CHAPTER 9

Nystagmus, Saccadic Intrusions/Oscillations, and Oscillopsia

L. F. Dell'Osso, Ph.D.

Professor, Departments of Neurology and Biomedical Engineering, Case Western Reserve University, Director, Ocular Motor Neurophysiology Laboratory, Veterans Administration Medical Center, Cleveland, Ohio

Understanding the pathophysiology of ocular oscillations necessitates differentiation between those involving only the slow system and those that are purely saccadic. Oscillations containing both saccades and slow phases require identification of both the causative phase (i.e., that which takes the eyes away from their intended direction) and the corrective phase. Modern recording methods have made possible these determinations and thereby clarified the underlying ocular motor mechanisms responsible for many oscillations. Table 1 lists 45 types of nystagmus (two of which were not included previously) along with many other terms found in the literature to describe them; similarly, Table 2 lists 16 saccadic oscillations and intrusions with other descriptive terms. The tables evolved from those that appeared in previous book chapters.[1–4]

The definitions and categorizations used herein result from applying criteria derived from accurate ocular motility recordings. They differentiate between nystagmus and saccadic oscillations, and as a result, I have found that some saccadic oscillations were originally described using the word *nystagmus*. Most oscillations were named before the benefits of accurate recordings. Quotation marks are used

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TABLE 1.
Forty-five Types of Nystagmus*

- Acquired
  - "Fixation"
- Arthrokinetic
  - Induced
  - Somatosensory
- Associated
  - Induced
  - Stransky's
- Audiokinetic
  - Induced
- Bartels'
  - Induced
- Bruns'
- Centripetal
- Cervical
  - Neck torsion
  - Vertebrobasilar artery insufficiency
- Circular/elliptic/oblique
  - Alternating windmill
  - Circumduction
  - Diagonal
  - Elliptic
  - Gyratory
  - Oblique
  - Radiary
- Congenital
  - "Fixation"
  - Hereditary
- Convergence
- Convergence-evoked
- Dissociated
  - Disjunctive
- Downbeat
- Drug-induced
  - Barbiturate
  - Induced
- Epileptic
  - Ictal
- Flash-induced
  - Flicker-induced
  - Induced
- Gaze-evoked
  - Deviational
  - Gaze-paretic
  - "Neurasthenic"
  - "Seducible"
  - "Setting-in"
- Horizontal

(Continued.)

TABLE 1 (cont.).

Forty-five Types of Nystagmus*

- Induced
  - Provoked
- Intermittent vertical
- Jerk
- Latent/manifest latent
  - Monocular "fixation"
  - Unimacular
- Lateral medullary
- Lid
- Miner's†
  - Occupational
- Muscle-paretic
  - Myasthenic
- Optokinetic
  - Induced
  - "Kinetic"
  - "Optic"
  - Optomotor
  - Panoramic
  - "Railway"
  - Sigma
  - "Train"
- Optokinetic after-nystagmus
  - Induced
  - Postoptokinetic
  - Reverse postoptokinetic
- Pendular
- Periodic/aperiodic alternating
  - Alternans
- Physiologic
  - End point
  - Fatigue
- Pursuit after-nystagmus
  - Induced
- Pursuit-defect†
- Rebound
- Reflex
  - Baer's
- See-saw
- Somatosensory
  - Induced
- Spontaneous
- Stepping around
  - Apparent/real
  - Induced
  - Somatosensory
- Torsional
  - Rotary
- Uniocular
- Upbeat

(Continued.)

TABLE 1 (cont.).
Forty-five Types of Nystagmus*

- Vertical
- Vestibular
  - A(po)geotropic/geotropic
  - Alternating current
  - Bechterew's
  - Caloric/caloric-after
  - Compensatory
  - Electrical/faradic/galvanic
  - Head-shaking
  - Induced
  - L-
  - Labyrinthine
  - Perverted
  - Pneumatic/compression
  - Positional/alcohol
  - Positioning
  - Postrotational
  - Pseudocaloric
  - Rotational/perrotary
  - Secondary phase

*Synonyms and other terms are indented under either the preferred or the more inclusive designation; some nystagmus types may be acquired or congenital; quoted terms are erroneous or nonspecific.
†May not exist.

for those oscillations that are *not* truly nystagmus or for subjective clinical terms found in the literature that are inadequate, are not clearly defined, or have been misapplied. Since these ambiguous or erroneous terms do not convey accurate information about the basic nature of the movement, they are better left at the bedside. In those cases where historical precedence supports their continued usage (e.g., abduction "nystagmus" and convergence-retraction "nystagmus"), the quotation marks show that these are saccadic oscillations. The best names reflect the mechanism thought to be responsible for the eye movement (e.g., saccadic pulse); such terms maximize the information carried to the reader by describing a movement by its well-defined component parts.

NYSTAGMUS

Nystagmus is defined as a biphasic ocular oscillation containing slow eye movements that are responsible for its genesis and continuation. Fast eye movements (saccades), if they are present, serve a corrective function and do not represent the basic ocular motor dysfunction. The two phases of ocular nystagmus are about equal in amplitude. What follows, in alphabetical order, is a discussion of each

TABLE 2.

Sixteen Saccadic Intrusions and Oscillations*

- Bobbing/dipping
  - Inverse bobbing
  - Reverse bobbing
- Convergence-retraction "nystagmus"
  - "Nystagmus" retractoris
- Double saccadic pulses (single/multiple)
  - Saccadic intrusions/oscillations
- Dynamic overshoot
  - "Quiver"
- Dysmetria
- Flutter
- Flutter dysmetria
- Macro–saccadic oscillations
- Macro–square-wave jerks (bursts/single)
  - Kippdeviationen/"Kippnystagmus"
  - "Pendular macro-oscillations"
  - Saccadic "nystagmus"
  - Saccadic oscillations/intrusions
- Myoclonus
  - Laryngeal "nystagmus"
  - "Lightning eye movements"
  - Pharyngeal "nystagmus"
- Opsoclonus
  - "Dancing eyes"
  - "Lightning eye movements"
  - Saccadomania
- Psychogenic flutter
  - Hysterical flutter
  - Hysterical "nystagmus"
  - "Ocular fibrillation"
  - "Ocular shuddering"
  - Psychological "nystagmus"
  - Voluntary flutter
  - Voluntary "nystagmus"
- Saccadic lateropulsion
  - Ipsipulsion
  - Contrapulsion
- Saccadic pulses/pulse trains
  - Abduction "nystagmus"
  - Ataxic "nystagmus"
  - Saccadic intrusions/oscillations
  - Stepless saccades
- Square-wave jerks/oscillations
  - Gegenrucke
  - Hopping "nystagmus"
  - "Lightning eye movements"
  - Myoclonus
  - Saccadic intrusions/oscillations
  - Zickzakbewegungen
- Superior oblique myokymia

*Synonyms and other terms are indented under either the preferred or the more inclusive designation; quoted terms are erroneous or nonspecific.

nystagmus type in which significant work has been done in the past 2 years. Discussions of all types of nystagmus may be found in the aforementioned previous publications.[1-4]

Associated Nystagmus

Associated (Stransky's) nystagmus is a high-frequency, induced nystagmus caused by the attempted forceful opening of one eye while the subject continues to keep the eyes closed tightly.[5]

Cervical Nystagmus

Cervical nystagmus is allegedly caused by cervical spondylosis, whiplash, cervical muscle impairment, and cervical cord disease. A recent critical review of the literature has concluded that the entire concept of cervical nystagmus is highly suspect.[6] In our laboratory, we are not convinced that we have ever seen a patient with cervical nystagmus.

Congenital Nystagmus

An excellent article contains illustrations of most of the known characteristics of congenital nystagmus (CN) as well as some new findings.[7] In it are discussions of both the effort to see and state of arousal on the intensity of CN; the presence of CN during various stages of sleep; aperiodic alternating nystagmus; bias reversals during steady fixation; and both accurate and inaccurate foveation during fixation. A dramatic change in waveform was shown for both an adult and an infant when going from fixation to a state of low arousal. During fixation the waveforms were jerk or pseudocycloid with amplitudes up to 4 degrees and frequencies of 2 to 4 Hz, whereas during the low state of arousal the waveform became pendular, the amplitudes rose to 15 degrees to 60 degrees, and the frequency dropped to about 1 Hz. The authors emphasized that although we normally refer to CN as " congenital," it may not be constantly present until many weeks after birth.

In an attempt to determine whether the pendular part of dual jerk waveforms were truly sinusoidal, Reccia et al.[8] used spectral analysis. Unfortunately, the data were obtained using low-bandwidth, bitemporal electro-oculography (EOG). By subtracting the power spectrum of a pure sawtooth waveform from the dual jerk waveform, the authors were able to emphasize the pendular component. Their statistical analysis concluded that a sinusoidal fit would better approximate the

data than an exponential fit; the latter was suggested by the model of Optican and Zee.[9]

One problem facing the clinician is the proper diagnosis of nystagmus occurring at birth or early infancy. Distinction must be made among CN, latent/manifest latent nystagmus (LMLN), spasmus nutans, or an acquired nystagmus indicating neurologic pathology. A recent article discussed some of the many pitfalls in distinguishing CN from LMLN and in identifying the nystagmus blockage syndrome in those patients with CN who use an esotropia to damp their CN or convert it to manifest latent nystagmus (MLN).[10] These authors pointed out the problems inherent in many published studies where these distinctions have not been made and where the patient population has included some with each of these conditions. Although both CN and LMLN are congenital types of nystagmus, to avoid confusion, I have deliberately refrained from using the word *congenital* when referring to LMLN. In the aforementioned article, the term *manifest* congenital nystagmus is CN and the term *latent and manifest latent congenital nystagmus* is LMLN. Despite the difference in terminology, this article clearly discusses some of the diagnostic problems. Though a causal relationship between esotropia and LMLN has not been proved, it is my opinion that since all patients with LMLN have strabismus, there is a strong suggestion of a causal (necessary but not sufficient) link.

An attempt has been made to use electromyographic (EMG) recordings to examine the possibility that there are two types of gaze-angle nulls present in CN.[11] One type is attributed to the active blockage of the nystagmus by an increase in the discharge of the synergistic extraocular muscles responsible for the gaze angle adopted. The second type is the classic null position, for which no good explanation has been proved, and is thought to result from an equilibrium position between the forces present in the push-pull ocular motor system. According to this article, the blockage type of null occurs at angles greater than 10 degrees from primary position and the so-called Kestenbaum null occurs closer to primary position. The key to differentiation, according to these authors, is analysis of time histograms of the EMG signal, since ocular motor activity reflected in ENG is the same for both types of nulls. Before discussing this article further, some definitions are in order. For a gaze angle position of low nystagmus to be a true null, the nystagmus must be higher at gaze angles to either side of it. Those patients who turn their heads in one direction so their eyes are in *extreme* lateral deviation do not fulfill this definition. It is well known that many patients with LMLN put the fixating eye in extreme adduction and take advantage of Alexander's law to minimize their nystagmus; this is not a true null angle. Similarly, some patients with CN also adopt an extreme head turn, forcing their eyes laterally and minimizing their nystagmus; this too is not a true null since the eyes cannot be deviated further to test whether the nystagmus increases at those gaze angles. Thus, patient 1 of the six studied in this article could not be said to have a true CN null. This is further evidenced by the observation that he adopted both a right and a left eye turn, during which his nystagmus was minimal; patients with CN with true nulls have only one such position. The other five patients appeared to have true nulls.

The question then remains: Are the differences reflected in EMG histograms indicative of different mechanisms? In the illustrations shown for each type of null, the histograms are peaked in the off-null positions and flatter where the nystagmus is minimal. This is to be expected, even if the nystagmus did not decrease; the increased activity caused by raising the firing level as the eye is deviated into its on direction would tend to broaden the histogram and lower the peak that is a measure of the relative number of intervals with given durations. Thus, even the EMG histograms are qualitatively the same. The authors claim that the difference is because for the Kestenbaum null the flatter curve is "five times faster" than for the other type; the meaning of that statement is obscure. Unfortunately, the two figures that must be compared are on different time scales both for the curve shown in primary position and for that shown at the null position. It is difficult to make comparisons between these two figures, and I was unable to find any measured points to which I could attribute a fivefold difference.

We have measured the CN of patients who have true nulls at most gaze angles as well as some whose nulls were so far in lateral gaze that we could not go further to prove that it was a true null. It is too simplistic to pick a number like 10 degrees and say that true nulls occur at smaller angles and that, beyond 10 degrees, only the so-called blockage null occurs. All nulls will exhibit the change in EMG histograms described above, since increased activity will result in a broader, flatter curve. The widths of the peaks shown in both primary positions and at the null for the two patients illustrated in this article bear the same relationship; the widths of both peaks for patient 5 are greater than those for patient 1. If these authors have uncovered a difference in the types of CN nulls present in the patient population, they have not proved it conclusively in this article. Furthermore, if the mere increase in activity of a muscle can be used to block the nystagmus, then all such patients should be able to do this in both directions. This is not seen with patients with binocular CN; it is with patients with strabismic LMLN. Also, this increase in synergistic activity is not equivalent to the blockage of CN with convergence where the increase in activity is in antagonistic muscles and affects the push-pull system in a different way.

An interesting Japanese pedigree of hereditary CN in five generations was attributed to X-linked irregular dominant transmission.[12] Absence of male-male transmission and generation skipping was noted. The CN waveforms were predominantly pendular, and there was good central vision and an absence of sensory defects in this pedigree. These patients are further examples that waveform cannot be used to classify CN as "sensory-defect" nystagmus. In another article, five male infants who showed findings of abnormal auditory brain-stem response and pendular CN were reported.[13] They also had hypotonia of head and limbs in the early infantile period and later paresis. Unfortunately, the method for recording the eye movements was poor, and it was impossible to identify the CN waveforms from the figures provided. If the time constant given was correct, the recordings more accurately reflect eye velocity than position. Given their other abnormalities, it is possible that these patients had both CN and other neurologic disorders as

suggested by the authors. An animal model for CN may have been produced in monkeys by monocularly depriving them of vision at birth, then reversing their sutures 25 days later.[14] A variable nystagmus was observed that could be jerk, pendular, or combinations of both; the slow phases of the jerk nystagmus were of increasing velocity. Also, a latent component was noted on cover testing. At present, there is no known animal model for CN, and if this procedure is repeatable, it may provide one.

In our early studies of CN, we noted that responses to changes in target position were often combinations of hypometric saccades and the slow phases of the CN waveform.[15] This observation led us to hypothesize that CN was caused by an abnormality in the saccadic system; we have long since realized that it is in the slow eye movement system. An excellent article contains the responses to step changes in target position exhibited by subjects with CN.[16] It was found that the hypometric saccades or slow phase responses occurred most frequently when the target was displaced in the direction of the slow phase. Since the retinal stimulus produced by step change in a target position is the same stimulus that normal subjects receive from a step change followed by a ramp motion back toward the fovea, comparison was made with responses of normal subjects to the standard Rashbass stimulus. The authors concluded that the ocular motor responses of patients with CN were *normal saccadic responses* to the sequence of retinal image motion produced by this stimulus and not contributions from the pursuit system in response to position displacements on the retina, as has been suggested by others. The responses of patients with CN with albinism were less likely to contain saccades, and this was attributed to their impaired sensory functioning. Thus, there is now additional evidence supporting the hypothesis that both the saccadic and pursuit systems of subjects with CN *function normally* given the ongoing oscillation; this hypothesis was contained in the earliest model of the saccadic and pursuit systems operating in the presence of an ongoing oscillation.[17] In another article, albinism has been linked with periodic alternating CN.[18]

Two interesting cases of CN that became manifest only during unidirectional pursuit were reported.[19] It is well documented that the attempt to pursue a slowly moving target significantly alters CN. Specifically, the static null is shifted in the direction opposite to the pursuit. Thus, the actual CN amplitude at a given gaze angle is greater during pursuit than when viewing a stationary target since that gaze angle is farther from the null during pursuit than during steady fixation. It is common to record a high-amplitude CN waveform during pursuit of rapidly moving targets across the whole field of gaze, from 20 degrees left to 20 degrees right, despite the fact that the same subject might have a broad null region somewhere in this range when looking at stationary targets. In the two subjects presented in this article, no CN was present during fixation of stationary targets at any gaze angle. However, when pursuing targets moving in one direction, a nystagmus developed that had increasing-velocity exponential slow phases and was diagnosed as being CN. These patients' complaints of oscillopsia (OSOP) only during such pursuit have been with them throughout their lives. It is interesting that the normal sup-

pression of OSOP seen in subjects with CN was not present in these cases where the nystagmus was only manifest during unidirectional pursuit. Given the effects of smooth pursuit on the CN null, the most parsimonious explanation for these observations is that these patients had a very broad null region that encompassed their whole range of gaze angles when viewing static targets. When they attempted to pursue in one direction, that null region was shifted in the opposite direction, causing a nystagmus in the same direction as the pursuit. These would be two extreme examples of the well-known null shift seen in most subjects with CN. Kawai[20] studied the effects of an optokinetic background on CN. He concluded (agreeing with my own observations) that the perceived circular vection is in the proper direction for the background movement and, furthermore, that the optokinetic nystagmus (OKN) dynamics were normal in subjects with CN.

In many subjects with CN, the nystagmus damps and acuity increases with convergence. The variety of clinical conditions under which this occurs suggests that this effect is determined by the convergence angle rather than the state of accommodation. A recent study confirmed this hypothesis and further found that binocular viewing was unnecessary for the damping of CN.[21] Also confirmed were the observations that asymmetric convergence damps CN (this is the basis for the use of composite prisms), and that, once converged, gaze angle plays no important role in CN amplitude (if it did, convergence prisms or bimedial recession operations would not be of much value since real-life situations require looking in all fields of gaze). By carefully controlling each variable, the author clearly demonstrated the importance of convergence.

In the final part of the article, the author attempted to evaluate the effects on visual acuity of prism therapy but neglected to adjust the patients' refractions while in the converged position. She correctly stated that base-out prisms have an identical effect on CN intensity at distance as at near fixation. However, both conditions cause accommodation that is beneficial at near but detrimental at distance. Therefore, the correct ("traditional") method of applying base-out prisms to increase acuity requires the addition of -1.00 diopters to the patient's distance refraction to negate the induced accommodation; this results in the clearest possible vision at distance. By merely adding prisms to the patient's normal distance refraction, the author was effectively "over-plussing" them at distance and requiring them constantly to fight their natural accommodated state induced by convergence; she did *not* duplicate the traditional therapy. This cannot result in optimal acuity and would be uncomfortable in the long term. Reducing CN increases acuity only when the afferent apparatus is functioning correctly. Either afferent defects (like foveal aplasia) or poor refraction will prevent maximal acuity even if the eyes were forcibly held still. Less than optimal refraction reduces acuity in all patients, so it is difficult to understand why an improvement in acuity was expected from patients with CN. The author's use of prisms without minus lenses produced two oppositely directed effects: CN was damped and vision was blurred. The net effect was no increase in acuity with prisms. When acuity is not compromised, the reduction of CN intensity *is* a sufficient condition for an increase in acuity. It is my

opinion that both the final conclusion and Figure 10 are invalid and should be ignored by the reader. Unfortunately, this well-done study of the variables involved is marred by the misapplication of its results. Also, the use of Fresnel prisms is *not* the method of choice in these cases. They degrade vision, and, more important, the many vertical lines are very distracting to one whose eyes are constantly oscillating across them; at night any side light causes inordinate glare from the vertical lines. Prisms should be ground from optical-quality plastic or glass.

Von Noorden and Wong[22] recently reported the surgical results in the nystagmus blockage syndrome (NBS). The results of unilateral recession-resection and bimedial recession with or without posterior fixation sutures were comparable and poor. Both over- and under-corrections occurred more frequently, and the number of operations was higher than in a controlled group of essential infantile esotropia without nystagmus. Normal binocular vision was not achieved in a single case. It would appear from these results that NBS is not conducive to simple surgical correction. A spectral analysis of the EOG was used to evaluate CN surgery quantitatively.[23] Unfortunately, the use of AC-coupled, low-pass–filtered, bitemporal EOG severely limits the usefulness of the data gathered. Performing high-powered analysis on such poorly gathered data does not significantly increase the authority of the paper's conclusions. I agree with the authors that spectral analysis can be a useful tool in the quantitative study of CN and its surgery, but it should be applied to accurately measured data.

In response to occasional anecdotal reports of CN damping when contact lenses are inserted, we studied the effects of soft contact lenses on CN amplitude and waveform and on visual acuity.[24] We found that the presence of a contact lens can damp CN, owing to sensory feedback from the inner eyelid, and improve visual acuity. The use of a topical anesthetic abolished the effect; pressure on the eyelid (with no anesthetic) also reduced the CN. This is a new, benign, and potentially useful therapy that expands the population of patients with CN who can be helped to those without gaze-angle or convergence nulls while including those with nulls. Even in those without need for refractive correction, plano contacts should damp their CN and improve their acuity.

In addition to surgery, optical methods, and biofeedback, another treatment for CN has been introduced: acupuncture.[25] Four single needles with a diameter of 0.02 mm and length of 40 mm were inserted about 10 mm through the skin in the distal and proximal points of both sternocleidomastoid muscles. This is not painful (personal observation). The needle tips are stimulated by an electrical current carrying a square wave or by manually beating the needle every 5 minutes, and the duration of the insertion is 20 minutes. The program is repeated two times per month. Acupuncture was effective in eight of 13 cases; there were four young children with poor cooperation and one with cerebral palsy. After treatment, the effects persisted for 4 to 7 days with a reduction in CN amplitude of 50% and frequency of 15%. Acupuncture was also tried in two cases of acquired nystagmus and resulted in a reduced sensation of OSOP.

Jan et al.[26] described a test that is supposed to determine the amblyopic eye in

infants with CN and associated sensory loss. The confusing and inaccurate term "sensory nystagmus" should be ignored by the reader; all CN is caused by a motor instability. The authors claim that when fixating with the poorer eye, the nystagmus will be "wider" (higher amplitude?) and slower and, when fixating with the better eye, faster and of smaller amplitude. They cautioned against applying this test in the presence of latent nystagmus (LN) or MLN but neglected CN with a latent component. Without recordings, none of these can be reliably differentiated. Applying this test to unknown types of nystagmus and attempting to evaluate the results without any objective criteria tells us nothing about the relationship (if any) between acuity of the fixating eye and measurable characteristics of CN. My experience has been that no such relationship exists for all patients, although a properly done study may show that many patients conform to the clinical impression of these authors. They have described a way to give parents a "quick answer"; I wonder if we can give them an accurate answer.

Convergence Nystagmus

An uncommon movement disorder (oculomasticatory myorhythmia) was identified in Whipple's disease.[27] It consists of convergence nystagmus and concurrent contractions of the masticatory muscles. The convergence nystagmus had dynamics characteristic of normal vergence movements and there was no palatal myoclonus. The authors concluded that patients with this sign should be treated presumptively for Whipple's disease.

Convergence-Evoked Nystagmus

An interesting case of convergence-evoked pendular nystagmus was reported.[28] Accommodative vergence was identified as the cause of this nystagmus, which only appeared when attempting to fixate a target moving toward the subject and was unaffected by covering one eye. The absence of neurologic or ophthalmologic abnormalities led the authors to classify this as a congenital type of nystagmus. Flutter, including voluntary flutter, was ruled out by the authors.

Dissociated Nystagmus

A common type of dissociated nystagmus (DISN) is that associated with spasmus nutans. Weissman et al.[29] identified the waveform as a pendular DISN whose key characteristic was the variable phase-shift between the oscillations of the two

eyes. This study makes it possible to distinguish spasmus nutans from CN without the obligatory wait for the nystagmus to disappear.

An interesting form of DISN has been described in the context of coma.[30] It consisted of a slow divergence from primary position followed by a rapid return to primary position; the entire cycle could last from 4 to 10 seconds and was repeated irregularly every 1 to 15 seconds. The authors termed this "repetitive divergence." Since this was a repetitive oscillation, albeit a very slow one, it can be considered a rare form of DISN.

Downbeat Nystagmus

Gresty et al.[31] investigated otolithic and semicircular canal influences on downbeat nystagmus (DBTN). They concluded that the DBTN in their patient arose from an asymmetry of vertical canal function that became manifest when the otoliths were tilted with respect to gravity. Since, in a second patient, the DBTN was insensitive to tilt, they further concluded that various cases of DBTN lie on a continuum between these extreme examples. Although DBTN is usually associated with Arnold-Chiari malformations, two patients were reported with DBTN and lesions remote from the craniocervical junction.[32] One patient had decompensated aqueductal stenosis and the other had a midbrain infarction. Downbeat nystagmus has also been reported in a patient with vitamin B_{12} deficiency, which presumably resulted in cerebellar or brain-stem lesions,[33] and as a presenting sign in von Hippel-Lindau disease.[34]

A case of DBTN that was diagnosed with magnetic resonance imaging (MRI) and disappeared immediately after surgical treatment was recently described.[35] The responsible lesion was a syringomyelic cyst in the medulla. After the cyst was incised and emptied of fluid, both the nystagmus and the accompanying OSOP disappeared immediately.

Drug-Induced Nystagmus

Diazepam was used to maximally disrupt pursuit performance as part of a study of the effects of training and disruption on smooth pursuit.[36] The authors uncovered inherent asymmetries in vertical smooth eye movements that contribute to drug-induced vertical nystagmus. Downbeat nystagmus was reported with lithium intoxication[37] and secondary to excessive alcohol intake[38]; the latter was reversible after a period of abstinence. More commonly, alcohol produces horizontal gaze-evoked nystagmus, and this has led to a "roadside sobriety" test conducted by law enforcement officers.[39] Using nystagmus as an indicator of alcohol intoxication is an unfortunate choice, since many normal individuals have physiologic

end-point nystagmus; small doses of tranquilizers that would not interfere with driving can produce nystagmus; nystagmus may be congenital or consequent to neurologic disease; and without a neuro-ophthalmologist or someone knowledgeable about sophisticated methods of eye movement recordings, it is difficult to determine whether the nystagmus is pathologic. It is unreasonable that such difficult judgments have been placed in the hands of minimally trained law officers.

Epileptic Nystagmus

Lavin[40] reported a case of pupillary oscillations synchronized with epileptic nystagmus. The nystagmus beat with a fast phase that was contralateral to the focus of the abnormality, and the pupils dilated as the eyes moved laterally and constricted when they returned toward the central position. This "clonic" phase lasted about 30 seconds, and the pupils remained symmetric during and after the seizures.

Induced Nystagmus

Electrical stimulation of the nucleus of the optic tract (NOT) resulted in horizontal nystagmus with ipsilateral slow phases followed by after-nystagmus in the same direction.[41] The time course of the slow-phase velocity was similar to that of OKN and optokinetic after-nystagmus (OKAN). Both the rising and falling time constants of the nystagmus and after-nystagmus were sensitive to the frequency and duration of the electrical stimulation. The authors concluded that the NOT stimulation elicited the component of OKN that is responsible for the slow rise in slow-phase velocity and for OKAN. Functionally, NOT was seen as a major source of visual information related to retinal slip. The indirect pathway that excites the velocity storage mechanism in the vestibular system to produce the slow component of OKN and OKAN probably lies in NOT in the monkey as it does for other animals.

Latent/Manifest Latent Nystagmus

Von Noorden et al.[42] recently reported on the successful application of conventional occlusion therapy in patients with LN and strabismic amblyopia. The diagnosis of LN or MLN was made on clinical grounds, and it is therefore possible that some of the patients diagnosed with LN may have had a low-amplitude MLN. It is also possible that some of the patients had CN with a latent component; without accurate eye movement recordings, the waveforms of the nystagmus cannot be

determined. I agree with the authors that it is clinically irrelevant to distinguish between LN, MLN, and CN for the purposes of amblyopia treatment, but it is extremely important to make this distinction when considering surgical or other therapy of the nystagmus itself. While it was previously thought that strabismus is almost always present in patients with LMLN, better recording techniques have led us to hypothesize that strabismus is a necessary condition for LMLN and is therefore present in 100% of the cases.[43]

Moidell et al.[44] studied the effect of early enucleation on the position of the egocenter. The egocenter, that is, the origin of judgments of visual direction, is normally located on the median plane of the head midway between the two eyes. The authors found a significant shift of the egocenter toward the remaining eye of the enucleates that was unrelated to the age at enucleation and concluded that egocenter location is plastic and can be modified by the complete monocularity imposed by enucleation. This documentation of egocenter shift is supportive evidence for the hypothesis that it is the inability to reconcile the difference between monocular egocentric direction and binocular egocentric direction that is the primary cause for LMLN.[45] We reported on a case of unidirectional LMLN in a patient with a congenitally blind eye that was replaced by a prosthesis.[46] We found that his unidirectional MLN spontaneously changed direction in the dark and beat toward his blind eye rather than the eye he always used for vision. We concluded that eye dominance was genetically predetermined and not influenced by visual development, this despite the shifting of his egocenter toward his good eye. In another study of children with congenital or early loss of vision in one eye, visual evoked responses (VER) were evaluated.[47] Half of these patients developed a jerky nystagmus whose clinical description strongly suggests MLN. The authors concluded that VER asymmetries correlated with ocular motor disturbances.

Lid Nystagmus

Howard described a case of convergence-evoked lid nystagmus in a patient with a large angiomatous lesion distorting the blood supply to the pontomesencephalic and pontomedullary junctions.[48] In addition, bilateral gaze-evoked and upbeat nystagmus were sometimes present; they were considered to be independent of the lid nystagmus that beat in the upward direction. Owing to the extent of the lesion, precise localization of the site causing dissociation between the lid and ocular nystagmus was not possible.

Optokinetic Nystagmus

Subjects with normal stereopsis will exhibit OKN when viewing moving stereoscopic contours in a dynamic random-dot stereogram. The use of this phenomenon

as an objective test of stereopsis requires that stereoblind subjects not develop OKN. Archer et al.[49] recently showed this to be the case. None of their stereoblind subjects had an optomotor response to stereoscopic contours regardless of the alignment angle at which the image pair was presented.

Zee et al.[50] reported the effects of occipital lobectomy on eye movements in rhesus monkeys. They found that OKN was markedly altered but not abolished in all animals. The velocity-storage component of OKN was present, but the immediate-pursuit component was eliminated. The maximum value of OKN that could be achieved was decreased to 10% of the preoperative values. The authors noted that the residual OKN was similar to that of afoveate animals, with a diminished response to high velocities of retinal-image motion and a predominance for temporal-to-nasal motion during monocular viewing. Immediately after occipital lobectomy, the necessity for the occipital cortex in normal primates was evident; it makes the predominant contribution toward the generation of the velocity-storage component of OKN. The long-term changes suggested that monkeys could learn to use extrastriate pathways to generate more volitional types of visual-ocular motor behavior. However, some of the more reflexive mechanisms for stabilizing images on the retina eventually deteriorate, presumably owing to the loss of visual feedback. Finally, the authors noted that as some of the ocular motor functions recovered, others deteriorated. Specifically, spontaneous and gaze-evoked nystagmus developed 3 to 6 months after surgery, and the time constant of the neural integrator dropped to values as low as 2.6 to 4.8 seconds.

In man it is difficult to distinguish true OKN from "pseudo-OKN" that is probably mediated by the pursuit system. Since pursuit is predominantly a foveal reflex, it is interesting to observe OKN in patients with central visual field defects. Crevits and Van Vliet[51] reported on six such patients. They found, in some cases, an inverted OKN. A prerequisite for eliciting this was directing the attention of the subject to the field defect; if attention was directed peripherally, the OKN was normal. In general, the reversed OKN was sporadic and occurred only at stimulus velocities of 120 degrees per second.

Verhagen et al.[52] examined the ocular motor responses of a patient who survived the locked-in syndrome. The patient had a pontine infarction mainly on the right side. There was a lack of cortical OKN and preservation of subcortical OKN in this patient. In contrast to previous studies of OKN in patients with the locked-in syndrome, a full-field stimulus was used rather than the small tape normally employed at the bedside; this may be the reason that OKN has been found to be absent in many of these cases. In addition to OKN, the vestibulo-ocular response (VOR) was measured. Postrotary nystagmus responses revealed vestibular hyperreactivity and short time constants with internuclear ophthalmoplegia (INO) on both sides. Since survival of such patients is rare, the recordings made yielded some insight into the ocular motor subsystems involved.

Yee et al.[53] studied both OKN and the VOR in patients with schizophrenia. They found that mean gains in OKN and pursuit of schizophrenic patients were significantly lower than those of normal subjects. Also, suppression of the VOR

by fixation was impaired in this patient population. The authors noted that the groups of patients they studied were taking psychotropic drugs at the time of the study, and these medications could be responsible for the impairment of eye movements found. Though they found some impairment in schizophrenics, they concluded that the magnitude of the impairment was less than suggested in previous studies and that specific eye movement abnormalities might not be a reliable biologic marker for schizophrenia.

Optokinetic After-Nystagmus

Lafortune et al.[54] investigated a dependence of human OKAN, velocity storage, and OKN characteristics on the optokinetic stimulus exposure time. Their results supported the hypothesis of two velocity storage integrators, one responsible for the short time constant decay (pursuit mediated) and the other for the long time constant decay (optokinetic system mediated). They demonstrated a dependence of the long time constant integrator of OKAN on the stimulus exposure time, but the short time constant integrator appeared to be independent of exposure time. They concluded that the charging time course of each component was independent of the other. The decay time constants of each component were found to be invariant. Their data suggested that the integrators were direction sensitive. In a related study, Jell et al.[55] studied the after-effects subsequent to pursuit of a single light-emitting diode (LED) target. Subjects pursued a target moving at 20 degrees per second from 30 degrees left to center gaze and repeated the pursuit after a 3-second period of darkness. The decay of pursuit began about 130 msec after the LED went out, and the authors used this value to obtain all pursuit decay curves. The measured decay time constant was exactly what would have been predicted because of the mechanical and viscoelastic properties of the eyeball in the orbit. The authors concluded that a neural integrator probably does not exist in the system, but if it did, its decay time constant would have to be 0.3 seconds or less.

Himi et al.[56] studied the asymmetry of vertical OKAN in squirrel monkeys. Optokinetic after-nystagmus commonly has two components: OKAN-I is in the same direction as the OKN and OKAN-II is in the opposite direction. These authors found that the slow-phase eye velocity of upward OKAN-I increased with increasing stimulus velocity, whereas that of downward OKAN-I diminished. The time constant of OKAN-I was shortened with an increase in stimulus speed in both directions. With a downward stimulus, a short stimulus duration failed to elicit OKAN-II. With a longer stimulus duration, the appearance of OKAN-II increased in percentage. Their data showed that the asymmetry of OKAN-I and OKAN-II differed.

Optokinetic nystagmus is attenuated by fixation of a stationary target, and illusory induced motion (IM) of that target occurs opposite to the direction of the stimulus motion. It has been proposed that the IM is caused by a perceptually reg-

istered efferent signal for pursuit that opposes an unregistered signal for OKN. If this were true, parallel changes in the magnitudes of IM and optokinetic reflexes during and after optokinetic stimulation should occur. Heckmann and Post[57] found that leftward IM magnitude and rightward slow-phase velocity of OKAN increased at similar rates across 90 and 160 seconds of 60 degrees per second motion of background contours and decayed at similar rates after stimulus termination. The close dynamic correlation of OKAN slow-phase velocity with IM of opposite direction parallels previous work studying the effects of stimulus direction, luminance, and velocity. The authors concluded that a voluntary efferent signal, which sums negatively with a signal in the optokinetic system to achieve stable fixation, produces IM of the fixated target. Their hypothesis also applies to the oculogyral illusion, autokinesis, and apparent motion of a fixated target during head movement. This study also contains a general discussion of several other related hypotheses.

Both OKN and OKAN in the vertical direction were studied using three head positions (upright, lateral, and head-hanging).[58] Normal subjects were subjected to full-field OKN stimulation; OKN asymmetry (downbeating greater than upbeating) was noted in the upright position at various stripe velocities. In three subjects this asymmetry was eliminated in the head-hanging position but only slightly reduced in the lateral position; in one subject the asymmetry was reversed in the head-hanging position. Downbeating OKAN was found in half of the subjects in the upright position but was not affected by position change; upbeating OKAN never occurred.

Pendular Nystagmus

Dehaene et al.[59] described a case of acquired pendular nystagmus (PNDN) in a patient with multiple sclerosis. The nystagmus was vertical and uniocular; after eye closure it became binocular. Although the authors stated that PNDN was present only in primary position, during upgaze the PNDN can clearly be seen superimposed on an upbeat jerk nystagmus in their Figure 1, part 3. During exacerbation of the multiple sclerosis, the PNDN disappeared and a bilateral INO and rebound nystagmus were observed. With the disappearance of the INO and rebound nystagmus, the PNDN reappeared. When the PNDN was present, the patient complained of OSOP. Cordonnier et al.[60] described a case of reversible acquired PNDN occurring after brain-stem hemorrhage. Their patient also had bilateral horizontal gaze palsy and skew deviation on downgaze; a pontine lesion was detected. These two cases illustrate the difficulty one encounters in trying to localize the disturbance that might be responsible for acquired PNDN. Pendular oscillations result from instabilities in closed-loop control systems, and therefore, pathology at any of several sites that interrupt or alter a neurologic closed loop can be the cause of the instability.

Periodic/Aperiodic Alternating Nystagmus

Nuti et al.[61] described two cases of aperiodic alternating nystagmus (APAN), one caused by vertebrobasilar insufficiency and the second by a whiplash injury. In addition to APAN, gaze-evoked nystagmus, rebound nystagmus, saccadic dysmetria, square-wave jerks (SWJ), vestibular nystagmus, and OSOP were exhibited at various times by these patients. The first patient was treated with baclofen with satisfactory results. In CN, APAN is common, but the acquired varieties are often much more periodic. The authors pointed out the connection between rebound nystagmus and periodic alternating nystagmus (PAN), with the former due to less severe pathology than the latter. Cohen et al.[62] studied the effects of baclofen on the VOR and velocity storage in rhesus monkeys. They found a reduction in the VOR time constant, reduced off-axis rotary nystagmus slow phases, reduced OKN gain, and reduced peak velocity and falling time constant of OKAN. The effects of baclofen were simulated on a model by reducing the falling time constant of the velocity-storage integrator. Functionally, baclofen reduces the effectiveness of compensatory ocular motor reflexes. Since PAN may result from an inability to stabilize elements of velocity storage, reducing the storage time constant would also shorten the VOR time constant and stabilize the system.

Larmande et al.[63] reported a case of periodic alternating ping-pong gaze (PAPPG). The lesion responsible for the movement was initially unilateral, and the PAPPG was intermittent and associated with Cheyne-Stokes respiration. Since PAPPG is usually observed during coma and in connection with widespread bilateral hemispheric ischemia, this particular manifestation is interesting and necessitates reevaluation of the possible cause of the disorder.

Reflex Nystagmus

Reflex (Baer's) nystagmus is a low-amplitude, high-frequency acquired nystagmus associated with erosion or other superficial lesions of the cornea.[64]

See-Saw Nystagmus

Although see-saw nystagmus (SSN) is usually pendular, a recent case was reported with jerk SSN due to brain-stem infarction.[65] No ocular motility records were presented. Two cases (siblings) of congenital SSN associated with retinitis pigmentosa have also been reported.[66] Although it has previously been reported that in congenital SSN the rising eye extorts and falling eye intorts, these patients exhibited the characteristic rising-eye intorsion and falling-eye extorsion seen in patients with acquired SSN. The authors concluded that it is not possible to diag-

nose congenital from acquired SSN solely from the torsional component. It was gratifying to read that, despite the presence of retinitis pigmentosa, the authors did not fall into the trap of ascribing this congenital nystagmus to the sensory defect. In another report, SSN was described in a patient who suffered bitemporal hemianopia due to head trauma.[67] Curiously, the SSN ceased temporarily after ingestion of alcohol; it also disappeared during sleep. See-saw nystagmus has been associated with a Chiari malformation.[68] This patient, who complained of OSOP, improved after surgical decompression. The authors pointed out the importance of the association between SSN and Chiari malformations since early diagnosis and decompression might improve neurologic function and prevent further deterioration. Kanter et al.[69] presented a patient with SSN whose lesion was localized by MRI to the paramedian ventral midbrain with involvement of the right interstitial nucleus of Cajal. This was the first MRI study of SSN associated with a presumed brain-stem vascular event.

Torsional Nystagmus

Using the new torsional search coil, Noseworthy et al.[70] reported on a case of torsional nystagmus secondary to a midpontine lesion that was probably a venous angioma. The waveforms had both linear and increasing-velocity slow phases. The nystagmus increased during active pitch rotations of the head and damped with convergence. The authors postulated a disruption of central vestibular connections.

Upbeat Nystagmus

Upbeat nystagmus was described in a family whose members have a dominantly inherited, early onset, nonprogressive syndrome that includes mild cerebellar ataxia associated with cerebellar vermian atrophy seen on MRI.[71] The slow phases were linear and the nystagmus obeyed Alexander's law. Two other patients with upbeat nystagmus were described; they suffered from caudal brain-stem dysfunction.[72] The authors suggested that dorsal paramedian damage in the rostral medulla was probably the critical lesion responsible for the upbeat nystagmus.

Vertical Nystagmus

Ishikawa et al.[73] described a patient with neuro-Behçet syndrome whose vertical nystagmus was upbeat and changed to downbeat with caloric stimulation. Some

months later her upbeat nystagmus had decreased and she developed downbeat nystagmus on downgaze. Initially, tilting her head decreased her upbeat nystagmus, but during the latter examination, the tilt caused the downbeat nystagmus to increase. Both findings suggested a vestibular component to the patient's vertical nystagmus. The eye movement recordings revealed slow phases that could be linear, or increasing- or decreasing-velocity waveforms. In another case, a patient exhibited upbeat and downbeat nystagmus as a result of a thalamic arteriovenous malformation and no sign of posterior fossa disease.[74] No eye movement recordings were provided.

Vestibular Nystagmus

Gresty et al.[75] studied eye movement responses to combined linear and angular head movement. The response to linear motion was evaluated by subtracting responses to head-centered angular oscillation about a vertical axis (on-axis) from those evoked by similar oscillation with the head displaced 30 cm eccentrically from the axis (off-axis). This assumes a linear summation of both effects. The eccentric position evoked movements of higher velocity, especially at the higher frequencies and when subjects imagined near targets. The gain of the linear response under this condition was about -1, suggesting that eye velocity compensates for linear head velocity, and is in accord with the thoretical prediction that, when viewing near targets, eye movements compensate for linear head motion. Grossman et al.[76] evaluated frequency and velocity ranges of natural head rotations. They found that the maximum fundamental horizontal frequencies during walking and jogging were 1 and 3 Hz, respectively, whereas the maximum vertical frequencies were 4 and 3 Hz. However, the vertical rotations contained significant harmonic components that could reach 15 to 20 Hz during vigorous jogging. Voluntary horizontal head rotations could produce 5 Hz, while vertical rotations produced up to 4 Hz. Since head velocities during walking and jogging (horizontal and vertical) were less than 160 degrees per second whereas voluntary head shaking could produce velocities greater than 500 degrees per second, it is not surprising that OSOP accompanies vigorous head rotations; they may reflect unnatural demands on the VOR.

Hain et al.[77] studied head-shaking nystagmus (HSN) in patients with unilateral peripheral vestibular lesions. In addition to HSN, whose slow phases are directed toward the side of the lesion, all subjects showed a prolonged, lower-amplitude reversal phase. The authors were able to explain this behavior using a model that included the central velocity-storage mechanism, Ewald's second law, and adaptation of primary vestibular afferent activity. They concluded that, in patients with unilateral peripheral lesions, HSN always has both phases and that the reversal phase does not suggest clinical recovery but rather a short-term adaptive process together with a significant unilateral peripheral lesion. When HSN does not have a

reversal phase it may be caused by a central lesion. The authors pointed out that HSN was not specific for vestibular lesions since one of their normal subjects also had HSN. However, Takahashi[78] reported three cases of bidirectional HSN caused by central vestibular disturbance. Thus, both central and peripheral lesions can cause bidirectional HSN.

It has been found that under conditions of microgravity, a nonconvective caloric nystagmus exists. However, under usual clinical conditions (normal gravity), about 85% of the maximal thermal reaction is explained by the convection hypothesis of Bárány.[79] An examination of nystagmus intensity in the supine and prone positions in 22 healthy ears found high interindividual variance and an exceptional case with predominately nonconvectively released nystagmus. A vertical nystagmus or vertical nystagmus component can sometimes be elicited with caloric stimulation of the horizontal canals. Such perverted nystagmus suggests brain-stem pathology. However, a case was presented with confirmed peripheral pathology in which vertical nystagmus was elicited in this way.[80] The author discussed the possibility of dissociated canal dysfunction secondary to a unilateral horizontal semicircular canal. Caloric nystagmus has been demonstrated in a chronic persistent vegetative state with intact brain-stem reflexes. Recently, caloric nystagmus was recorded in a patient with an isoelectric EEG.[81] This is further evidence of the brain-stem nature of the caloric nystagmus reflex.

Lin et al.[82] studied direction-changing positional nystagmus. Although this is commonly considered an indication of central disease, these authors found peripheral vestibular lesions. They also found that apogeotropic nystagmus was no more frequent in central lesions than was geotropic nystagmus. Using a portable recording apparatus, Nishikawa and Nishikawa[83] were able to record the nystagmus in a case of Meniere's disease before, during, and after the vertiginous attack. Before the attack, the nystagmus was toward the affected side, at the onset of the attack, toward the nonaffected side, and both during and after the attack, toward the affected side. No hypothetical mechanisms were proposed to account for this time course. Hain et al.[84] studied postrotary nystagmus in normal subjects and patients with cerebellar lesions. The vestibular response of the normal subjects decayed with a time constant of 19.6 seconds when upright and 7.2 seconds after tilting prone, whereas those with midline cerebellar lesions showed no effect of the tilting. The authors concluded that the midline cerebellum regulates a neural network in the brain stem that perseverates peripheral vestibular input.

Barratt et al.[85] studied responses of patients with neuro-otologic disease to off-axis rotation. Patients with vestibular neurinectomies who had asymmetric responses to on-axis rotation showed greater asymmetry with off-axis rotation at high stimulus frequencies. Some patients with cerebellar lesions showed abnormally enhanced or depressed and asymmetric responses in the off-axis rotation when compared with on-axis rotation. All patients with abnormal off-axis responses had positional nystagmus. In contrast, patients with benign paroxysmal vertigo (BPV) or chronic linear vertigo did not show abnormal off-axis responses. The authors concluded that off-axis testing may be useful in elucidating patho-

physiology but is not a decisive clinical test for the presence of otolith dysfunction.

Baloh et al.[86] reported on 240 cases of BPV. A torsional paroxysmal positional nystagmus always occurred after a rapid position change from sitting to head-hanging. Their data suggested a peripheral posterior semicircular canal origin of BPV. The authors noted that unless the patient was tested during the period of acute episodes of vertigo, positional nystagmus was not observed. Lackner and Graybiel[87] found that Coriolis cross-coupled angular acceleration stimulation readily induced motion sickness under terrestrial conditions. This susceptibility was in direct proportion to gravity. These findings explain the low susceptibility of Skylab astronauts to motion sickness during in-flight stimulation. In another study, these authors found that head movements in free fall, especially pitch movements, were provocative until adaptation occurred.[88] They concluded that space motion sickness was a consequence of prolonged exposure to a nonterrestrial force background rather than of exposure to free fall per se. Watt[89] concluded that prolonged exposure to inappropriate VOR will usually lead to motion sickness. He suggested that though VOR gain may be decreased in weightlessness initially, rapid compensatory mechanisms restore it to normal within minutes of reaching weightlessness. This compensation is not enough to protect against the development of motion sickness, however.

SACCADIC INTRUSIONS AND OSCILLATIONS

Nonnystagmic ocular motor intrusions and oscillations represent solely saccadic or saccadically initiated instabilities. I have identified 16 varieties of saccadic intrusions and oscillations that have been characterized in the literature by many other terms, including ten that erroneously contain the term *nystagmus*. This section contains discussions of recent studies of saccadic intrusions and oscillations; they are in alphabetical order. For discussions of other types, see previous reports on the subject.[1–4]

Bobbing/Dipping

Rosa et al.[90] studied five cases of ocular bobbing, with postmortem examination in two. Three of the cases were described as typical, one atypical, and one asymmetric. The commonly damaged nervous structure in four of the five cases was the pons. One of their cases was the first reported case of bobbing in a patient with Creutzfeldt-Jakob disease. The authors hypothesized that both mesencephalic and medullary burst neuron centers played a prevalent part in ocular bobbing. There is some disagreement regarding both the necessity for patients to be comatose to ex-

hibit dipping and whether cerebral structures are involved.[91, 92] Although coma may not be necessary, it is agreed that a reduced level of consciousness is required for dipping to occur. In a report on the clinical spectrum of ocular bobbing and dipping, Mehler[93] reported the first case of reverse dipping. Bobbing begins with a fast downward phase, reversed bobbing with a fast upward phase, and dipping with a slow downward phase; all have been previously described. Reverse dipping begins with a slow upward phase, and its description completes the four possibilities. Rosenberg[94] described two patients who had three of these four types of movements: bobbing, dipping, and reverse bobbing. He suggested that they may all be differing manifestations of the still-unknown pathophysiologic process. The description of a case of atypical bobbing in acute organophosphate poisoning is another instance where a severe intrinsic pontine lesion is not required for atypical bobbing.[95] Dipping has been described in a patient with deafness and pinealoblastoma.[96] Since this was a discrete lesion, the authors discussed possible mechanisms. A clear difference between this and previous cases was that the patient was alert. In this and most other articles on bobbing and dipping, the absence of good ocular motility records weakens speculation and hypotheses on responsible mechanisms.

Convergence-Retraction "Nystagmus"

Oohira et al.[97] studied five patients with convergence-retraction "nystagmus." In four of the five, the waveforms were typical: adducting fast phases and decreasing-velocity slow phases. In the other, the fast phases appeared to be slow saccades possibly because of small or absent innervational pulses. The authors compared the fast phases of convergence-retraction nystagmus with the associated and voluntary adducting movement seen during vertical saccades in normal subjects. They suggested that a disorder in the neural circuitry that regulates the excitability of neurons involved in these eye movements is responsible for convergence nystagmus.

Flutter

Herishanu et al.[98] reported an unusual case of blink-induced flutter in a patient with multiple sclerosis. The flutter was present whenever the patient blinked or attempted to blink while her lids were held open; no spontaneous saccadic intrusions or oscillations were observed during fixation. Furman et al.[99] reported spontaneous remission of ocular flutter and other saccadic intrusions and oscillations in two women. Since their remission preceded the appearance of a primary neoplastic process remote from the nervous system, the authors warned that such remis-

sion does not necessarily imply a benign outcome. Bergenius[100] studied the saccadic abnormalities in patients who exhibited ocular flutter. He found opsoclonus, increased frequency of SWJ, high peak velocity of voluntary saccades, and hypermetria. Salonen et al.[101] reported a case of sarcoidosis where flutter was the only neurologic sign. The flutter disappeared in 2 months, and the authors suggested that neurosarcoidosis should be considered as a potential cause.

Hotson investigated the proposal that voluntary flutter is an inherent capability in humans.[101a] He found no difference in saccadic peak velocity between control subjects and those capable of voluntary flutter. With training, control subjects were able to learn to produce runs of voluntary flutter. This learned flutter was composed of recurrent complete saccades rather than saccades interrupted in midflight. He concluded that voluntary flutter is not a genetic trait but a learned event that is usually undeveloped in man.

Macro–Square-Wave Jerks (Bursts/Single)

Fukazawa et al.[102] reported on a patient with marked atrophy of the cerebellum and brain stem. Both macro–square-wave jerks (MSWJ) and macro-saccadic oscillations (MSO) were recorded. By recording both horizontal and vertical eye movements, the authors demonstrated that both these oscillations had a vertical component (i.e., they were diagonal movements). The patient's oscillations almost completely disappeared after administration of several sedative drugs; she denied having OSOP. The discussion of MSWJ, SWJ, and MSO is confused, reflecting the misconception that MSWJ are merely large SWJ; this is an unfortunate and common result of the original naming of MSWJ. Although SWJ are usually of smaller amplitude than MSWJ, we have recorded large SWJ in some patients and small MSWJ in others. Thus, as pointed out in an earlier report,[103] the real difference between MSWJ and SWJ is in the latency between the initial and final saccade. In SWJ it is 200 msec, and in MSWJ it can vary between 50 and 150 msec. The authors also seemed confused about the differences between MSWJ and MSO; these were also pointed out in the earlier study. Table 3 contains the similarities and differences between these three oscillations and square-wave oscillations (SWO), a more recently described phenomenon.[104]

Myoclonus

Myoclonus is a pendular oscillation that conforms to the definition of nystagmus and is so regarded by European writers. It is usually classified separately because of associated rhythmic movements of nonocular muscles in synchrony with the eyes. It was originally referred to as "pharyngeal and laryngeal nystagmus."[105]

TABLE 3.
Characteristics of Square-Wave Instabilities

	SWJ	SWO	MSWJ	MSO
Amplitude, degrees	0.5–5, constant*	0.5–5, constant*	4–30, variable	1–30, increasing then decreasing
Time course	Sporadic/ bursts	Bursts	Bursts/ sporadic	Bursts
Latency, msec	200	200	50–150	200
Foveation	Yes	Yes	Yes	No
Presence in darkness	Yes	Yes	Yes	No

*Amplitudes occasionally may reach 10 degrees.

Ocular myoclonus is a form of segmental myoclonus[106] and is characterized by continuous, rhythmic, to-and-fro pendular oscillation, usually in the vertical plane. Nakada and Kwee[107] pointed out that myoclonus may have oblique and rotary components. They speculated that the generation of myoclonus involved the VOR adaptation mediated by the flocculus and provided a model that might account for it. Myoclonus that is present even during sleep usually persists as a chronic sign until the death of the patient.[108] There have been reports of myoclonus responding to treatment using valproic acid[109] and trihexyphenidyl.[110]

Opsoclonus

Adding to the many substances that have been reported to induce opsoclonus, Dehaene and Van Vleymen[111] presented a case in which phenytoin and diazepam caused opsoclonus. Hunter and Kooistra[112] discussed the neuropathologic findings in a case of opsoclonus. They found structural lesions limited to the cerebellum and inferior olives and severe depletion of Purkinje cells with preservation of granular cells in the neocerebellum and paleocerebellum. Purkinje cells were preserved in the archicerebellum. No abnormalities were found in the paramedian pontine reticular formation of the caudal pons. In a case of opsoclonus associated with adenocarcinoma of the breast, steroid treatment was successful.[113] Ridley et al.[114] looked at omnipause neurons in two cases of opsoclonus associated with oat cell carcinoma of the lung. The light-microscopic appearance of these neurons was normal. The authors concluded that, although the cells appeared normal, it is still possible that their function was disturbed and opsoclonus resulted. Borodic et al. have extensively reviewed the literature of 19 autopsied cases of opsoclonus.[115]

Saccadic Lateropulsion

Benjamin et al.[116] recorded both lateropulsion and upbeat nystagmus as manifestations of central vestibular dysfunction. The patient had a right hemispheric cerebellar infarction. The lateropulsion was contralateral to the side of the lesion. The authors discussed the lesion (shown on a computed tomogram of the brain) as it related to vestibulo-ocular pathways and concluded that the combination of lateropulsion and upbeat nystagmus was caused by an interruption of these pathways.

Saccadic Pulses/Pulse Trains

We hypothesized that the fast phases of so-called abduction nystagmus of INO are really saccadic pulses present in the abducting eye as a result of the weakness in the adducting eye.[117] Zee et al.[118] recently supplied additional evidence in support of that hypothesis. They studied four patients with INO and patched one eye for 1 to 5 days to allow the nervous system time to optimize innervation for the habitually viewing eye. They found that the conjugate adjustment of innervation diminished the abduction overshoot and backward postsaccadic drift made by the habitually viewing eye. The authors conclude that abduction nystagmus is a manifestation of a normal adaptive response in patients with INO.

Square-Wave Jerks/Oscillations

Ohtsuka et al.[119] studied the origin of SWJ under conditions of fixation of targets of variable size. The size of SWJ was found to be directly related to the size of the target; the frequency and duration of SWJ remained constant and were independent of target size. The SWJ within the range of microsaccades disappeared as larger SWJ appeared. The authors concluded that SWJ were enlarged versions of microsaccadic SWJ that appear when viewing very small targets. The authors agreed with Doslak et al.[119a] and concluded that the saccadic intrusion (i.e., the first saccade of a SWJ) is caused by a spurious error signal that takes the eye off the target, creating a real error that is corrected by the return saccade after a normal latency. Even in the dark, the eye position after an SWJ equaled the mean eye position before the SWJ. Thus, SWJ occur normally but are usually of microsaccadic amplitudes. The instability of eye position in the dark in patients with cerebellar degeneration was found to differ from that of normal subjects.[120] In addition to SWJ, unidirectional drifts and corrective saccades were observed. Patients with spinocerebellar degeneration showed unsteadiness in nonvisual eye position con-

trol. In normal subjects, both in light and in darkness, saccadic intrusions provoked position error signals, and corrective saccades were elicited producing SWJ. However, in patients with moderate and marked instability, the saccadic intrusions did not elicit corrective saccades, and SWJ were not observed. The authors concluded that a position error signal was not provoked in these patients in the dark. They further hypothesized that proprioceptive afferent information from eye muscles contributes to controlling eye position in the dark through the cerebellum.

Both SWO and SWJ were reported in an infant preceding the appearance of CN 5 months later.[121] No other nervous system abnormalities were found. The authors hypothesized that these saccadic oscillations and intrusions precluded the early appearance of the developing CN. Such oscillations in infancy may be a benign condition.

Superior Oblique Myokymia

Rosenthal and Selhorst[122] studied a patient with adenocarcinoma who exhibited continuous nonrhythmic cycloversions. The amplitudes of these cycloversions were 2 degrees and 5 degrees, and superimposed on them were conjugate jerky clockwise clycloversions up to 30 degrees that lasted 3 to 5 seconds. These occurred with a frequency of one to three per minute. It is possible that this is an uncommon form of superior oblique myokymia. Until more recordings are made (with the torsional eye coil) of superior oblique myokymia, it will not be possible to differentiate cases such as this from those with a more common form of superior oblique myokymia. It is important to know what the waveforms are before attempting to differentiate one eye movement from another. Without such information, it is virtually impossible to deterrnine if an oscillation is different from, or merely an unusual form of, another oscillation. Simple clinical descriptions of oculomotor oscillations are not accurate enough and can be erroneous.

OSCILLOPSIA

Oscillopsia is the subjective, illusory movement of the stationary world.[123] Normal ocular motor control systems can prevent slip of images on the retina from exceeding about 4 degrees per second. When retinal image velocity (RIV), commonly called "retinal slip," exceeds that value, visual acuity begins to decline and OSOP may result. Oscillopsia is a common consequence of acquired nystagmus but not of saccadic intrusions or oscillations. Also, most patients with CN do not experience OSOP despite slow-phase RIV that may exceed 100 degrees per second. This subjective feeling of motion of the environment has been associated

with acquired nystagmus caused by an Arnold-Chiari malformation[124] or even carbamazepine therapy.[125] Reduction of the nystagmus has caused an equal diminution of the OSOP.[126] Since any excessive RIV may be related to OSOP, nystagmus need not be present. A change in the VOR gain sufficient to cause high RIV may also result in OSOP. Verhagen et al.[127] reported three cases in a family with congenital vestibular areflexia. There was no nystagmus in these patients, and the OSOP occurred only during head or body movements. Oscillopsia has also been caused by aminoglycoside ototoxicity.[128] The authors referred to this as "subjective oscillopsia," which is redundant since all OSOP is subjective. In these patients, OSOP occurred during movement, and there was no nystagmus or other ocular oscillations.

Although there is a direct relationship between RIV and visual acuity, there is not one with OSOP. The magnitude of OSOP cannot be directly related to RIV; in acquired downbeat nystagmus, the magnitude of OSOP is approximately 0.37 of the nystagmus magnitude. At the far end of the spectrum are those patients with CN in whom there are both high RIV and no OSOP. Brandt and Dieterich[129] proposed that the dissociation between RIV and OSOP can be explained by a combination of two separate mechanisms that involve motion perception. These are a physiologic elevation of thresholds to detect object-motion with moving eyes, and a pathologic elevation of thresholds to detect object-motion with either infranuclear ocular motor palsy or supranuclear ocular oscillations. They found that thresholds for egocentric detection of object motion were higher in patients with downbeat nystagmus than in normal subjects and the thresholds increased with increasing nystagmus amplitude. There was a partial suppression of visual motion perception for both the RIV caused by the nystagmus and for objects moving within the visual scene. During smooth pursuit, the motion detection threshold of the visual background increases proportionally with eye velocity. Thus, there appears to be a visual motion perception suppression during eye movements that may reflect a basic sensorimotor mechanism. It has been suggested that the nervous system contains mechanisms whose tuning characteristics in both space and time can be modified and make it possible to do what a camera cannot: resolve form and motion simultaneously and independently.[130] This independence is used clinically by the oscillatory movement displacement threshold (the smallest amplitude of oscillation that causes the perception of movement) to access ocular neural dysfunction in the presence of media opacities.[131]

In an attempt to further understand the relationship between RIV and OSOP, Leigh et al.[132] studied retinal image stabilization in subjects with CN. All subjects reported OSOP during retinal image stabilization, but the condition of stabilization varied from one individual to another. They concluded that several mechanisms operate to maintain special constancy in CN; some individuals appear to use one more than another. The mechanisms used included extraretinal signals, elevated threshold for motion detection, and "suppression" of visual input except during foveation periods. When only part of the visual field was stabilized, one subject reported OSOP of that stabilized central field while the peripheral surround was

perceived as not moving. He could reverse the perceptions, causing OSOP of the surround instead of the central field.

Since RIV is related to OSOP in acquired nystagmus, the effects of retinal image stabilization were studied in nystagmus caused by neurologic disease.[133] The stabilization was progressively increased in eight patients with acquired nystagmus until OSOP was abolished; this was achieved at RIV of 5 degrees per second or less. In five patients, further increases in stabilization caused the OSOP to reappear in the opposite direction. In addition to reducing OSOP, stabilization improved visual acuity in four of five patients tested. Both electronic stabilization and an optical device were used to stabilize images of the real world on the retina.[134, 135] Although the optical device provides stabilization over a small central field, it is useful in helping patients to read or watch television by reducing or eliminating their OSOP.

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NEURO-OPHTHALMOLOGY

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Edited by

Simmons Lessell, M.D.
Professor of Ophthalmology
Harvard Medical School
Director of Neuro-Ophthalmology
Department of Ophthalmology
Massachusetts Eye and Ear Infirmary
Boston, Massachusetts

and

J. T. W. van Dalen, M.D., Ph.D.
Associate Professor of Ophthalmology
Department of Ophthalmology
The University of Arizona Health Sciences Center
Tucson, Arizona

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