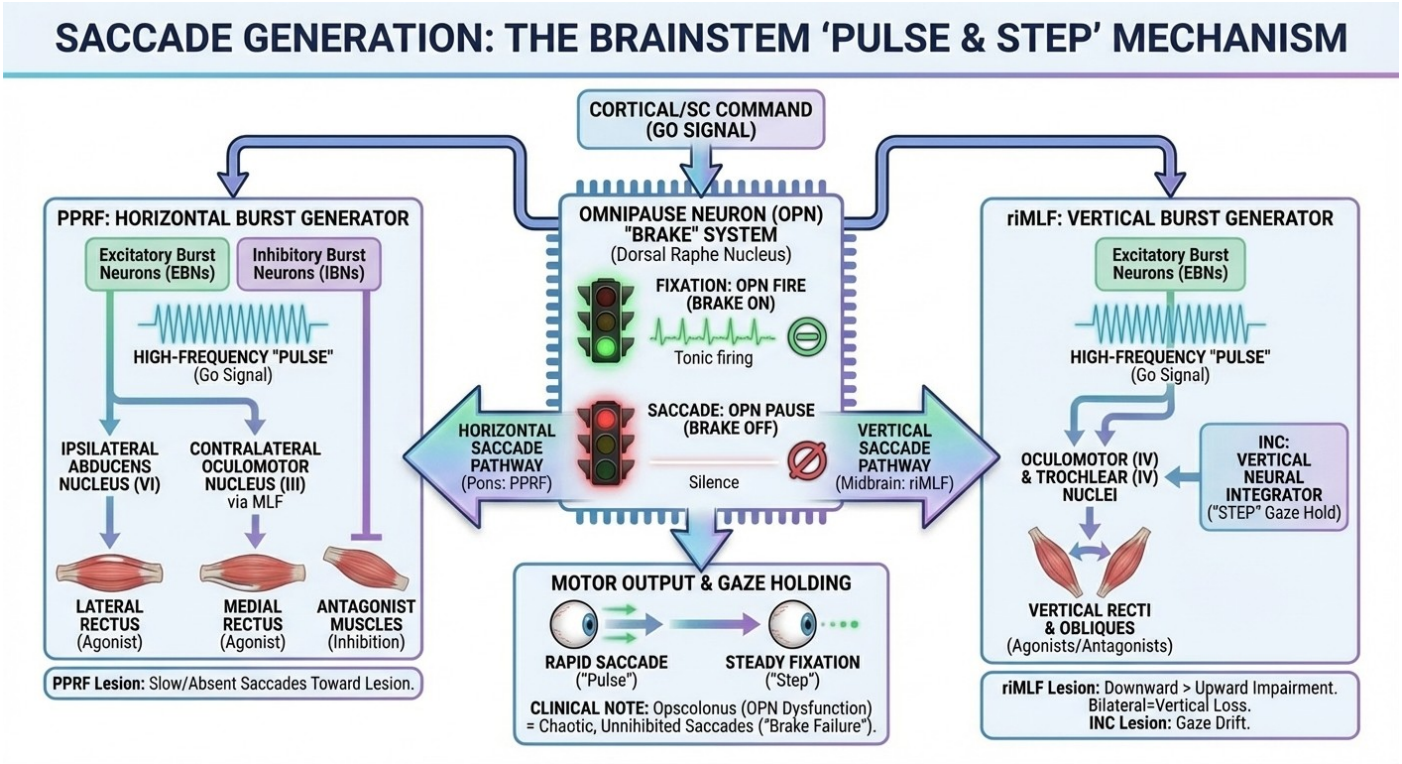
Interposed between the cortical eye fields and the brainstem, the basal ganglia act as a **gatekeeper for saccadic initiation.** The caudate nucleus (input nucleus of the basal ganglia) receives signals from the FEF and PEF regarding desired saccades. Through the inhibitory output of the substantia nigra pars reticulata (SNpr), the basal ganglia tonically suppress the superior colliculus [2, 2]. To initiate a saccade, this inhibition must be released: the caudate, when activated by cortical input, inhibits the SNpr, which in turn disinhibits the SC. This classic "disinhibition" circuit is crucial for permitting a saccade to occur. It prevents random, unwanted saccades during fixation and ensures that a saccade is initiated only when a proper target is selected. Dysfunction in this system is exemplified by Parkinson's disease, where dopaminergic depletion leads to excessive inhibition of the SC by the SNpr. The result is a higher threshold for saccade initiation and often mild hypometria (small undershooting saccades) due to impaired generation of full-scale motor commands. Patients with Parkinson's may have normal or near-normal saccadic velocities, but they exhibit increased latency (slower to initiate saccades) and a tendency to undershoot targets, needing corrective catch-up saccades. This reflects the difficulty in overriding the basal ganglia "brake." Conversely, conditions with reduced basal ganglia inhibition (e.g. certain hyperkinetic movement disorders or frontal lobe damage affecting the caudate) can result in intrusive saccades or inability to maintain steady fixation.



2.3. Brainstem Burst Neurons and Omnipause Neurons: The Pulse Generator

At the final common pathway for saccades are the brainstem burst neuron networks, which generate the high-frequency bursts of action potentials that drive the ocular motor nuclei. These networks are distinct for horizontal and vertical saccades:

- **Horizontal Saccades:** The horizontal burst generator is located in the **paramedian pontine reticular formation (PPRF)** of the pons [1]. The PPRF on each side contains excitatory burst neurons (EBNs) that project to the ipsilateral abducens nucleus (cranial nerve VI). When activated, the right PPRF EBNs, for example, send a powerful burst to the right abducens nucleus, causing the right lateral rectus to contract, while internuclear neurons from CN VI simultaneously activate the left oculomotor nucleus via the medial longitudinal fasciculus (MLF) to contract the left medial rectus [2, 2]. This coordinated innervation produces a conjugate rightward saccade (right eye abducts, left eye adducts). For leftward saccades, the mirror-image occurs via the left PPRF and left CN VI. In addition to excitatory burst neurons, the brainstem horizontal system includes inhibitory burst neurons **(IBNs) in the medullary reticular formation,** which during a rightward saccade will inhibit the antagonist muscles (in this case, the left lateral rectus and right medial rectus) by silencing the left abducens motor neurons [2]. The IBNs ensure that as the agonist muscles receive a “go” pulse, the opposing muscles get a simultaneous “stop” signal, allowing the eyes to move rapidly without resistance. Lesions of the PPRF effectively remove the burst input for ipsilateral horizontal saccades – the result is slow or absent saccades toward the lesion side, often a conjugate horizontal gaze palsy if the lesion is complete [2, 2]. For example, a pontine stroke damaging the right PPRF will cause an inability to generate rapid saccades to the right (the eyes will either not move or will drift slowly rightward, often with only delayed catch-up), whereas leftward saccades remain relatively normal.
- **Vertical Saccades:** The vertical burst generator resides in the **midbrain, in the rostral interstitial nucleus of the MLF (riMLF),** adjacent to the rostral end of the MLF and near the periaqueductal gray [1, 2]. There are distinct burst neurons for upward vs. downward saccades. Each riMLF projects to motor neurons for the vertical recti and oblique muscles (via CN III and IV nuclei) in a somewhat complex bilateral pattern: generally, each riMLF drives torsional and vertical components in both eyes, with a single riMLF lesion more markedly affecting downward saccades (because downward burst neurons are thought to be less redundant) [2, 2]. A unilateral riMLF lesion can cause slow or poorly initiated downward saccades, mild impairment of upward saccades, and a loss of ipsitorsional quick phases (e.g. on head tilt, quick phases of nystagmus toward the side of the lesion are missing) [2, 2]. A bilateral riMLF lesion (as seen in progressive supranuclear palsy, PSP, which affects the rostral midbrain) causes severe slowing or complete loss of vertical saccades. Vertical saccade generation also involves the **interstitial nucleus of Cajal (INC),** which is not a burst generator per se, but a neural integrator that holds vertical gaze. INC lesions lead to vertical gaze drift and asymmetric saccade outcomes (e.g. after a vertical saccade, one eye may drift due to impaired gaze holding).



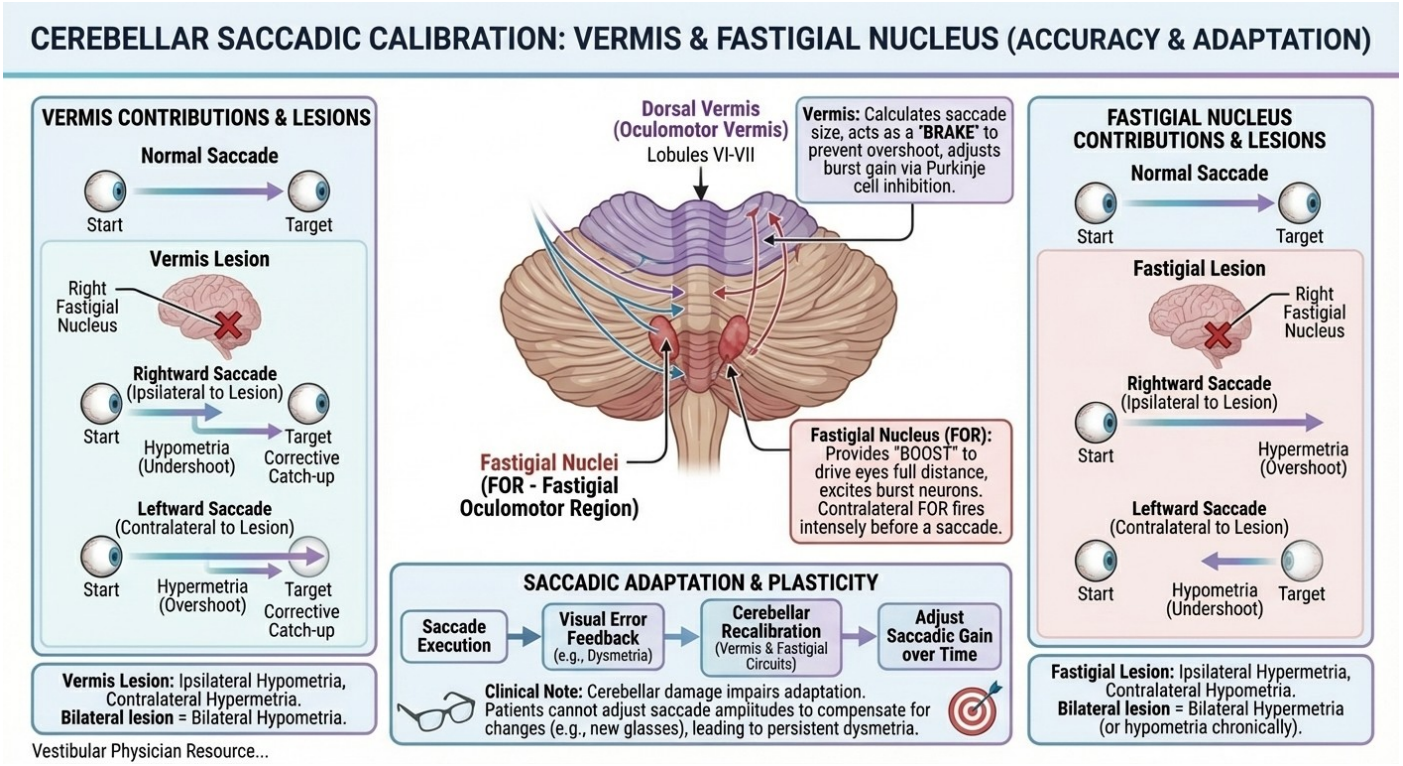
Crucial to both horizontal and vertical systems is the presence of **Omnipause Neurons (OPNs)** in the **dorsal raphe nucleus (nucleus raphe interpositus) of the pons.** These specialized neurons act as a saccadic "brake." During fixation, omnipause neurons fire continuously, exerting inhibitory tone on the burst neuron circuits to prevent unwanted saccades [4, 4]. When a saccade is initiated (from cortical/SC command), the omnipause neurons suddenly cease firing, releasing the inhibition on burst neurons, which then fire their high frequency burst to create the saccade [4]. At the end of the saccade (just a few tens of milliseconds later), the omnipause neurons resume firing to clamp down the burst and terminate the eye movement, ensuring the eye stops on target. This pause-and-resume mechanism times each saccade's duration. If omnipause neurons fail to hold the burst in check, abnormal eye movements result: for instance, lesions or dysfunction of OPNs are thought to underlie opsoclonus, the chaotic burst of back-to-back saccades without inter-saccadic intervals [4, 4]. In opsoclonus (discussed later), it is as if the braking mechanism is completely removed, allowing continuous saccadic firing. Conversely, an OPN that fails to pause (for example, due to certain pontine lesions) would prevent saccades from initiating, locking the eyes in fixation despite intact burst neuron capability.

To summarize the brainstem mechanism: a saccade is generated when cortical/SC input triggers a cessation of omnipause firing and a simultaneous activation of burst neurons in the PPRF (for horizontal) or riMLF (for vertical). The burst neurons send a pulse signal to the extraocular motor nuclei, causing the eyes to move at high speed. This pulse is then followed by a lower-frequency step signal (from neural integrator networks and cerebellum) to hold the eye at the new position. The conjugacy of eye movement is maintained by interneuronal connections like the MLF, which coordinates lateral rectus of one eye with medial rectus of the other.

2.4. Cerebellar Contributions: Vermis and Fastigial Nucleus in Saccadic Accuracy

The cerebellum is the **great calibrator of saccades.** Two regions in particular **– the dorsal cerebellar vermis (lobules VI-VII, including the oculomotor vermis)** and the **deep cerebellar fastigial nuclei** (especially the fastigial oculomotor region, FOR) – are critical for saccadic accuracy and adaptation [2]. While the brainstem burst neurons provide the raw "pulse" for a saccade, the cerebellum fine-tunes that pulse to ensure the eyes land exactly on target (correct amplitude) and do so in a single, smooth movement without appreciable overshoot or undershoot.

- **Dorsal Vermis (Oculomotor Vermis):** The vermis is thought to calculate the necessary saccade size and send corrective signals to adjust the burst gain (the relationship between burst firing and eye movement). Vermal Purkinje cells, via their projections to the fastigial nucleus, act almost like a brake to avoid overshooting. A lesion in one side of the dorsal vermis produces a characteristic saccadic dysmetria: saccades toward the ipsilateral side of the lesion are typically hypometric (undershoot), whereas saccades toward the contralateral side become hypermetric (overshoot) [2]. This happens because the vermis normally applies a breaking impulse to saccades; without the vermis on one side, saccades directed toward that side don't get enough brake (hence they fall short, requiring a catch-up) and saccades toward the other side get too little braking (from the lesioned side) and overshoot [2, 2]. Bilateral vermis damage (as in certain degenerative cerebellar ataxias) leads to bilateral hypometria, with all saccades falling short because the overall braking function is in excess [2]. Patients with degenerative cerebellar disease (e.g. spinocerebellar ataxias or alcoholic cerebellar degeneration) often show this bilateral undershoot of saccades combined with post-saccadic drift.
- **Fastigial Oculomotor Region (FOR):** The fastigial nucleus (one in each cerebellar hemisphere, deep within the vermis) exerts an opposite influence. The FOR excites burst neurons to help drive the eyes the full distance. In fact, just before a saccade, the contralateral fastigial nucleus fires intensely to push the eyes toward the target. A lesion of the fastigial nucleus (or its immediate output pathways) on one side causes ipsilateral saccades to overshoot (hypermetria) and contralateral saccades to undershoot [2]. This is essentially the inverse of the vermis effect, because the fastigial nucleus normally boosts saccade amplitudes. If both fastigial nuclei are lesioned, saccades to either side become small and hypometric (fastigial nucleus lesions often produce bilateral hypermetria initially due to loss of decussating inhibition, but chronic bilateral lesions tend toward hypometric saccades since both "boost" engines are gone) [2, 2]. In practice, bilateral fastigial lesions are rare (seen in surgical cerebellar tumour resections or severe atrophy) but cause a striking inability to make accurate saccades – patients will overshoot targets in both directions (fastigial ocular dysmetria).



The cerebellum is also essential for saccadic adaptation. If wearing new glasses or after a partial muscle paralysis, the brain must adjust saccade amplitudes to avoid constant dysmetria. The vermis and fastigial circuits, through error feedback (visual error detected after a dysmetric saccade), gradually recalibrate the saccadic gain over hundreds of trials. This plasticity is often tested experimentally by displacing a target during a saccade (tricking the brain into learning a new amplitude); patients with cerebellar damage show impaired adaptation, remaining dysmetric.

3. Clinical Assessment of Saccades

Evaluating saccadic eye movements at the bedside is a quick and informative component of the neuro-otologic examination. By observing the eyes as they make rapid refixation movements, the clinician can assess the integrity of cortical decision-making, brainstem execution, and cerebellar calibration in one fell swoop. Bedside saccade testing should systematically examine saccades in all principal directions (horizontal, vertical, and oblique) and note four key attributes: **latency** (delay from target command to movement onset), **velocity** (peak speed of the eye movement), **accuracy** (whether the saccade lands on target or is dysmetric), and **conjugacy** (coordination between the two eyes) [2, 2]. Each of these attributes can yield clues to specific dysfunctions, as detailed below.

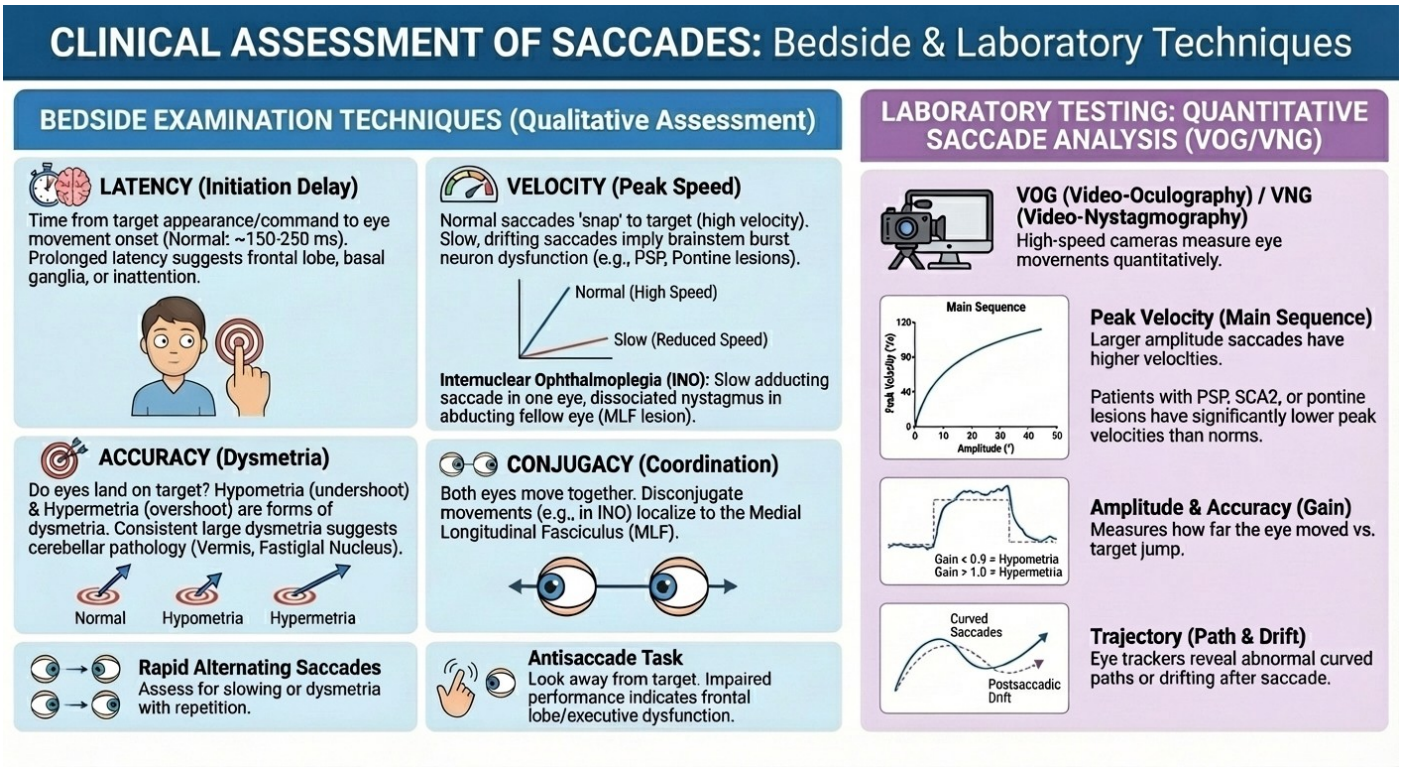
3.1. Bedside Examination Techniques

Setup: The standard approach is to have the patient fixate back-and-forth between two targets. A common method is the “nose-to-finger” test: the patient alternates gaze between the examiner’s nose (central position) and the examiner’s fingertip held ~15–20° to one side, repeatedly [2]. This is done horizontally (finger held to the left and right of centre) and vertically (finger held above and below). The examiner should ensure the patient’s head remains still (to

isolate eye movement) and that the targets are small and distinct (e.g. the tip of a pen or finger) to encourage a precise foveal saccade.

What to observe:

- **Latency:** Normally, there is a brief reaction time (~150–250 milliseconds) after a target appears or is commanded before the saccade begins**. Delayed initiation (prolonged latency) suggests either inattention, frontal lobe/executive dysfunction, or basal ganglia slowing**. For example, a patient with Parkinson's or frontal apraxia may take an abnormally long time to initiate each saccade.
- **Velocity:** Even though one cannot measure peak eye speed by eye, abnormally slow saccades are often apparent. A normal saccade "snaps" to the target in one quick motion. If the eyes appear to drift or take a noticeably long time to reach the target, saccadic velocity may be reduced. This is a critical sign of brainstem burst neuron dysfunction [2]. In internuclear ophthalmoplegia (INO), caused by a medial longitudinal fasciculus lesion (often due to MS or stroke), the hallmark is a slow adducting saccade in one eye with a dissociated horizontal nystagmus in the abducting fellow eye [2].
- **Accuracy (Dysmetria):** When the patient shifts gaze from the examiner's finger back to the nose (or vice versa), do the eyes land on target, or do they overshoot/undershoot? Consistent, large dysmetria is abnormal. Hypometria can be a nonspecific finding (seen in fatigue, Parkinson's, or cerebellar vermis lesions), whereas hypermetria strongly suggests cerebellar pathology (particularly dorsal vermis or fastigial nucleus dysfunction) [2, 2]. At bedside, overshoot when looking left but not right implies a lesion in the circuitry for leftward saccades [2].
- **Conjugacy:** Observe whether both eyes move together. INO is best seen on quick horizontal saccades and is highly localizing to a lesion in the ipsilateral medial longitudinal fasciculus [2].
- **Rapid Alternating Saccades:** Vestibular clinicians often have the patient perform self-paced saccades – rapidly looking back and forth between two targets as fast as possible [2]. Pay attention to whether saccades slow down or become markedly dysmetric after a few repetitions.
- **Antisaccade Task:** The patient is instructed: "When I wiggle my finger to one side, look to the opposite side by the same amount, instead of looking at my finger." Impaired antisaccade performance is seen in disorders of the frontal lobes (e.g. frontal cortex strokes, traumatic brain injury) and diffuse executive dysfunction [5, 6]. High error rates on antisaccades are a well-established marker of frontal/executive dysfunction and have been studied as a potential endophenotype in conditions like schizophrenia and ADHD [6, 7].



3.2. Laboratory Testing: Quantitative Saccade Analysis (VOG/VNG)

Video-oculography (VOG) systems are used to measure saccades with high-speed cameras. Key metrics include:

- **Peak Velocity:** The maximum speed reached by the eye. There is a well-known "main sequence" relationship: larger amplitude saccades have higher peak velocities [8, 8]. For example, a 10° horizontal saccade might reach ~300–400°/s [8]. In the lab, patients with PSP, certain spinocerebellar ataxias (e.g. SCA2), or pontine lesions will have peak velocities significantly lower than norms [9, 9].
- **Amplitude and Accuracy:** The system measures how far the eye moved vs. how far the target jumped. Gain < ~0.9 indicates hypometria, and >1.0 indicates hypermetria.
- **Trajectory:** Eye trackers can reveal curved saccades or postsaccadic drift.

Specialized clinics might utilize the antisaccade task with eye tracking to evaluate frontal lobe function quantitatively [5].

4. Pathophysiology and Disorders of Saccadic Eye Movements

4.1. Saccadic Dysmetria: Overshooting or Undershooting the Target

- **Hypermetric Saccades (Overshoot):** Classically cerebellar in origin. Lesions in the dorsal vermis remove the braking function on the ipsilateral side, causing overshoot of contralaterally directed saccades [2]. Lesions in the fastigial nuclei also lead to hypermetria [2]. Unilateral hypermetria is a strong localizing sign to an ipsilateral cerebellar lesion [2].

- **Hypometric Saccades (Undershoot):** Large hypometria indicates pathology. Diffuse cerebellar degeneration can cause bilaterally hypometric saccades. Parkinson's disease characteristically causes hypometric saccades due to the basal ganglia's influence.

4.2. Slow Saccades: Sluggish Eye Movements and Their Causes

- **Progressive Supranuclear Palsy (PSP):** Early impairment of vertical saccades – downgaze in particular becomes slow and limited.
- **Spinocerebellar Ataxia Type 2 (SCA2):** SCA2 can present with markedly slow horizontal saccades even before gait ataxia is prominent [9, 9]. In SCA2, saccades, though slow, may remain accurate [9, 9].
- **Brainstem Strokes:** Infarcts in the pons affecting the PPRF or in the midbrain affecting the riMLF will cause acute slow saccades or outright gaze palsy [2].

4.3. Saccadic Intrusions and Oscillations: Breaking Fixation

Saccadic intrusions reflect a disinhibition in the ocular motor system [4, 4].

- **Square-Wave Jerks (SWJ):** Small saccadic shifts (0.5° to a few degrees) away from target, then a correction ~200ms later [4]. Frequent or large SWJs are associated with degenerative diseases like PSP and cerebellar disorders [4, 4, 4]. Large-amplitude SWJs (>5°) are pathological [4]. Increased SWJ frequency is also seen in Parkinson's and Alzheimer's [6]. Pathological SWJs commonly point to PSP or cerebellar syndromes [4].
- **Macrosaccadic Oscillations:** Eyes make a series of saccades that oscillate around the target in a decreasing pendular amplitude [4]. Highly suggestive of **cerebellar fastigial nucleus lesions**.
- **Ocular Flutter and Opsoclonus:** Saccadic oscillations without intersaccadic intervals [4]. Ocular flutter is rapid horizontal oscillation [4]. Opsoclonus is chaotic, multi-vectorial saccades [4]. Both indicate severe brainstem dysfunction, often paraneoplastic or post-viral [4, 4]. Toxic causes like extreme phenytoin toxicity can also cause opsoclonus [10]. Square-wave pulses are pathological variants [4]. Opsoclonus is theorized to result from damaged omnipause neurons [4, 4].

5. Differential Diagnosis: Saccadic Clues to Diseases

5.1. Central vs. Peripheral Vestibular Disorders

- **Peripheral Vestibulopathy:** Purely peripheral lesions (like vestibular neuritis) do not affect saccades.
- **Central Vestibular:** Strokes often produce abnormal saccades (slow, dysmetric, or intrusive).

5.2. Cerebellar Ataxias and Degenerations

- **SCAs:** SCA2 causes slow saccades [9].
- **Wallenberg Syndrome:** Ipsilesional hypermetric and contralesional hypometric saccades occur ("Wallenberg dysmetria") [2, 2].

5.3. Parkinsonian Syndromes

- **Parkinson's Disease:** Mildly hypometric saccades with normal velocities.
- **PSP:** Early vertical saccadic slowing and frequent square-wave jerks.
- **MSA:** MSA-C can present with gaze-evoked nystagmus and saccadic dysmetria [4].

5.4. Demyelinating Disease: Multiple Sclerosis (MS)

- **INO:** MLF lesion causes slowing of the adducting eye saccade [2].

6. Pharmacological and Toxic Influences on Saccades

- **Benzodiazepines:** Reduce saccadic peak velocity (10–15% reduction) [11] and slightly prolong latency [3].
- **Anticonvulsants:** Phenytoin toxicity causes bilateral dysmetric saccades [12]. Barbiturates reduce saccade velocity [12].
- **Alcohol:** Impairs the cerebellar flocculus, leading to inaccurate saccades.
- **Lithium:** Toxicity causes saccadic dysmetria and some slowing.

7. Age-Related Changes in Saccadic Function

- **Reduced Velocity:** Saccadic peak velocity declines ~1°/s per year [6, 13, 13].
- **Increased Latency:** Reaction time increases with age [14].
- **Decline in Accuracy:** Seniors show more hypometria [14].
- **Square-Wave Jerks:** Become more frequent with age [4, 4, 4].
- **Quantitative datasets:** Normative datasets help identify if findings are pathological or just senescent [6].

8. Advanced Insights: Cognitive and Behavioural Aspects of Saccades

- **Frontal Executive Dysfunction:** Patients with frontal damage fail the antisaccade test dramatically [6]. High error rates correlate with cognitive tests [5, 5].
- **ADHD:** Increased rate of antisaccade errors and shorter latencies (impulsivity) [12, 7]. Difficulty maintaining fixation [12, 12].
- **Schizophrenia:** Robust antisaccade impairment [6]. This is considered an endophenotype [6, 6].
- **Fixation Control:** ADHD patients show deficits in fixation control [12, 7].

9. Conclusion

Saccadic eye movements, though lasting only milliseconds, speak volumes about the integrity of the nervous system. The clinical utility of saccade examination cannot be overstated. An antisaccade test can quantitatively reflect frontal executive capacity [5, 6]. Armed with the knowledge from both this saccade review and its smooth pursuit counterpart, the vestibular physician can approach each patient's ocular motor exam with confidence and nuance.

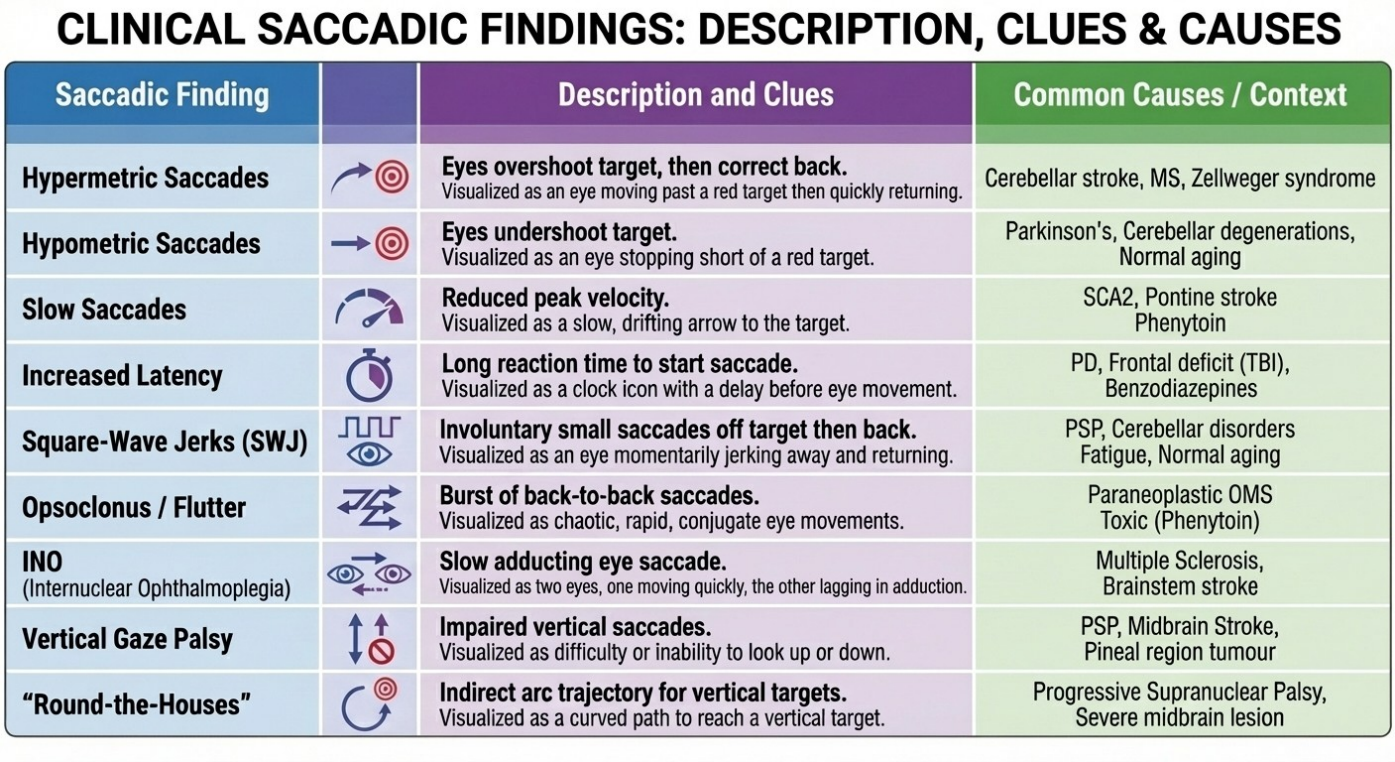


Table 1: Selected Saccadic Abnormalities and Their Clinical Significance

Saccadic Finding	Description and Clues	Common Causes / Context
Hypermetric Saccades	Eyes overshoot target, then correct back.	Cerebellar stroke, MS, Zellweger syndrome
Hypometric Saccades	Eyes undershoot target.	Parkinson's, Cerebellar degenerations, Normal aging
Slow Saccades	Reduced peak velocity.	PSP, SCA2 [9], Pontine stroke [2], Phenytoin [12]
Increased Latency	Long reaction time to start saccade.	PD, Frontal deficit (TBI), Benzodiazepines [3]
Square-Wave Jerks (SWJ)	Involuntary small saccades off target then back.	PSP [4], Cerebellar disorders [4], Fatigue [4], Normal aging [4]
Opsoclonus / Flutter	Burst of back-to-back saccades.	Paraneoplastic OMS [4, 4], Toxic (Phenytoin) [10]
INO	Slow adducting eye saccade.	Multiple Sclerosis, Brainstem stroke [2]
Vertical Gaze Palsy	Impaired vertical saccades.	PSP, Midbrain Stroke, Pineal region tumour
"Round-the-Houses"	Indirect arc trajectory for vertical targets.	Progressive Supranuclear Palsy, Severe midbrain lesion

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Accuracy and Currency

While every effort has been made to ensure the accuracy and completeness of the content, vestibular medicine is a rapidly evolving field. Clinicians are encouraged to verify specific protocols, normative values, and therapeutic recommendations against current published guidelines and primary literature.

References and Attribution

All referenced works are cited in good faith for educational purposes. Where specific normative data or clinical criteria are cited, the original sources should be consulted for full methodological detail and applicability to individual patient populations.

Version History

Version 3.0 — April 2026 | Full ADC standard rebuild with front matter, table of contents, callout boxes, and image-preserving reformatting.

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