REVIEW ARTICLE

Saccadic Intrusions and Oscillations

James A. Sharpe and William A. Fletcher

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.

RÉSUMÉ: Plusieurs types de mouvements involuntaires saccadés des yeux montrent leurs différences des saccades normales car ils emportent la fovea de sa cible. Cet article présente une classification et une description de ces mouvements anormaux et essaye de signaler les façons de les employer pour l'amélioration de la précision dans le diagnostic neurologique.

Saccades are fast eye movements which occur in resonse 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 such as threading a needle, or sighting a rifle, the frequency of microsaccades decreases to about 70 per minute. Most normal subjects can reduce the rate of their microsaccades voluntarily (Winterson and Collewijn, 1976). When they are asked to suppress them, saccade rate is reduced to about 30 per minute. 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.

SACCADIC INTRUSIONS	SACCADIC OSCILLATIONS
Square wave jerks	Square wave oscillation
Sporadic macro square wave jerks	Macro square wave jerks
Saccadic impersistence of gaze	———
———	Macro saccadic oscillation
	Microsaccadic oscillation
Saccadic pulses	Convergence-retraction pulses*
Double saccadic pulses	Opsoclonus
	Flutter
	Voluntary flutter*
Myoclonic ocular jerks	———
Sporadic bobbing	Bobbing

* 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 1A). 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 conditon (Steele et al., 1964), rather than basal ganglia dysfunction alone. Patients with nonparalytic 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 cerebral 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 1A) 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 bifurction 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 funduscopy 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.
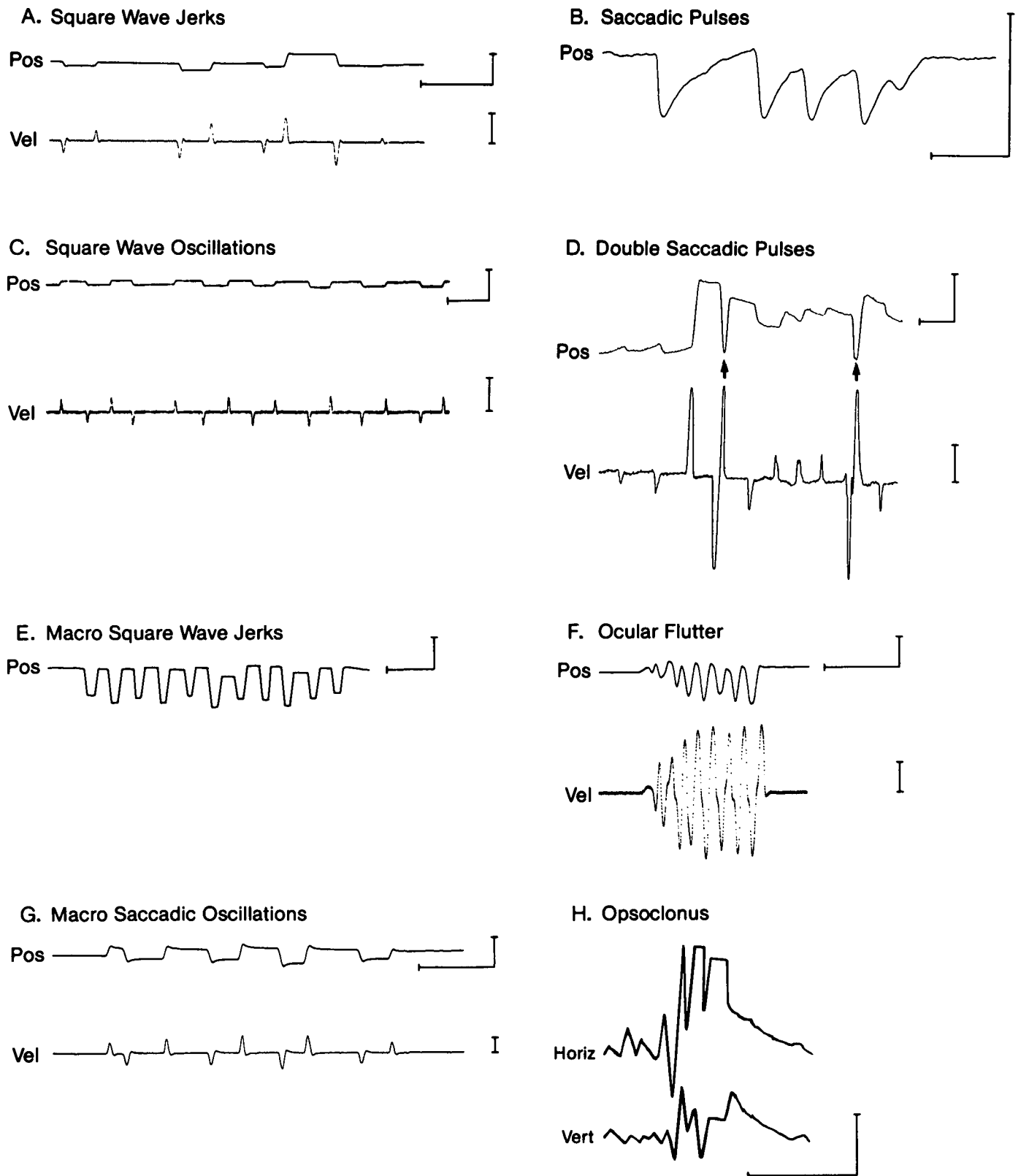
Figure 1 — Examples of saccadic intrusions and oscillations. Horizontal calibrations indicate a time base of 500 msec. Vertical calibration marks signify 10 degrees for the eye position (Pos) traces and 200 deg/sec for the eye velocity traces (Vel). The small arrows in D indicate double saccadic pulses. 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 (Table) 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 oculagraphy using dc 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.
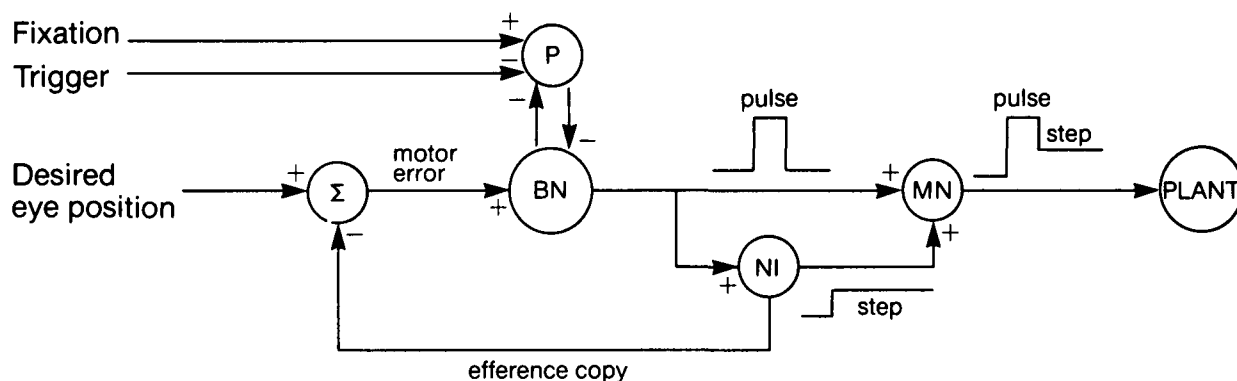
Figure 2 — Model of generation of horizontal saccades. Fixation maintains activity of pause cells (P) until a saccadic trigger signal inhibits them, thereby releasing medium lead burst neurons (BN). The burst neuron sends a velocity command (the pulse) to ocular motor neurons (MN). Mathematical integration of the pulse, by a neuronal network called the neural integrator (NI), creates an eye position command (the step). The pulse and step of innervation are delivered to the ocular motor plant (orbital viscoelastic mechanical properties) as an assembled velocity and position command. The burst cells continue to discharge in response to motor error, the difference between desired eye position and an extraretinal signal of actual eye position (efference copy). When actual eye position is equal to desired eye position, the burst cells cease firing, the pause cells are disinhibited, and the saccade stops (Robinson, 1975). Burst cells on both sides of the paramedian pontine reticular formation are reciprocally inhibitory. In addition, inhibitory burst neurons (IBN) in the medulla (not shown) prevent antagonist motor neurons from firing; IBN are inhibited by pause neurons (Nakao et al., 1980). BN for horizontal saccades are located in the caudal paramedian pontine reticular formation and BN for vertical saccades are in the rostral paramedian midbrain reticular formation.

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

Figure 3 — Saccadic impersistence of gaze in a patient with Alzheimer's disease. Rightward is up, downward left.

expected target motion. These anticipatory saccadic intrusions are involuntary in the sense that patients cannot wilfully 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 instrusive 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 1-C, E and G) and those composed of back-to-back saccades (Figure 1-D, 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 intregrity 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 Square Wave Jerks

This horizontal oscillation occurs as bursts of to and fro saccades to one side of fixation; each saccade is separated by an interval. Macro square wave jerks are not simply enlarged

square wave jerks as they have a shorter off-target intersaccadic interval (50-150 msec), and they oscillate on one side of fixation (Figure 1E). Although tentatively associated with cerebellar outflow damage (Dell'Osso et al., 1975), the brainstem or cerebellar patho-anatomic substrate of macro square wave jerks is uncertain. The following case illustrates this disorder.

A 68 year old woman presented with sudden onset of severe headache followed by dizziness, weakness of her limbs, double vision, nausea, vomiting and drowsiness. She had been hypertensive for 15 years.

On examination the patient was drowsy but fully orientated. Her speech was slurred. Her eyes were deviated to the left and gaze paretic nystagmus was present on rightward gaze. The right arm and leg were ataxic. The remainder of the neurologic examination was normal. A CT scan showed a cerebellar hemorrhage in the deep dorsal vermis, to the right of midline. The hemorrhage was surgically evacuated without complications. She gradually improved, but 8 days after the hemorrhage complained of blurred vision and difficulty focussing. She did not experience oscillopsia. Clinical and infrared oculographic examinations showed bursts of macro square wave jerks to the left of midline, during forward fixation. Occasional salvos of macro saccadic oscillation straddled the fixation position. The episodes of visual blurring, and the corresponding oscillations gradually subsided over the next 3 weeks.

This case demonstrates that macro square wave jerks are directed away from the side of rostral cerebellar damage involving outflow projections of the brachium conjunctivum.

In one patient with diffuse undiagnosed encephalopathy macro square wave jerks were stopped by occlusion of one eye (Dell'Osso et al., 1977). The oscillations were latent during monocular fixation. Since "latent" is the conventional term for an ocular oscillation that is absent during binocular fixation but present only when one eye is covered, this dyskinesia was called "inverse latent" macro square wave jerks. One parameter of saccadic oscillation is the ratio of time off target to the time on target. This ratio is called the duty cycle (Abel et al., 1984). A duty cycle of 0.5 indicates a perfectly symmetrical oscillation. Macro square wave jerks always have a duty cycle of less than 0.5 (Figure 1E).

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 clinico-pathological 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 oposoclonus (Keane, 1980; Keane and Devereaux, 1974) but the cases described had horizontal oscillations and without oculographic confirmation it is not certain that the dyskinesia 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 dyskinesia. Opsoclonus is occasionally a transient phenomenon in otherwise healthy infants (Hoyt et al., 1980).
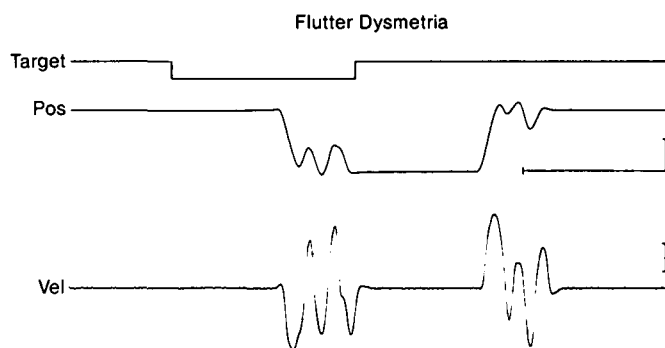

Figure 4 — Flutter dysmetria in a patient with encephalitis. Vertical calibration bars indicate 10 degrees for eye position (Pos) and 200 degrees/sec for velocity (Vel). Horizontal bar indicates 500 msec. In response to target steps of 20 degrees amplitude, the eye oscillates, without interval between saccades, before foveating the new target position.

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 nystagmus, they are not truly nystagmus, 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.

REFERENCES

Abel LA, Traccis S, Dell'Osso LF, Daroff RB, Troost BT (1984) Square wave oscillation: the relationship of saccadic intrusions and oscillations. Neuro-ophthalmology (Amsterdam) 4:21-25

Ciuffreda KJ, Kenyon RV, Stark L (1979) Fixational eye movements in amblyopia and strabismus. J Amer Otom Assoc 50:1251-1258

Cogan DG (1968) Opsoclonus, body tremulousness and benign encephalitis. Arch Ophthalmol 79:545-551

Daroff RB (1977) Ocular oscillations. Ann Otol Rhinol Laryngol 86:102-107

Dell'Osso LF, Abel LA, Daroff RB (1977) Inverse latent macro square wave jerks and macro saccadic oscillations. Ann Neurol 2:57-60

Dell'Osso LF, Troost BT, Daroff RB (1975) Macro square wave jerks. Neurology 25:975-979

Dolsac MJ, Dell'Osso LF, Daroff RB (1983) Multiple double saccadic pulses occurring with other saccadic intrusions and oscillations. Neuro-ophthalmology (Amsterdam) 3:109-116

Ellenberger C, Keltner JL, Stroud MH (1972) Ocular dyskinesia in cerebellar disease: evidence for the similarity of opsoclonus, ocular dysmetria and flutter like oscillations. Brain 95:685-692

Gay A, Brodkey J, Miller JE (1963) Convergence retraction nystagmus. Arch Ophthalmol 70:456-461

Hepp K, Henn V (1983) Spatio-temporal recording of rapid eye movement signals in the monkey paramedian pontine reticular formation. Exp Brain Res 52:105-120

Herishanu YO, Sharpe JA (1981) Normal square wave jerks. Invest Ophthalmol Vis Science 20:269-272

Herishanu YO, Sharpe JA (1983) Saccadic intrusions in internuclear ophthalmoplegia. Ann Neurol 14:67-72

Hotson JR (1982) Cerebellar control of fixation eye movements. Neurology 32:31-36

Hoyt CS, Mousel DK, Weber AA (1980) Transient supranuclear disturbances of gaze in healthy neonates. Amer J Ophthalmol 89:708-713

Keane JR, Devereaux MW (1974) Opsoclonus associated with an intracranial tumor. Arch Ophthalmol 92:443-445

Keane JR (1980) Transient opsoclonus with thalamic hemorrhage. Arch Neurol 37:423-424 .

Knobler RL, Somasundaram M, Schutta HS (1981) Inverse ocular bobbing. Ann Neurol 9:194-197 .

Kowler E, Steinman RM (1980) Small saccades serve no useful purpose (letter). Vision Res 20:273-276

Kuban KC, Ephos MA, Freeman RL, Laffell LB, Bresnan MJ (1983) Syndrome of opsoclonus-myoclonus caused by Coxsackie B3 infection. Ann Neurol 13:69-71

LeGrand Y (1967) Form and space vision. University of Indiana Press.

Leigh RJ, Hewman SA, Folstein SE, Lasker AG, Jensen BA (1983) Abnormal ocular motor control in Huntington's disease. Neurology 33:1268-1275

Nakao S, Curthoys IS, Markham ChH (1980) Direct inhibitory projections of pause neurons to nystagmus-related pontomedullary reticular burst neurons in the cat. Exp Brain Res 40:283-293

Ochs AL, Stark L, Hoyt WF, D'Amicio D (1979) Opposed adducting saccades in convergence-retraction nystagmus. A patient with sylvian acqueduct syndrome. Brain 102:497-508

Orzechowski C (1927) De l'ataxie dysmetrique des yeux: Remarques sur l'ataxie des yeux dite myoclonique (opsoclonie, opsochorie). J Psychol Neurol 35:1-18

Rivner MH, Jay WM, Green JB, Dyken PR (1982) Opsoclonus in hemophilus influenzae meningitis. Neurology 32:661-663

Robinson DA (1975) Oculomotor control signals. In: Basic Mechanisms of Ocular Motility and Their Clinical Implications, G Lennerstrand and P Bach-y-Rita (eds) Toronto, Pergamon Press, pp 337-374

Roper AH (1981) Ocular dipping in anoxic coma. Arch Neurol 38:297-299

Ross AT, Zeman W (1967) Opsoclonus occult carcinoma and chemical pathology in dentate nuclei. Arch Neurol 17:546-551

Selhorst JB, Stark L, Ochs AL, Hoyt WF (1976) Disorders in cerebellar ocular motor control. II. Macrosaccadic oscillation. An Oculographic control system and clinico-anatomical analysis. Brain 99:509-522

Selhorst JB, Ochs AL, Selbst RG (1983) Myoclonic ocular jerks. Ann Neurol 13:212-213

Sharpe JA (1981) Central eye movement disorders. American Academy of Neurology, Neuro-ophthalmology Course, 14:77-91

Sharpe JA, Herishanu YO, White OB (1982) Cerebral square wave jerks. Neurology 32:57-62

Shults WT, Stark L, Hoyt WF, Ochs AL (1977) Normal saccadic structure of voluntary nystagmus. Arch Ophthalmol 95:1399-1404

Solomon GE, Chutorian AE (1968) Opsoclonus and occult neuroblastoma. New Eng J Med 279:475-477

Stark L, Hoyt WF, Ciuffreda K, Kenyon R, Hsu F (1981) Time optimal saccadic trajectory model and voluntary nystagmus. In: Models of Oculomotor Behavior and Control, BL Zuber (ed), CRC Press, Boca Raton, pp 75-89

Steele J, Richardson JC, Olzewski J (1964) Progressive supranuclear palsy. Arch Neurol 10:333-359

Steinman RM, Hadad GM, Skavenski AA, Wyman D (1973) Miniature eye movement. Science 181:810-819

Susac JO, Hoyt WF, Daroff RB, Lawrence W (1970) Clinical spectrum of ocular bobbing. J Neurol Neurosurg Psychiat 33:771-775

Toupet M, deGramont A, Bacri D, Haguenau M, Pepin B (1982) Le flutter-opsoclonie. Rev Neurol (Paris) 3:219-239

Troost BT, Daroff RB (1977) Ocular motor defects in progressive supranuclear palsy. Ann Neurol 2:397-403

van Gisenbergen JAM, Robinson DA, Geilin S (1981) A quantitative analysis of generation of saccadic eye movements by burst neurons. J Neurophysiol 45:417-442

White OB, Saint-Cyr JA, Tomlinson RD, Sharpe JA (1983) Ocular motor defects in Parkinson's disease. II. Control of the saccadic and smooth pursuit systems. Brain 106:571-587

Winterson BJ, Collewijn H (1976) Microsaccades during finely guided visuomotor tasks. Vision Res 16:1387-1390

Zahn JR (1978) Incidence and characteristics of voluntary nystagmus. J Neurol Neurosurg Psychiat 41:617-623

Zee DS, Robinson DA (1979) A hypothetical explanation of saccadic oscillations. Ann Neurol 5:405-414