Saccadic Intrusions and Oscillations

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ABSTRACT: There are a number of different types of involuntary saccadic eye movements which differ from normal saccades in that they take the fovea away from its target. This article presents a classification and description of these abnormal eye movements and indicates how they may be used to increase the precision of neurologic diagnosis.

Saccades are fast eye movements which occur in response to a variety of stimuli. They include voluntary re-fixations to targets, reflex movements toward a visual stimulus appearing suddenly off the fovea, and the fast phases of nystagmus induced by vestibular or optokinetic stimulation. The goal of all of these saccadic eye movements, is to place the target image at the fovea. However, there are other types of involuntary fast eye movements which take the fovea off the target. These include saccadic intrusions which are sporadic involuntary fast movements away from the target, and saccadic oscillations which are sustained oscillations that are initiated by fast eye movements. The latter should be differentiated from nystagmus where the oscillations are initiated by smooth eye movements.

These abnormal fast eye movements which disrupt visual fixation, occur in association with cerebellar, brainstem, or cerebral disease. Recognition of these movements can increase the precision of neurologic diagnosis.

Normal Fixation

Steady fixation is an illusion of the examiner. Fixation actually consists of three distinct types of miniature movements that cannot be detected by visual inspection of the eyes:

1) Microsaccades: These miniature saccades usually have amplitudes of less than 25 minutes of arc (average about 6 minutes). They occur at a mean frequency of approximately 120 per minute (Steinman et al., 1973). During finely guided visual motor tasks the amplitudes of microsaccades increase somewhat, despite reduction in their frequency. By ophthalmoscopy, the examiner can detect saccades as small as about 10 minutes of arc. Microsaccades have no known function (Kowler and Steinman, 1980). They are considered superfluous to visual perception.

2) Microdrift: Microdrift consists of smooth eye movements at rates less than 20 minutes of arc per second (Steinman et al., 1973; Ciuffreda et al., 1979). The drift appears to be necessary to prevent fading of a stable image. Most subjects effectively reduce the rate of microdrift during attentive fixation.

3) Microtremor: A continuous high frequency buzz of ocular motor activity underlies both microdrift and miniature saccades. Microtremor occurs at rates of 50 to 100 Hz. The average amplitude is much less than 1 minute of arc, usually 5 to 30 seconds (Steinman et al., 1973). Since the foveal bouquet of optimal visual acuity subtends about 20 minutes of arc (LeGrand, 1967) microtremor is too small to sweep the retinal image across any appreciable population of cones. There are only about 3 cone receptors per minute of arc in the foveal bouquet. Microtremor may consist of back-to-back saccades (Stark et al., 1981) but even the most refined oculographic techniques do not provide the resolution required to establish its saccadic composition.

Saccadic Intrusions

Saccadic intrusions are sporadic biphasic disruptions of fixation. Although one phase can be a smooth eye movement, the phase that takes the eyes off target is always a saccade (Herishanu and Sharpe, 1983; Dolsak et al., 1983). Saccadic intrusions include square wave jerks, saccadic pulses, double saccadic...
pulses, sporadic macro square wave jerks, and sporadic ocular bobbing (Table).

**TABLE: Classification of Intrusions and Oscillations.**

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* Although usually referred to as nystagmus, convergence-retraction pulses and voluntary flutter are not genuine nystagmus. They are initiated by saccades rather than by the slow (smooth) eye movements which initiate nystagmus.

**Square Wave Jerks**

Square wave jerks (SWJ) are sporadic horizontal conjugate saccades away from the intended position of fixation, followed after an interval by saccadic return to the fixation position (Herishanu and Sharpe, 1981). They vary in amplitude from 0.5 to 10 degrees (Figure IA). Many normal subjects have low amplitude, low frequency SWJ. The mean amplitude of normal square wave jerks is 1.2 degrees (SD 0.3) and their frequency is less than 10 per minute. Elderly subjects have more frequent SWJ. Their occurrence in older patients is less significant in the diagnosis of pathological fixation instability (Herishanu and Sharpe, 1981). SWJ may represent sporadically enlarged microsaccades.

Square wave jerks that exceed these normal frequencies and amplitudes are a feature of cerebellar system disease (Sharpe et al., 1982). They are prominent in cerebellar degenerations and in multiple sclerosis with cerebellar involvement. They also occur in Parkinson's disease (White et al., 1983). SWJ are more prominent in progressive supranuclear palsy (Troost et al., 1977) than in idiopathic Parkinson's disease, probably reflecting the cerebellar degeneration of the former condition (Steele et al., 1964), rather than basal ganglia dysfunction alone. Patients with nonaparalytic strabismus have SWJ while they fixate with the nondominant eye. The intrusions are related to the strabismus, not the amblyopia associated with strabismus (Ciuffreda et al., 1979).

SWJ also occur in patients with focal cerebral lesions. In a study of 17 patients with cerebral tumors, infarcts, or hemorrhages, seventy per cent of patients were found to have abnormally frequent SWJ (Sharpe et al., 1982). Comparison of the metrics of SWJ in patients with cerebellar system disease revealed that cerebellar SWJ were significantly lower in amplitude than cerebellar SWJ; the frequencies did not differ.

After the primary saccade of each SWJ couplet, which takes the eyes off target (Figure IA) the return saccade is usually triggered by visual feedback. Visually corrected saccadic errors have a latency of about 200 msec, so the duration of SWJ usually approximates that value. Occasionally very short duration SWJ occur. The short intersaccadic intervals indicate that nonretinal (internal, efference copy) feedback of eye position errors is used to generate the corrective return saccade (Figure 2). Although some eye position information is available from orbital proprioceptive inflow, the eye position information used for short-latency corrective movements is probably derived from an efference copy of eye position error caused by the saccadic intrusion (Sharpe et al., 1982).

Low amplitude SWJ are often undetected during inspection of the eyes. During ophthalmoscopy saccades as small as 10 minutes of arc can be detected as flicks of fundus landmarks. Blood vessels near the optic disc margin provide convenient landmarks to estimate the amplitudes of saccadic intrusions. Arteries adjacent to the poles of the disc, just distal to the second bifurcation of the central retinal artery subtend an angle of about 0.5 degrees. While the patient fixates a small distant object with one eye, low amplitude saccadic intrusions can be detected during funduscopic of the other eye (Sharpe et al., 1982). Horizontal flicks that equal or exceed the diameters of peripapillary arteries signify fixation instability. Their frequency can be timed for 1 minute. To and fro flicks of 0.5 degrees or more, each separated by an interval are SWJ. Observation of 10 or more to and fro flicks implicate abnormal SWJ. Although not of specific localizing value, they are a sensitive oculomotor index of brain dysfunction.

**Saccadic Pulses**

Saccades are produced by burst-tonic firing patterns of motor neurons. Single unit recordings in the paramedian pontine reticular formation have identified burst neurons that generate a phasic high frequency pulse of innervation (van Gisenbergen et al., 1981; Hepp and Henn, 1983). The pulse moves the eye rapidly against orbital viscous forces. Neural integration of the pulse is considered to yield a tonic change, called a step in other neurons of the tegmentum (Figure 2). This tonic step of innervation sustains the new eye position against elastic restoring forces in the orbit. If the step is absent, the eye returns to its previous position in a decreasing velocity exponential smooth eye movement. Saccadic pulses are composed of pulses of motor neuron innervation, without steps (Figure 1B). They are stepless saccades. Both conjugate saccadic pulses, and monocular saccadic pulses have been observed in patients with multiple sclerosis (Herishanu and Sharpe, 1983; Dolsak et al., 1983).

In internuclear ophthalmoplegia forward fixation may be disrupted by monocular abducting saccadic pulses in the eye opposite to the involved medial longitudinal fasciculus. The eye returns to the midposition in a decreasing velocity slow phase. The return toward midposition signifies a stepless saccade. Absence of the step is attributed to damage to the neural integrator, the neural network in the brainstem tegmentum which normally generates a tonic step of innervation and thereby sustains intersaccadic eye position (Herishanu and Sharpe, 1983). This waveform resembles gaze paretic nystagmus, but, unlike nystagmus, the fast phases of saccadic pulses take the eyes away from the fixation position.
Figure 1 — Examples of saccadic intrusions and oscillations. Horizontal calibrations indicate a time base of 500 msec. Vertical calibration marks signify 10 degrees for the eye position (Pos) traces and 200 deg/sec for the eye velocity traces (Vel). The small arrows in D indicate double saccadic pulses. Intrusions and oscillations in the left column are separated by intersaccadic intervals. Those in the right column have no interval between saccades. All intrusions and oscillations are composed of biphasic saccades, with the exceptions of convergence-retraction pulses, ocular bobbing (E) and saccadic pulses (B) which consist of monophasic stepless saccades followed by negative exponential return toward the fixation position. Recordings were obtained by infrared reflection oculography using ac coupled amplifiers with a bandwidth of 250 hertz. Vertical signals were recorded by conventional electrooculography. Velocities were obtained by electronic differentiation of analogue position signals.
During steady fixation saccades are held in abeyance by pause neurons in the midline of the caudal pontine tegmentum (Figure 2). Pause cells inhibit saccadic burst neurons in the paramedian pontine reticular formation. Saccadic pulses probably signify damage to the pause cells or their supranuclear inputs. The monocular abducting saccadic intrusions in patients with internuclear ophthalmoplegia are a sign of involvement extrinsic to the medial longitudinal fasciculi.

**Double Saccadic Pulses**

These saccadic intrusions are composed of isolated pairs of back-to-back saccades. They are sporadic conjugate movements. Double saccadic pulses have been reported in multiple sclerosis (Dolsak et al., 1983). They are also a sign of metabolic encephalopathy (see case description below). Brief disruption of pause cell activity may cause the intermittent back-to-back saccadic couplets (Figure 1D).

**Macro Square Wave Jerks**

These are sporadic large amplitude (4 to 30 degrees) horizontal fast eye movements to one side of fixation and back, separated by a brief interval. Often macro square wave jerks consist of a sustained salvo of oscillation (see below). They are considered to be a sign of damage to cerebellar outflow in the superior cerebellar peduncle (Dell’Osso et al., 1975).

**Saccadic Impersistence of Gaze**

Patients with diffuse cerebral cortical damage often exhibit large amplitude saccades away from the object of regard. After an interval of several hundred milliseconds the patient makes a saccade back to the target (Figure 3). This saccadic intrusion is a feature of Alzheimer’s type senile dementia. Clinically, the intrusions appear as furtive glances away from the examiner. Saccadic impersistence becomes quite prominent during attempted fixation of targets which shift position and during smooth pursuit. Anticipatory saccades occur in the direction of expected target motion. These anticipatory saccadic intrusions are involuntary in the sense that patients cannot willfully prevent their occurrence. Similar jerks occur sporadically in Huntington’s chorea when the background is rich in visual contours, but tend to subside when a single target is available for fixation, as under laboratory conditions (Leigh et al., 1983). In Alzheimer’s disease, saccadic impersistence occurs regardless of the visual surroundings.

**Ocular Bobbing**

Ocular bobbing consists of randomly occurring vertical saccades followed by delayed slow return to the midposition. These saccadic intrusions are less often detected than rhythmic ocular bobbing (see below) but they have the same significance. The intrusive saccade is usually downward (Susac et al., 1970) but upward bobbing also occurs (Knobler et al., 1981).

**Myoclonic Ocular Jerks**

This sporadic dyskinesia is composed of oblique, mainly upward, saccades followed after a variable interval (0 to 200 msec) by a saccade to the midposition. Familial myoclonus of skeletal muscles accompanies the disorder, but it is not synchronous with the eye movements. In one case reported, the jerks subsided with clonazepam treatment (Selhorst et al., 1983). Myoclonic ocular jerks are readily distinguished from...
ocular myoclonus which refers specifically to the vertical pendular nystagmus that is sometimes associated with palatal myoclonus (Daroff, 1977). Ocular myoclonus is not a saccadic oscillation.

**Saccadic Oscillations**

Saccadic oscillations occur as bursts or as nearly continuous disruptions of fixation (Table). They may be thought of as salvos of saccadic intrusions. Frequent intrusions may form a continuum with saccadic oscillations. Two fundamental variations can be identified by quantitative oculographic studies: those with intersaccadic intervals (Figure 1C, E and G) and those composed of back-to-back saccades (Figure 1D, F and H).

Saccades are initiated by supranuclear trigger signals that inhibit pause neurons in the midline pontine tegmentum. Inhibition of pause cells releases the discharge of burst neurons and the duration of their firing determines the amplitude of saccades. According to Robinson’s (1975) model, a signal of desired eye position (for example, retinal target error) which is independent of the trigger signal, determines how long the burst cells fire (Figure 2). Collaterals of burst neurons inhibit the pause cells to sustain the saccadic trajectory. The burst output is also mathematically integrated to yield both a new eye position command (the step) and an internal (extraretinal) signal of eye position (Figure 2). This efference copy is fed back to inhibit the burst neuron. Once the efference copy of actual eye position matches the desired eye position, the burst cells cease firing, the pause cells resume activity, and the saccade stops. Computer simulation studies of this model (Zee and Robinson, 1979) predict that burst cells on each side of the tegmentum will break into oscillations of back-to-back saccades if: 1) the trigger signal is inappropriately prolonged, 2) input to the pause neurons is inadequate to keep them active, or 3) if the pause cells are sick. Thus, the back-to-back saccades of double saccadic pulses, opsoclonus and flutter may be explained by a prolonged trigger signal, or inadequate pause cell activity. On the other hand, intrusions and oscillations having an interval between sequential saccades signify integrity of both pause cells that stop saccades and the neural integrator that sustains eye position between saccades.

**Square Wave Oscillation**

This disorder is a regular periodic oscillation in which each half cycle is indistinguishable from a sporadically occurring square wave jerk. The intersaccadic interval is of the order of 200 msec, a figure compatible with the visual reaction time. We have encountered square wave oscillations in Parkinson’s disease combined with alcoholic cerebellar degeneration (Figure 1C). They also occur in progressive supranuclear palsy (Abel et al., 1984), a condition characterized pathologically by degeneration of nigrostriatal pathways, the brainstem tegmentum, and the deep cerebellar nuclei (Steele et al., 1964). Square wave oscillations are horizontal and the eyes typically move to one side of the fixation position and back again (Figure 1C). Frequent blinking often accompanies this dyskinesia.

**Macro Saccadic Oscillations**

These are horizontal salvos that straddle the intended fixation position. Individual saccades are separated by an interval of about 200 msec. The amplitudes of sequential saccades increase, then decrease in a crescendo-decrescendo pattern (Selhorst et al., 1976). Amplitudes of 40 degrees or more are often attained. The spindles of oscillation usually occur during forward fixation (Figure 1G), but sometimes they are precipitated only by refixation saccades, or by smooth pursuit. Unlike macro square wave jerks, macro saccadic oscillations depend on visual feedback; they stop in darkness. Relatively acute structural damage in the dorsal cerebellum affecting the deep cerebellar nuclei is the clinicopathological correlate of this distinct oscillation. Metastatic tumor, hematoma, and demyelination at this site are documented causes (Selhorst et al., 1976). The following case illustrates macro saccadic oscillations caused by metabolic encephalopathy.

A 24 year old man with chronic renal failure due to Goodpasture’s disease presented with abdominal pain and fever of 2 days duration. He had been on chronic peritoneal dialysis. Investigation showed staphylococcus aureus peritonitis and aortic insufficiency. His course was complicated by hypoxic acidosis, hyponatremia and gastrointestinal bleeding. The patient lapsed into coma without any focal signs. A CT scan with serial overlapping cuts of the posterior fossa was normal.
After treatment of his multiple metabolic problems, he gradually regained consciousness. Two weeks after admission he had downbeat nystagmus in the primary position and frequent square wave jerks. Bursts of horizontal saccadic oscillation straddled the attempted fixation position. Oculographic recording showed spindles of macro saccadic oscillation (Figure 1G) and sporadic double saccadic pulses (Figure 1D). A repeat CT scan was normal. The ocular oscillations subsided over the next several weeks. The patient was discharged without any neurologic deficits and remained stable on ambulatory peritoneal dialysis.

Multiple metabolic abnormalities included uremia, sepsis, hypoxia, metabolic acidosis, and electrolyte imbalance. Despite the absence of clinical or radiological evidence of structural damage to the cerebellum, macro saccadic oscillations were a feature of the encephalopathy. Although function of the dorsal cerebellar vermis was possibly compromised, this case indicates that macro saccadic oscillations can be a manifestation of metabolic encephalopathy.

Microsaccadic Oscillations
Spindles of low amplitude horizontal oscillations straddle the intended fixation position in normal subjects. These microsaccadic oscillations have amplitudes of 1 degree or less and are not visible during inspection of the eyes. Identical microsaccadic oscillations occur in cerebellar degeneration (Hotson, 1982). The pathological oscillations are distinguished from normal microsaccadic oscillations only by their higher frequency. In cerebellar degeneration the frequency of spindles is about 8 per minute, as opposed to normal microsaccadic oscillations which occur about twice per minute.

Opsoclonus
Opsoclonus is the name for multidirectional back-to-back saccades of varying amplitude (Figure 1H) (Daroff, 1977; Toupet et al., 1982; Kuban et al., 1983). The saccades are often oblique or torsional. Disordered inhibitory control of pause neurons in the pons (Figure 2) may be the pathophysiological basis of opsoclonus (Zee et al., 1979). Although opsoclonus is typically associated with cerebellar signs (Ellenberger et al., 1972), dysfunction in the brainstem tegmentum is consistent with Orzechowski's (1927) original speculation about the source of this dyskinesia.

Cerebellar ataxia, myoclonic jerks of the limbs, and postural tremor accompany opsoclonus in a benign encephalitis of early childhood (Cogan, 1968). A variety of posterior fossa lesions can cause opsoclonus. Recently Coxsackie B virus (Kuban et al., 1983) and hemophilus influenza meningitis (Rivner et al., 1982) have been incriminated in the myoclonic encephalopathy of childhood. Remote, nonmetastatic carcinoma of the lung, breast, or uterus should be sought in adults (Ross and Zeeman, 1967) and in children an occult neuroblastoma must be excluded (Solomon and Chutorian, 1968). Thalamic tumor or hemorrhage extending into the midbrain is reported to cause opsoclonus (Keane, 1980; Keane and Devereaux, 1974) but the cases described had horizontal oscillations and without oculographic confirmation it is not certain that the dyskinesias consisted of back-to-back saccades without intersaccadic intervals (Figure 1H) rather than macro square wave jerks (Figure 1E). Intoxication with lithium, thallium, amitriptyline, or chlordecone also causes the dyskinesias. Opsoclonus is occasionally a transient phenomenon in otherwise healthy infants (Hoyt et al., 1980).

Ocular Flutter
Flutter refers to bursts of closely spaced saccades in one plane, during forward fixation (Figure 1F). It may be an isolated oscillation, or a continuum of opsoclonus that is observed as the underlying disease resolves (Ellenberger et al., 1972). Flutter is typically horizontal, but it occurs occasionally in the vertical plane (Toupet et al., 1982). Flutter at the completion of refixational saccades is termed flutter dysmetria (Figure 4). Flutter dysmetria differs from ocular dysmetria which is a "flat topped" oscillation induced by saccadic shifts of gaze (Selhorst et al., 1976). Isolated flutter is often observed in patients with multiple sclerosis and signs of cerebellar and brainstem dysfunction; it may also be the predominant manifestation of neurological disease, as demonstrated by the following case.

A 32 year old man presented with a one week history of generalized severe headache, nausea and vomiting, and three days of blurred vision associated with the illusion that the environment appeared to move rapidly from side to side. Examination revealed horizontal large amplitude ocular flutter (Figure 1F). Quantitative oculography showed normal smooth pursuit. Refixation saccades elicited flutter dysmetria (Figure 4). Apart from mild ataxia of gait, the remainder of the neurological examination was normal. The results of CT scanning, CSF examination, electroencephalography, and brainstem auditory evoked responses were all normal. A search for an occult malignancy by chest x-ray, IVP, upper GI series and barium enema was negative. His intermittent oscillopsia gradually subsided over two months. Examination four months later showed no ocular flutter. Neurological examination was normal.

In this case an acute encephalopathy was associated with oscillopsia caused by ocular flutter. Although a search for a viral etiology was unrewarding, encephalitis, or monophasic demyelination were considered probable. The abrupt onset of symptoms, and the total resolution of the oscillations indicated pathological ocular flutter rather than voluntary flutter.

Voluntary Flutter
This benign movement is evoked at will in some eight per cent of the population (Zahn, 1978). It consists of horizontal salvos of back-to-back saccades having a pendular oculographic waveform (Schultz et al., 1977; Stark et al., 1981). Although the oscillations have been habitually referred to as voluntary nystagnus, they are not truly nystagnus, since smooth (slow) eye movements are absent (Sharpe, 1981). In the absence of
other neurological or ocular motor signs pathological ocular flutter, which has the same saccadic composition, can be mistaken for voluntary flutter. A high familial incidence of this movement implicates either a genetic mechanism or learned behavior (Zahn, 1978). Voluntary flutter is a brainstem stunt that testifies to integrity of reciprocally acting horizontal burst units in the pons. It causes oscillopsia. Sometimes when patients discover that they can initiate this ocular tremor, they become alarmed that they have acquired a disease. Reassurance, oculographic studies to establish integrity of other eye movement systems, absence of other neurological findings, and prolonged observation may be required to definitively exclude pathological ocular flutter.

**Convergence-retraction Pulses**

The paralysis of upward gaze caused by prectectal or periaqueductal midbrain damage is associated with bursts of retraction that are elicited by attempted upward saccades. The eyes often show opposed adducting saccades, in violation of Hering’s law (Ochs et al., 1979). They are not true convergence movements, since they are saccades, not smooth eye movements. Nor is this oscillation nystagmus, since it is initiated by saccadic bursts (Sharpe, 1981). We advocate the designation convergence-retraction pulses, rather than the familiar name nystagmus for this saccadic oscillation. The adducting saccades are asynchronous and they have normal velocities which indicate normal reciprocal inhibition of the lateral rectus during adduction (Ochs et al., 1979). Retraction is caused by co-contracting of vertically and horizontally acting ocular muscles (Gay et al., 1963).

**Ocular Bobbing**

Rhythmic, abrupt, downward jerks of the eyes followed by slow return to the midposition either immediately, or after a delay as long as 10 seconds constitute ocular bobbing. Typical bobbing is associated with paralysis of horizontal eye movements. Pontine hemorrhage or infarction is usually responsible. Atypical bobbing refers to bobbing with intact horizontal eye movements (Susac et al., 1970). Metabolic encephalopathy, obstructive hydrocephalus, or cerebellar hematoma cause atypical bobbing. Reverse bobbing is a saccadic oscillation in which the eyes move rapidly up from the midposition and show delayed slow return (Knobler et al., 1981). Metabolic encephalopathy is usually responsible. These upward and downward saccadic oscillations should be distinguished from ocular dipping (Ropper, 1981), also called inverse bobbing (Knobler et al., 1981). Ocular dipping is not a saccadic oscillation since it is initiated by slow downward eye movements and followed by rapid return to the midposition. Dipping is a sign of anoxic coma; it has no known localizing significance, in contrast to ocular bobbing.

**CONCLUSION**

The recognition of fast eye movements which take the eyes off target has added a new dimension to neurological diagnosis. Further correlation of saccadic intrusions and oscillations with other motor deficits, and with radiological and neuropathological findings, should provide increased diagnostic precision for neurologists who learn to distinguish and interpret this spectrum of ocular dyskinesias.

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