CHAPTER 9

Nystagmus and Other Ocular Motor Oscillations and Intrusions

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If one is to understand the pathophysiology of ocular oscillations it is imperative to distinguish between those involving only the slow system and those that are purely saccadic. Oscillations containing both saccades and slow phases require identification of the causative phase (i.e., that which takes the eyes away from their intended direction) and the corrective phase. Modern methods of recording have enabled us to make these determinations and thereby clarify the ocular motor mechanisms responsible for the particular oscillation in question. Table 1 lists 43 types of nystagmus 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 chapters on the subject.[1–4]

The definitions and categorizations used herein are a result of the systematic application of criteria derived from accurate ocular motility recordings. Such criteria clearly differentiate between nystagmus and other ocular motor oscillations and, as a result, some eye movements described by the word ''nystagmus,'' were found to be saccadic oscillations. Most oscillations were named without the benefit of accurate ocular motor recordings. Quotation marks are used for those os-

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TABLE 1.

Nystagmus*

- Acquired
  - "Fixation"
- Arthrokinetic
  - Induced
  - Somatosensory
- Audiokinetic
  - Induced
- Bartels'
  - Induced
- Bruns'
- Centripetal
- Cervical
  - Neck torsion
  - Vertebral-basilar 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
  - Gaze-paretic
  - Deviational
- Horizontal
- Induced
- Intermittent vertical
- Jerk
- Latent/manifest latent
  - Manifest latent
  - Monocular "fixation"
- Lateral medullary
- Optokinetic
  - Induced
  - Kinetic
  - Optic
  - Optomotor
  - Panoramic
  - Railway
  - Sigma
  - Train
- Optokinetic after-
  - Induced
  - Post-optokinetic
  - Reverse post-optokinetic
- Pendular
- Periodic/aperiodic alternating
  - Alternans
- Physiological
  - Endpoint
  - Fatigue
- Pursuit after-
  - Induced
- Pursuit-defect†
- Rebound
  - Induced
- See-Saw
- Somatosensory
- Spontaneous
- Stepping around
  - Apparent/real
  - Induced
  - Somatosensory
- Torsional
  - Rotary
- Uniocular
- Upbeat
- Vertical
- Vestibular
  - A(po)geotropic/geotropic
  - Alternating current
  - Bechterew's
  - Caloric/caloric-after
  - Compensatory
  - Electrical/faradic/galvanic
  - Head-shaking
  - Induced
  - L-nystagmus
  - Labyrinthine
  - Perverted
  - Pneumatic/compression

TABLE 1.—*Continued*

Lid	Positional/alcohol
Miner's†	Positioning
Occupational	Postrotational
Muscle-paretic	Pseudocaloric
Myasthenic	Rotational/perrotary
	Secondary phase

*Synonyms and other terms found in the literature are indented under the preferred or more inclusive designation.
†May not exist.

cillations that are *not* truly nystagmus or for purely subjective clinical terms found in the literature that are inadequate, not clearly defined, or have been misapplied to several different types of oscillations. Since these ambiguous and/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 indicate that these are non-nystagmic oscillations (i.e., saccadic). The best terms reflect the mechanism thought to be responsible for the eye movement (e.g., saccadic pulse); such terms maximize the information carried to the reader and clearly describe a movement by its well-defined component parts.

NYSTAGMUS

Nystagmus is a biphasic oscillation containing slow eye movements that are responsible for its genesis and continuation. Fast eye movements (saccades), if they are present, are corrective and not the basic ocular motor dysfunction. The two phases of nystagmus are approximately equal in amplitude. What follows is a discussion of each nystagmus type in which significant work has been done in the past three years, presented in alphabetical order. Definitions and discussions of all types of nystagmus may be found in the above previous volumes.

Bartels' Nystagmus

Bartels' nystagmus is an induced nystagmus not previously listed in the previous volumes. It is elicited by placing +20 D lenses over the eyes and turning the head to some position other than primary.[5, 6] The induced nystagmus beats in the direction of the head turn and is also elicited using −20 D lenses.

TABLE 2.
Saccadic Oscillations and Intrusions*

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

*Synonyms and other terms found in the literature are indented under the preferred or more inclusive designation.

Bruns' Nystagmus

A recent article suggests floccular involvement is responsible for the development of Bruns' nystagmus.[7] Depending on the amount of flocculus compression, no nystagmus, vestibular nystagmus, Bruns' nystagmus, or bilateral gaze-evoked nystagmus (GEN) is possible. Successful tumor removal caused Bruns' nystagmus to disappear in two of three cases and bilateral GEN to disappear in three of four cases.

Congenital Nystagmus

An interesting article recently appeared on the differential diagnosis of congenital nystagmus (CN).[8] Many useful diagnostic criteria were reviewed and comparisons made to features common to certain forms of acquired nystagmus. However, the suggestion that many patients with CN experience oscillopsia when attempting

to track moving targets or when reading in a moving vehicle is not consistent with our observations. Also, references to deficits in the pursuit or optokinetic systems are misleading, since it is the CN that reverses during pursuit and not a reversed pursuit, as has been claimed elsewhere.

Waveform analysis has been shown to result in the classification of patients with CN into three unambiguous groups.[9] These groups exhibit both different clinical signs and relationships to strabismus. Also contained are the statistics of CN and latent/manifest latent nystagmus (LMLN). This includes percentages of patients with null angles, convergence nulls, various CN waveforms and combinations of waveforms, strabismus, and so on. These statistics show that heredity plays a large role in determining the waveform as well as these other characteristics. An interesting finding is that, given a patient with strabismus and nystagmus, it is more probable the nystagmus will be CN rather than LMLN despite the fact that all LMLN patients have strabismus and only 29% of pure CN patients have strabismus; this is due to the greater numbers of CN patients as opposed to LMLN patients. An article on CN in identical twins concluded that differences were a result of environmental influences[10]; the previous article used the statistics of CN to show that even in this case of twins the similarities and differences are related to inheritance.

An interesting article has reported several CN patients with two nulls.[11] We have never recorded two nulls in a patient with CN. Patients with alternating fixation and LMLN that varied according to Alexander's Law, and CN patients with a latent component and alternating fixation, can *appear* to have two nulls. Also, CN patients with periodic alternating nystagmus (PAN) can appear to have more than one null. It is possible that the patients discussed in this article had some of these conditions and, in such cases, one should be cautious about corrective surgery on nulls that are not static. The authors compared the static null with the dynamic null; the latter was measured both during smooth pursuit and suppression of the VOR. Thus, static nulls that were originally in right gaze would appear as dynamic nulls in left gaze during pursuit or VOR suppression eye movements to the right and vice versa. This dynamic null results from the shifting of the static null that originally was at a gaze angle in the other direction and should not be confused with what seemed to be a second null.

The question of whether or not CN patients can generate smooth pursuit eye movements is one that has been dealt with extensively in previous chapters on nystagmus. It is now generally (and grudgingly in some cases) agreed that patients with CN can indeed pursue moving targets. The only remaining question is, by what mechanism? Kommerell has recently introduced the hypothesis that the mechanism is target offset from the fovea rather than retinal slip velocity.[12] He found that CN patients slowly pursued parafoveal afterimages. This, combined with the observation that CN patients often make refixations between stationary targets that consist of both saccades and slow eye movements, resulted in that hypothesis. The slow eye movements are usually part of the CN waveform. An-

other interpretation of this observation is that CN patients have the same ability to slowly move their eyes in the direction of a foveal afterimage as normal subjects do; it was Kommerell who showed the latter in an earlier study. Since this ability in normal subjects is insufficient proof that they pursue by target offset alone (no one has yet claimed that target offset is the main stimulus for smooth pursuit), it is equally insufficient proof that this is the mechanism used by CN patients. The ocular motor system seems capable of using all the information at its disposal whether it be position, velocity, or acceleration. Both normal subjects and CN patients use this information, and it has been shown that CN patients do perceive retinal shift velocities at a cortical level.[13]

The clinical observation that CN is usually discovered after several weeks or months of life has been used to support the idea that CN develops secondarily to a primary defect of retinal slip control. However, CN is often seen at or shortly after birth and the waveforms of such patients are exactly the same as those in whom it was discovered later; this implies the mechanisms responsible for both are the same. The sensory-defect theory of causation thus fails to account for the CN seen in all patients. Ocular motor instability that is not secondary to any sensory defect can explain all cases of CN. In an attempt to demonstrate that patients with CN have and use normal smooth pursuit systems, I have presented data that spans two decades and illustrates the strategies used by many CN patients to pursue a moving target.[14] A substantial portion of that article deals with the interpretation of eye movement records that contain spontaneous oscillations (CN or other types of nystagmus) superimposed on smooth pursuit. Methods are discussed that enable one to separate the ongoing waveform from the pursuit signal. My conclusion (perhaps not surprising) is that the smooth pursuit system is intact and both retinal slip velocity and position information are used to match eye velocity and position to those of the target; just as normal subjects do. An updated model of my original hypothesis[15] is included in the article to illustrate how this may be accomplished.

Just as the presence of CN waveforms complicates the analysis of smooth pursuit gain, it also disrupts the analysis of the vestibulo-ocular reflex (VOR) and the optokinetic reflex (OKR). Gresty et al. found that the phase relationship of the nystagmus to the motion stimulus was the same as in normal subjects, and concluded that estimates of VOR gain were not meaningful because of the contamination caused by the CN waveform.[16] These authors found clear evidence of VOR suppression by noting that the amplitude modulation and reversal of nystagmus was in phase with the vestibular stimulus at all frequencies of oscillation. They concluded that the underlying vestibular function in CN patients is normal. Clear nulls were identified during VOR suppression and correlated with expected null shifts that are caused by pursuit. A final conclusion was that the null shifts known to occur during smooth pursuit occur immediately at the onset of pursuit for transient stimuli. This supports the conclusion I reached after studying the Rashbass data presented by Optican et al.[3, 17]

Carl et al. investigated the mechanisms underlying head shaking in CN.[18] These

authors realized that if head shaking was to increase visual acuity at least some of the acuity loss must be due to the CN waveform-induced retinal slip, the VOR gain had to be something other than one and the head shaking had to be correlated with the nystagmus. Their method for estimating VOR gain in the presence of CN waveforms compared the velocities of head and eye at the point of peak head velocity in each half cycle of head oscillation. Unfortunately, this method totally disregards the interval during the CN waveform when the target is foveated. If the VOR is to be used effectively during active head shaking to stabilize the eye in space, it must do so during these foveation periods in order to improve acuity; at all other times during the CN waveform the eye is not on target and stabilizing the eye in space would not result in any change in acuity. For reasons too complicated to discuss in this chapter, it is my opinion that the ''gain'' figures arrived at by their methodology are not true indications of the VOR gain of these patients. In fact, the VOR ''gains'' calculated did not correlate well with either the acuities exhibited by the particular patients studied or their lack of vestibular symptoms. This article does contain a very good illustration of how head shaking can be used to steady the gaze signal during the interval of target foveation. By comparing the steady fixation and head shaking records of patient 4 in their Figures 1 and 5, one can see a large extension of the stationary foveation periods during head movement. In agreement with other authors, it was concluded that head shaking was generally of no apparent visual benefit and probably represented a tremor associated with the CN.

A recent review discussed head oscillations and positions associated with CN and other types of nystagmus.[19] In another article, the conclusion that head position does not influence CN[20] is inconsistent with the observation of preferred head positions in some patients; it was not stated whether any of those tested had a preferred position. Given the importance of fixation attempt, the data on CN under various conditions of eyelid position and room illumination are meaningless, as are the extremely variable values of VOR ''gain'' reported (see above). No specific waveforms were identified, but two were evident in the Figures, jerk and jerk with extended foveation. Finally, the observation that CN patients sometimes use their slow phases to move their eyes to a new position during combined eye and head movements is not surprising, since they do so with a fixed head. The description of this adaptive behavior as normal or a type of apraxia is not justified by the data.

An excellent study on the OKR of patients with CN concluded that the responses seen when such patients are exposed to optokinetic stimuli are the results of an adapted optokinetic nystagmus (OKN) system[21]; The data did not support the hypothesis that a basic defect in the OKN system was the cause of CN. These authors showed normal optokinetic responses to vertical OKN stimuli by patients with horizontal, vertical, and torsional CN. They also showed that the response of a patient with horizontal CN to a horizontal optokinetic stimulus was not the normal sawtooth response even when the CN was reduced to zero both by biofeedback and by base-out prisms. Their experiments further suggested that despite

the abnormal OKR exhibited in the plane of the spontaneous CN, the full-field retinal image motion did excite the subcortical OKN pathways; this is consistent with the above-mentioned findings that retinal slip is perceived at a cortical level and with my previous assertion that the OKN stimulus-induced null shift was prima facie evidence of an OKR in the correct direction.[3]

Several articles have examined the eye movements of human albinos. As a result of their studies, Collewijn et al. divided the patients into three classes.[22] The first, and largest, class was characterized by vigorous spontaneous nystagmus, the absence of true horizontal OKN, and the ability to control gaze direction. Class 2 contained two subjects with vigorous unidirectional jerk nystagmus that reversed direction spontaneously or as the result of visual stimulation. The stimuli caused a reversal of the nystagmus that was unfortunately termed ''inverted pursuit,'' and the third class had very little spontaneous nystagmus and virtually normal eye movement responses. Only pursuit of motion in the temporal direction projected onto the temporal half of the retina was defective in these patients. Using visual-evoked cortical potentials, anomalous visual projections were confirmed in all subjects. It was hypothesized that these anomalous projections were a likely cause underlying the nystagmus. Large intersubject differences were evident. The problem with assuming a causal relationship between the anomalous visual projections of albino patients and CN is that the albino patients have the same CN as patients who are otherwise normal, and, therefore, presumably have no abnormal visual projections. This is a good in-depth study of albino patients with CN and contains a wealth of data. I disagree with some of the terms used that imply reversal of pursuit or reversal (inversion) of OKN, but if one remembers that what really reverses is the CN waveform, the article has much to recommend it. For reasons that are fully discussed in my article on CN and smooth pursuit,[14] I would disagree with the authors' contention that no ''true'' horizontal smooth pursuit is shown by their records.

Another article on the eye movements of human albinos by St. John et al. is not recommended reading.[23] It contains numerous errors as well as yet another meaningless discourse on the effect of light and dark on CN; they failed to control for fixation attempt. The waveforms shown in their Figures were misidentified (probably because of low bandwidth recording), no bandwidth was given and no indication was given of which eye was recorded or was fixating. The authors were surprised that albino patients with CN had difficulty in producing smooth pursuit segments even at low stimulus velocities. To their credit, however, they did recognize the superimposition of CN on pursuit records and the fact the smooth pursuit might be difficult for albino patients who have poorly developed foveas. Another study of albino subjects with CN concluded that their vestibular responses showed an abnormally short time constant and that full-field optokinetic stimulation generated no nystagmus response.[24] Speculation was offered that these abnormalities might be due to defects in mathematical integrator networks (gaze-holding or velocity-storage). For reasons discussed above, I disagree that calculating VOR gain by taking the ratio of maximum eye velocity and maximum head velocity is

indicative of true gain in the presence of spontaneous oscillations. The slow phases have high velocities due to the CN waveforms. The authors themselves recognized the problem by stating that their VOR ''gains'' might reflect the superposition of the vestibular response and the underlying CN; they do. Since the values of these ''gains'' are suspect, it serves no purpose to comment on the speculations that resulted from them. By generalizing the results found in three albino patients to the whole population of patients with CN, the authors fell into a dangerous trap. The statement that there would be little advantage to stabilize images against retinal slip due to low-frequency head movements when retinal blur is being generated by the CN, is flatly contradicted by the extended foveation periods and high acuities present in many CN patients who are not albinos. It is just as important to them to have a good VOR as it is to normal subjects.

A study of visual acuity and CN concluded that, in general, intensity is not the best indicator of acuity.[25] Near fixation was used to improve acuity and the waveforms of CN noted both at near and far. The intensity of eight patients decreased, while the other five increased despite the acuity of all patients increasing. The authors concluded that foveation time is a more important indicator of visual acuity than intensity and suggested that the waveform factor advocated by Dell'Osso and Flynn[26] would yield a better indication of expected visual acuity.

Foveation time per cycle has long been recognized as the key factor in better acuity. Intensity is useful as a measure of foveation time per cycle only when the waveform of the CN does not change. If waveform changes yield a greater foveation time per cycle, acuity will increase regardless of the intensity change. Dickinson and Abadi recently published a unique study on the influence of CN-type oscillations on the contrast sensitivities of normal observers.[27] They subjected normal observers to the same retinal image motion experienced by CN patients by moving the target with waveforms that approximated CN waveforms. They found that, for all waveforms, the length of foveation period showed good correlation with visual resolution of the targets and that the contrast sensitivity functions exhibited by normal subjects under these conditions were similar to those shown by CN patients. In a related finding, Loshin and Browning found that the contrast sensitivity shown by albinotic patients was due mainly to the CN.[28] In another study relating visual acuity and various parameters of CN waveforms, Funahashi et al. also concluded that foveation time per cycle (''plateau time'' after the fast phase) was the most important parameter.[29] In a previous work, while discussing sensory defects, I stated that waveform (i.e., pendular vs. jerk) was not correlated to visual impairment. This was misinterpreted (mistranslated) in this article to apply to visual acuity. Waveform is obviously related to acuity, since foveation time is directly responsible for good acuity. A second article by Dickinson and Abadi reported that there was a greater than normal incidence of high spectacle astigmatism in CN patients (albino patients included).[30] This astigmatism is predominantly with-the-rule and corneal in origin (anterior surface).

A study of several patients with reduced vision in one eye and a variable esotropia found that they had the nystagmus blockage syndrome.[31] From the descrip-

tions of the patients it was clear that all had CN or at least a combination of CN and LMLN. Unfortunately, no eye movement recordings were shown to verify the exact type of nystagmus present. These authors also confirmed the results first obtained by Spielmann,[32] who recommends combining posterior fixation sutures (Faden) with the Kestenbaum procedure: Spielmann has also reported on the surgical correction of vertical null positions in patients with CN and MLN[33] and of patients with both a null when fixating at distance and a blockage null at near.[34] These are interesting articles with practical insights into the problems associated with such complicated cases.

Optican and Zee have recently put forth a model that attempts to explain the genesis of CN.[35] They postulated a reversed velocity pathway associated with the neural integrator. The details of this model and the variety of findings it produces are too extensive to discuss in this chapter. The model was able to duplicate some CN waveforms, but produced behaviors never found in CN patients and, by its very nature, would seem to preclude the type of accurate smooth pursuit that has been reported elsewhere. Nevertheless, this first attempt at modeling the cause for CN does provide some insights into the types of oscillations possible with certain instabilities introduced via the feedback pathways. The model's value lies in both the positive and negative results is produced.

Two reports have appeared that describe vertical CN. In one, a downbeat nystagmus was found in an 8-year-old boy whose 28-year-old mother also had downbeat nystagmus.[36] The eye movement records in this paper are of very poor quality and, therefore, it is impossible to accurately identify the waveform. Despite a null in up-gaze, this patient was given base-up prisms, which effectively put him in down-gaze. One year later, base-down prisms were prescribed, which allowed him a normal head posture while reading. It was noted that convergence damped his downbeat hystagmus, but no attempt was made to use base-out prisms. Since the discussion section of this paper returns to extolling the virtues of using base-up prisms, it would seem that the author was as confused as I was after reading it. It is difficult to understand how base-up prisms could do this patient any good at all; the base-down prisms should, and did, work and base-out prisms probably would have worked as well or better.

Hoyt and Gelbart studied 13 patients, nine of whom presented with vertical nystagmus associated with congenital ocular abnormalities.[37] Four patients had upbeat nystagmus associated with Leber's amaurosis. In three, the upbeat nystagmus became horizontal prior to 1 year of age; the other patient, at age 8, still had upbeat nystagmus. Five patients with albinism initially had see-saw nystagmus. In two cases it resolved by 1 year of age, but in the other three it was still intermittently seen; their ages ranged from 2½ years to 5 years old. Unfortunately, no ocular motility recordings are shown in this article, and despite the authors' acknowledgement that there are no basic differences between so-called ''sensory-defect'' and ''motor-defect'' nystagmus, they persist in using these meaningless terms in their discussion.

One should always search carefully for associated ocular lesions in all infants

presenting with nystagmus; it is my opinion that the continued use of terms that incorrectly imply causality and/or waveform does more harm than good. Congenital nystagmus is a specific ocular motor sign, not a syndrome. That CN can appear in a large variety of patients with a myriad of other clinical signs is not in question. Regardless of the clinical picture, the CN waveforms and behavior (implying mechanism) are the same for all of these patient groups, including those without any sensory defect. Since the very same CN is present with or without associated sensory defects, such defects cannot be the cause of CN. This is true even for those rare cases where the CN can be alleviated by correcting the sensory defect. This reduces the fixation effort and thereby may reduce, or eliminate, the CN much in the same way that closing one's eyes usually reduces that effort and can eliminate CN. However, when that effort is caused to return, the CN does also. Thus, it was not the sensory defect nor the eyelid position that caused the CN but the accompanying effort to see.

Dissociated Nystagmus

Weissman et al. recently presented preliminary results of a study of the nystagmus associated with spasmus nutans.[38] They identified the waveform as a pendular, dissociated nystagmus where the key characteristic was the variable phase relationship of the oscillations of the two eyes. From second-to-second, the amplitudes, frequeny, and phase of the nystagmus can vary; this makes it distinguishable from CN, which is a conjugate oscillation (i.e., the eyes are in phase with each other).

An interesting study of a patient with internuclear ophthalmoplegia (INO) showed a dissociated downbeat nystagmus.[39] During gaze downward to the left, the left eye had a prominent downbeat nystagmus and the right eye, one of lower amplitude. On gaze downward to the right, the nystagmus in the right eye virtually disappeared but remained in the left eye; the patient had a left INO. In addition to the dissociated downbeat nystagmus, the patient exhibited contralateral incyclorotatory nystagmus. These signs were interpreted as selective interruption of tonic fibers presumed to be carried in the medial longitudinal fasciculus.

Downbeat Nystagmus

An excellent review of 62 cases of downbeat nystagmus found that the slow-phase velocity was dependent on vertical head position and velocity in pitch.[40] The most common causes for downbeat nystagmus were found to be cerebellar ectopia and cerebellar degeneration; in 40% of the cases the cause was undiagnosed. The authors concluded that at least some cases of downbeat nystagmus

were due to an imbalance in otolith-ocular reflexes. They hypothesized the lesion to be in the vestibulocerebellum, perhaps the nodulus, since that structure normally inhibits otolith-ocular reflexes. The authors claimed that all patients exhibited slow phases that were linear, although, in the two figures shown, some appeared to have a slight increasing exponential shape. No vertical pursuit was evident in either direction.

An interesting case of downbeat nystagmus as a result of herpetic brain-stem encephalitis was reported in which pathologic examination of the brain stem revealed no lesions that would explain the nystagmus.[41] However, immunoperoxidase studies revealed virus-infected neurons throughout the brain stem. Downbeat nystagmus has also been reported with alcoholic cerebellar degeneration.[42] Oscillopsia and disturbances of balance were studied in patients with downbeat nystagmus.[43] The authors described a pathomechanism of ocular vertigo and oscillopsia with acquired ocular oscillations that postulated an inappropriate efference copy signal associated with these involuntary oscillations. The resulting oscillopsia causes impairment of visual stabilization of balance, since retinal image motion is a major cue for body stabilization. It was found that, in downbeat nystagmus, the postural sway with eyes open is dependent on gaze direction and it increases with increasing nystagmus amplitude.

Drug-Induced Nystagmus

A very thorough and well-referenced review of the nystagmus caused by various drugs has been written by Esser and Brandt.[44] The ocular oscillations are correlated to drug uptake or serum levels. Despite the many different drugs and induced oscillations, a pharmacologically induced disfunction of the vestibulocerebellar flocculus loop is postulated to be a common cause. Recently, a nicotine-induced upbeat nystagmus was described.[45] The nystagmus was dubbed ''bow tie'' nystagmus due to the downward slow phases interspaced with fast phases that were diagonally upward and alternating in direction; when traced on an XY plotter the bow tie figure results. Only a few puffs from a cigarette in either a smoker or nonsmoker causes the release of this upbeat nystagmus in the dark. Turning on the lights to allow fixation eliminates the nystagmus.

Epileptic Nystagmus

Epileptic nystagmus was first described by Féré in 1890.[46] However, one could not be sure of its directions, types, or slow phase waveforms until it was properly studied with accurate recording methods. Thurston et al. reported a case of epileptic gaze deviation and nystagmus.[47] This article contains the first eye movement

recordings of epileptic nystagmus that are of sufficiently good quality to expose the slow phase waveform. These authors found it to be a decreasing velocity exponential, similar to that of GEN. They suggested that the etiology of the nystagmus was due to a leaky brain-stem neural integrator, as had been previously hypothesized for GEN.[48]

Flash-Induced Nystagmus

Flash-induced nystagmus has been found more often in albino than in pigmented rabbits.[49] The nystagmus was not changed by fixing the stimulated eye, but lesioning of the nucleus of the optic tract on one side abolished flash-induced nystagmus and OKN responses from the other side. Bilateral labyrinthectomy caused diminished response and loss of after-responses. Flash-induced nystagmus was shown to algebraically combine with the vestibular reflex. The authors also discussed flash-induced after-nystagmus (FIAN) and after-after-nystagmus (FIAAN), which has been reported in the rabbit, cat, and monkey.

Intermittent Vertical Nystagmus

Intermittent vertical nystagmus may be an early sign of a Chiari malformation.[50] In this recently reported case, the downbeat nystagmus was episodic and accompanied with vertical oscillopsia.

Latent/Manifest Latent Nystagmus

A very interesting article by Bedell and Flom on the eye movements of strabismic amblyopes discusses in great detail the manifest latent nystagmus (MLN) exhibited by these patients without ever mentioning the terms latent nystagmus (LN) or MLN.[51] They described the unsteady fixation of these patients as consisting of nasal drifts alternating with temporal saccades; this is, by definition, MLN. They used the term ''fixation'' nystagmus in their table to describe these movements. Since MLN can be elicited in the dark without true fixation, MLN is not a fixation nystagmus, nor is CN. The authors found that the pursuit movements exhibited by these patients consisted of smooth pursuit plus the slow phases of MLN. They concluded that the centrally generated nasal drift bias was related to an impairment of spatial directionalization. These findings support the hypothesis that MLN is due to a defect in egocentric direction location.[52] Schor has suggested that subcortical binocular suppression affects the development of both LMLN and

OKN.[53] He presented a model describing how abnormal binocular interactions both in the cortex and midbrain might disturb the development of OKN and the control of eye movements in amblyopia and strabismus. The author concluded that the slow phases of MLN add to pursuit eye movements, the pursuit system is intact, since there is no slow buildup of OKN, and, since LMLN occurs in the dark, the source of the slow phase is extraretinal. Although it is commonly thought that the most likely motor disturbance accompanying strabismus is LN, the results of our recent study (discussed above) suggest that it is CN.

In a subsequent article, Schor and Westall investigated these extraretinal sources for fixation instability in strabismic amblyopes.[54] They found a high correlation between the dark drift bias and an imbalance in the VOR. The author studied the after-effects of nasalward and temporalward image motion on ocular drifts in the dark and introduced the term ''motion after nystagmus'' (MAN); there seems to be no real difference between this and optokinetic after-nystagmus (OKAN), and there is no need for another redundant term in the literature. Van Weerden and Houtman recently recorded a case of MLN supposedly of late onset.[55] According to the case history, the patient was noted to have LN in 1969 by clinical examination. At that time no eye movement recordings were made. In 1981 the patient was seen because of complaints of oscillopsia; recordings revealed a spontaneous nystagmus that reversed with alternate cover, thus her original LN had now become MLN. Surgical correction of her hypertropia reduced the MLN, but as Figure 2, shows it still could be present. Based on our experience with the incidence of pure LN, I would suggest that had a recording been made in 1969, a low-amplitude MLN would have been found. It is very rare to document pure LN with accurate eye movement recordings. Thus, her MLN may have been subclinical during the interim years and its increase in amplitude due to a worsening hypertropia made it appear as though it was MLN of late onset. Without documentation in the form of eye movement recordings (DC-coupled simultaneous recordings of both eyes rather than bitemporal recordings), one cannot presume that the MLN was not present all along albeit at a low amplitude. The explanations presented for LN in the discussion of this article, which contend that the slow phase starts as a pursuit movement and that an optokinetic stimulus then evokes a slow movement to the other side, cannot explain the occurrence of LN and MLN in darkness.

A case of unidirectional MLN in a patient with a congenitally blind eye (replaced by a prosthesis) was recently reported.[56] In the dark, the LN spontaneously reversed and the direction was dependent on the eye the patient was trying to ''see'' through. Thus, although never having sight through one eye, the intent to fixate still controlled the direction of the LN and the preferred eye was the one that was blind. This suggests that eye dominance is genetically predetermined and not influenced by visual development. This case also is strong evidence supporting the hypothesis that LMLN is related to egocentric rather than optokinetic asymmetries. In an article on right-left differences of nystagmus direction, Piper reported a case where operation of a congenital cataract converted a vertical nystag-

mus into a latent nystagmus.[57] He stated that in congenital squint syndromes monocular vertical nystagmus may be seen if one eye or both eyes are closed and postulated separate pathways from the vestibular system which may go only to the vertical-moving muscles of one eye. Spielmann recommended the use of translucent occluders to observe patients with MLN and CN.[58] She found it very useful clinically to separate MLN patients from CN patients to ensure proper treatment.[59] Using these occluders, Spielmann has observed the simultaneous appearance of MLN and its accompanying strabismus,[60] in agreement with our initial documentation of this phenomenon.[61]

Optokinetic Nystagmus

Optokinetic nystagmus and OKAN were first described by Purkyně.[62] This, and Purkyně's other contributions, can be found in an excellent review.[63] Recent studies have found that alertness (stimulated by sound and vibration) increased the means slow-phase velocity of OKN and also increased OKAN.[64] Alpha-rhythm was correlated with a decrease in the slow phases of OKN. In another study, age was found to cause a decrease in OKN velocity.[65] Also decreased by age were OKAN velocity and smooth pursuit velocity. Severe bilateral vestibular impairment resulted in a decrease in OKN gain and OKAN initial velocity and duration.[66] This study provided additional evidence for an optokinetic pathway in humans whose function varies with VOR gain. Two recent articles examined the relation between cortical binocular function and deficits in monocular OKN.[67, 68] Stereodeficient observers showed monocular OKN deficits (directional asymmetry or a reduction in both directions) in one or both eyes; the deficits were more pronounced at higher stimulation velocities and with smaller stimulus field sizes. The observers related the severity of the OKN deficit to the degree of residual foveal stereopsis and concluded that the effects of early developmental conditions on cortical and subcortical binocularity, responsible for the deficits of both, showed very close functional parallels. Another study concluded that there was no correlation between the monocular OKN asymmetries and stereoacuity.[69] The asymmetries were said to have their origin in binocular processes that are independent of stereopsis. The gain of the OKN system was found to decrease by occlusion of central retina at stimulus velocities above 30° per second.[70] The suppression of the high gain of the centrally driven OKN as well as the effect of stationary edges on the occlusion of the central retina are discussed in this and a subsequent article by the same authors.[71] They found that stationary edges not parallel to the direction of the stimulus motion exerted inhibitory influence on OKN, but only suppressed it when the images were on the fovea.

Hainline et al. studied small-field OKN in human infants and found that their horizontal OKN was of significantly higher frequency and lower amplitude than their vertical OKN.[72] While they found an asymmetry within their vertical OKN,

none was found in horizontal OKN. Also, there was no evidence of a buildup of slow-phase velocity over time. In general, infants had lower OKN gains and frequencies and larger slow-phase amplitudes than adults. It is interesting to note that Wallman and Velez found similar results with chicks when compared with grown chickens.[73] They found little directional asymmetry in the horizontal direction; however, OKN was better for upward rather than downward moving stimuli in chicks. Recently, a screening test for color blindness using OKN was described.[74] The authors varied the red-green luminosity ratios and used the OKN reaction as a measure of color acuity. This method might be applied to nonverbal subjects such as infants and animals.

Several investigators have recently studied the interactions between OKN and smooth pursuit. Van der Steen et al. found that sigma-pursuit was superior to pursuit of beta- or real motion; the gain was higher, the saccadic rate lower and the detrimental effect of a structured background smaller.[75] Yee et al. found that the affects on smooth pursuit of OKN backgrounds was greatest in subjects with lower pursuit gains while tracking against blank screens.[76] Their experimental observations could not be explained by simple algebraic summation of independently induced pursuit in OKN eye movements. Barnes and Crombie found pursuit gain significantly less when subjects pursued a sinusoidally oscillating target against a stationary structured background than when the background was blank.[77] These authors concluded that the human observer, when presented with several moving targets, can selectively enhance feedback gain from one particular source in order to dominate the stimuli from the other sources. Kowler et al. concluded that the effectiveness of voluntary selection in eliminating the influence of background stimuli on smooth eye movements can be virtually complete.[78] The attention and effort required to do so, however, may impair the accuracy of psychophysical judgments made about that background. These authors ruled out both location and perceived motion as the characteristics of voluntary selection; they concluded that the perception of a target as a distinct perceptual configuration was keyed to successfully distinguishing it from the background.

Using apparent motion, Schor et al. studied OKN.[79] Their results suggested that pursuit can occur as a response to apparent motion generated by both small and large image displacements, while OKN and vection were responses to apparent motion generated by small image displacement only. They concluded that different afferent sources are utilized with the control of pursuit and of the slow phase of OKN. Barratt et al. studied patients with various neurological diseases and compared their pursuit and OKN responses.[80] They found, in a patient in whom pursuit was intact, a severely impaired OKN response and in another patient, in whom the OKN responses were intact, the presence of severely deranged pursuit. These dissociations suggested that pursuit and immediate onset OKN responses are mediated by separate mechanisms. The third patient, who had virtually no pursuit or passive OKN, showed high slow-phase velocities of active OKN; this demonstrated that the active form of OKN is more than a linear addition of pursuit and passive OKN responses. In eight patients with unilateral acute or chronic

lesions of parietooccipital lobes, it was found that ipsilateral pursuit was more disturbed than OKN responses.[81] Wyatt and Pola studied the optokinetic responses to oscillating fields of dots and the suppression of these responses by a foveally stabilized target.[82] Such a target provided neither retinal target motion nor offset from the fovea, yet suppression was possible. The authors concluded that suppression of OKN is accomplished by means other than by smooth pursuit eye movements.

Maurer et al. found, in children treated for unilateral congenital cataract, that OKN occurred significantly more often with nasally moving stripes than when they moved temporally.[83] No asymmetry was observed in any of 13 children treated for traumatic cataracts incurred after 3 years of age. Kömpf found significant OKN asymmetry in patients with hemispheric lesions.[84] Such patients show contralateral deficits in OKN, and monocular stimulation can reveal further OKN deficits in some patients. Lesion studies in animals have shown that unilateral floccular lesions cause a deficit in the OKR, but not in the VOR.[85] Barmack and Pettorossi concluded that the flocculus contributes to low-velocity eye movements through the inhibitory modulation of the activity of the subadjacent vestibular nuclei. Büttner et al. found that, despite central retinal lesions in monkeys, fast OKN responses could still be obtained.[86] Thus, fast OKN responses can be obtained from extrafoveal areas that are not generally involved in smooth pursuit. Despite this finding, the authors concluded that there could still be common premotor structures shared by the OKN and smooth pursuit systems. Lynch and McLaren showed that unilateral lesions of the inferior parietal lobule and prestriate cortex caused diminished slow phases of OKN.[87] These deficits were greater at higher rates of OKN stimulation and are similar to those that are commonly associated with posterior parietal damage in humans.

Optokinetic After-Nystagmus

Several recent articles examined the characteristics of OKAN.[88–90] Mean eye displacement was less for OKAN than for OKN, slow phase duration was greater for OKAN, and eye displacement per slow phase remained fairly constant during OKAN. The OKAN decay was found to be a two-component process that can be closely approximated by the sum of two exponentials with time constants of 1.15 seconds and 48.8 seconds. The OKAN decay commenced at a time after the lights were turned out, which was dependent on the timing of an intervening fast phase. Repeated exposure to OKAN stimulation produced significant changes in the response characteristics. Decrements were seen in cumulative displacement, short and long time constants, and the coefficient of the long time constant component. These changes were still present after one week, and up to eight weeks after testing. The OKAN characteristics were also dependent on the OKN stimulus velocities. Two distinct types of response were found, a low-level and high-level

response. These responses were related to the direct (pursuit) and indirect (nonpursuit) pathways respectively. It has been reported that symmetrical OKAN loss can result from Wallenberg's syndrome and multiple sclerosis.[91] The authors concluded that bilaterally absent OKN in the presence of retained caloric responses indicates a brain-stem rather than a peripheral site for the lesion. In an interesting study, Schor et al. found that infants below the age of 4 to 5 months exhibited an OKAN with a nasalward slow phase regardless of the direction of the OKN stimulus. It was not until after this age that temporalward OKN stimuli would evoke a similar OKAN.[92] The authors related this to the delayed development of reflex following eye movements in the temporalward direction.

Periodic/Aperiodic Alternating Nystagmus

Periodic alternating nystagmus has been recently reported associated with periodic alternating skew deviation in a patient with cerebellar degeneration.[93] Treatment with various drugs had no affect on either the PAN or the skew deviation. Another article described PAN that cleared after cataract surgery.[94] The PAN developed in association with decreased vision due to cataracts. It disappeared on the first postoperative day when both eyes were open. Unfortunately, the tracings shown in this article are the result of low bandwidth, alternating current, bitemporal electrodes. Therefore, the statement that ''occasionally there is an increasing slow phase velocity'' is not deducible from the data. Such recordings cannot show the motion of either eye, let alone whether or not the slow phases are increasing, decreasing, or linear. That statement is an illustration of the dangers inherent in poor recording techniques.

Physiological Nystagmus

Hess et al. recently studied normal eye drift and the resulting endpoint nystagmus in darkness.[95] These authors confirmed the findings of Abel et al. that physiological end-point nystagmus can occur at gaze angles as little as 20°. Also confirmed were the conclusions of Becker and Klein that both gaze dependent (centripetal) and constant (unidirectional) eye drifts are found in the dark.[96]

Rebound Nystagmus

The recent literature has provided clinicopathological correlation for rebound nystagmus in a patient with olivocerebellar atrophy.[97] Degeneration was found of

the inferior olivary nuclei and the cerebellar cortex, leading the authors to speculate that rebound nystagmus can be a sign of involvement confined to the olivocerebellar circuit and that extraretinal signals of eye position are monitored independently of this system. The slow phases of the rebound nystagmus shown appear to be linear. Rebound nystagmus was also studied in five normal subjects in whom the nystagmus was elicited after varying degrees and durations of eccentric fixation of the target light.[98] Their results led them to conclude that a velocity bias alone could not account for rebound nystagmus. The authors hypothesized that the null shift in the direction of prior eccentric gaze was caused by two processes: development of velocity bias, which causes the null to move, and a decrease in the time constant of the neural integrator, which determines the exact location of the null. Since the time courses of the decay of the bias and of the recovery of the time constant were different, the two components of rebound nystagmus may reflect separate neural mechanisms.

See-Saw Nystagmus

See-saw nystagmus was reported in a 7-year-old child as a result of surgical correction of a divergence excess type exotropia.[99] The patient was able to utilize convergence to damp the see-saw nystagmus and base-out prisms were prescribed. The author speculated that, prior to surgery, the patient was using convergence to overcome her exodeviation and thereby, was preventing the nystagmus. Since the surgery consisted of an overcorrection, the need for convergence was obviated and the patient rapidly developed see-saw nystagmus.

Stepping Around Nystagmus

Somatosensory information during locomotion contributes to the sense of self-motion and induces nystagmus that has been called stepping around nystagmus.[100] In the laboratory both real stepping around and apparent stepping around will cause nystagmus. Real stepping around nystagmus is due to somatosensory and vestibular effects and apparent stepping around nystagmus is due to only the former. In patients with loss of labyrinthine function, both real and apparent stepping around resulted in motion sensation and nystagmus.[101] Both conditions induce self-motion and Coriolis effects.[102] Somatosensory-vestibular interactions have been compared with visual-vestibular interactions and direct and indirect somatosensory pathways to the common velocity-storage mechanism hypothesised.

Torsional Nystagmus

Two patients with amyotrophic lateral sclerosis, proved postmortem, were reported to have torsional nystagmus in addition to their typical clinical signs.[103] Both patients had gaze-evoked torsional nystagmus; one of the patients also had horizontal nystagmus in the primary position. This is the first report of nystagmus in postmortem-verified ALS.

Vertical Nystagmus

A recent study of six members in a family representing three successive generations reported primary position vertical nystagmus in all patients.[104] The family members had hereditary cerebellar ataxia. The four members of the family who were tested showed horizontal gaze–paretic nystagmus, three showed rebound nystagmus, and their truncal ataxias varied from minimal to severe. The three cases of vertical nystagmus that were recorded were upbeating and pendular in one patient, primarily upbeating with rare pendular components in another, and mostly pendular in the third. The pursuit records, both horizontal and vertical, show what one would expect in the presence of spontaneous nystagmus and for reasons that have been elucidated elsewhere in this chapter I would disagree that they prove defective or absent pursuit.

Vestibular Nystagmus

The nystagmus produced by otolith stimulation (linear acceleration) has been designated L-nystagmus.[105] This nystagmus is independent of any rotation of the gravitational vector and consists of slow phases opposite to the acceleration interrupted periodically by fast phases in the direction of acceleration. Hain et al. studied head-shaking nystagmus in six subjects with unilateral peripheral vestibular lesions.[106] Head-shaking nystagmus has two phases, HSN1 and HSN2. The HSN1 phase is a strong nystagmus lasting five to 15 seconds with the fast phases beating away from the side of the lesion, and HSN2 is a lower amplitude reversal phase. The authors were able to simulate HSN using a model that incorporated Ewald's second law. Robinson et al. have suggested a hypothesis for Alexander's law[107] that differs from that originally proposed by Doslak et al.[108] They proposed that in patients with a vestibular lesion, the phenomenon of Alexander's law is due to the sum of vestibular nystagmus and an abnormally large gaze-evoked nystagmus that is consequent to the lesion. Doslak et al. had proposed that a copy of intended eye position was added to the integrator input, which would create

linear slow phases; this new hypothesis predicts exponential slow phases. Unfortunately, due to the numbers involved, it is very difficult to differentiate between the two on eye movement tracings. An important finding of this article was that normal subjects can alter the time constant of their neural integrators by intent. Another difference between the two hypotheses concerns whether or not the rate of change of slow phase velocity is constant or changes with nystagmus intensity. The present authors support the latter, whereas the Doslak et al. data suggest that for steady-stage lesions, it is a constant independent of nystagmus intensity.[109] Other differences between the transient model of these authors and the steady-state model of Doslak et al. are easily resolved. This is an interesting article with an attractive hypothesis that deserves further study.

A recent series of articles has championed the cause for bilateral models of ocular motor control.[110–113] These articles, and the models they contain, have particular appeal for this author, who has always been both partial to modeling the system as it exists in nature and wary of making the assumptions necessary to collapse such models into unilateral lumped linear systems. Also, it was refreshing and educational to have to review signal flow graphs and Mason's gain formula to read these works. Thus, only those whose background in control systems is adequate should attempt this task. To those who do, the results will be rewarding; the models are novel in that they suggest that the commissural pathways that connect the two-sided models are actually important in determining both the static and dynamic gains and time constants of the system. Phenomena such as Alexander's law, vestibular compensation, adaptive change, and visual-vestibular interactions are examined from this viewpoint. The complexities of these models preclude their analysis in this chapter, however, they are recommended reading for the interested and well-prepared student of the VOR. For those without an extensive modeling background, some interesting theoretical and practical thoughts on vestibular asymmetry and vestibular compensation are contained in a paper by McClure and Lycett.[114]

A caloric nystagmus was elicited in microgravity during the flight of Spacelab I.[115] This nystagmus was contrary to the prediction of the classical endolymph flow theory put forth by Bárány. Studies stimulated by these results have found that the normal caloric response is the sum of a convection current component, a smaller position-dependent component of unclear origin, and a direct temperature effect on the canals' sensory apparatus.[116] Possible mechanisms include a direct volume displacement[117] and changes in endolymphatic density.[118] Under normal conditions, Bárány's theory continues to be valid, since convection forms a greater proportion of the effective total stimulus than expansion.[119]

Barnes found that VOR suppression is dependent on the location of the visual target.[120] Suppression is greatest when fixating a central target and decreases as the target becomes more peripheral. He found that even with central fixation there was incomplete cancellation of the VOR. Curio and Grüsser did find complete suppression of the VOR within the limits of their EOG recordings.[121] They also found suppression stronger with sigma-OKN than during fixation. Waespe et al.

examined the role of the flocculus and paraflocculus in OKN and visual-vestibular interaction.[122] Flocculectomy prevented the suppression of the initial jump in eye velocity at the onset of the velocity step. However, the subsequent nystagmus was suppressed. They concluded that the monkeys retained their ability to discharge activity from the velocity-storage mechanism. The OKAN was also suppressed after flocculectomy. A model was presented that contained a common velocity-storage integrator used by both the visual and vestibular systems. A dump mechanism shortened the time constant of the integrator when eye velocity exceeded surround velocity. The authors also concluded that the flocculus was not important in cancellation of the horizontal VOR or in affecting the dynamics of vestibular nystagmus, OKAN, or the velocity-storage mechanism. Demer and Robinson presented a model that demonstrated how a single, common, velocity-storage element that both generated OKAN and prolonged postrotary nystagmus could still be the source of the different time constants that can be present in the optokinetic and vestibular systems.[123] In another study, using a model of visual-vestibular interaction, Davidson et al. concluded that fixation suppression is mediated almost entirely by the pursuit system.[124] They used the model to investigate the possible contributions of pursuit and the optokinetic system. In their study of the velocity-storage mechanism, Matsuo and Cohen found an asymmetry in vertical velocity storage.[125] They found vertical asymmetry in OKN, OKAN, and in vestibular nystagmus suppression; downward OKN and vestibular nystagmus were not suppressed as well as upward nystagmus.

Leigh et al. studied the VOR of unresponsive patients using head rotation.[126] They compared their responses to a position-step of head rotation with those of normal subjects. Normal subjects exhibited an oppositely directed eye movement followed by an exponential drift back to the midline. The time constant of the drift was greater than or equal to 10 seconds for normals, 1.5 seconds for unconscious patients, and less than 0.5 seconds for patients in a vegetative state. Dysfunction of reticular and cerebellar connections were implicated in these shortened time constants. Wennmo et al. studied the visual-vestibulo-ocular response (VVOR) in patients with pontine, medullary, cerebellar, and combined cerebello-brain-stem disorders.[127] They found dissociations between pursuit and OKN slow phases (more pronounced in medullary disease) and enhanced VOR but normal VVOR, in cerebellar patients. Studies of patients with bilateral labyrinthine lesions showed intact cervicoocular reflexes but abolished VOR.[128] Buizza and Schmid modeled VVOR interaction and discussed parametric changes to describe labyrinthine- and cerebellar-defective patients.[129] They discussed the model's role in assessing the state of the system and the validity and significance of test procedures.

Tychsen et al. studied the VOR and infantile strabismus.[130] They found intact VOR in both infantile and noninfantile strabismus, but an inability to enhance or cancel the VOR in the former group. This was attributed to a deficit in temporally directed pursuit that exceeded the ever-present nasal slow phases of LN present in this group. The authors concluded that such patients are unable to process retinal

error-velocity signals in the foveopetal direction on the temporal hemiretina. Sherman and Keller studied the VOR in adventitiously blind, congenitally blind, and normal subjects.[131] They found no VOR in the congenitally blind and lower gain than normal in the adventitiously blind. They concluded that vision was necessary for VOR development and maintenance. Fetter et al. studied the role of vision in compensation for the bias (spontaneous nystagmus) and dynamic disturbances (directional asymmetry and decreased gain) resulting from unilateral labyrinthectomy.[132] They compared the results of unilateral labyrinthectomy on three normal and three cortically blind monkeys. The rate of recovery of the spontaneous nystagmus was variable, but the VOR gains did return to normal in the normal monkeys (exposed to light) but not in the blind monkeys. The authors concluded that the occipital cortex is essential for gain corrections, but not for bias allevaition.

Black and Nashner found distinct differences in postural instabilities between patients with benign paroxysmal positional vertigo (BPPV) and those with other vestibular deficits.[133] The BPPV patients employed an unstable, visually dependent postural sway. In contrast, the vertical VOR of BPPV patients was found to be normal.[134] An extensive review of vertigo as a symptom was written by Oosterveld.[135]

OTHER OCULAR MOTOR OSCILLATIONS AND INTRUSIONS

Non-nystagmic ocular motor oscillations and intrusions represent solely saccadic or saccadically initiated instabilities. I have identified 16 varieties of saccadic oscillations and intrusions that have been characterized in the literature by approximately 50 different terms, including ten which erroneously contain the term ''nystagmus.'' This section contains discussions of recent studies of nine types of saccadic oscillations and intrusions from the 16 originally identified in Table 2; they are in alphabetical order. For detailed definitions and discussions of these and other types of saccadic intrusions and oscillations, see previous volumes on the subject[1–4] and a recent review article.[136]

Bobbing

A recent study of five patients with ocular bobbing and ''locked-in'' syndrome found that upwards voluntary eye movements were abnormal.[137] The authors suggested that, rather than considering bobbing and abnormal eye movement, it be considered the result of the abnormality of eye movements in other directions. Since these patients had pontine lesions that affected only upward movements, the authors hypothesized that downward movements are generated by mesencephalic structures, but that upward movements are under pontine control A recent case

with typical bobbing has been reported where the lesion was in the most dorsal median portion of the pontine tegmentum.[138] The lesion was caused by a very small hemorrhage that was confirmed pathologically to have resulted in very limited primary bleeding. This patient also had intermittent reverse bobbing.

Flutter

Hainline et al. recently studied the saccades in human infants.[139] They found that infants (14 to 151 days old) exhibited saccadic oscillations that had no intersaccadic latency. This oscillation, which is indistinguishable from flutter, was related to attentional factors. The flutter usually occurred in episodic bursts of two to three cycles rather than a continuous oscillation. Also found were multiple, closely spaced saccades. The authors noted the similarities between the flutter observed and the voluntary ''nystagmus'' that adults can display. They suggest that the so-called opsoclonus previously reported in infants may be the normal, occasional phenomenon of flutter in young infants and unrelated to brain damage. They concluded that their observations were compatible with a mature infant saccadic generator, but one that shows variability due to fluctuations in overall arousal.

Flutter Dysmetria

A patient with multiple sclerosis has been reported who exhibited both a pendular nystagmus at 4.5 Hz and the combination of flutter and flutter dysmetria.[140] The flutter dysmetria was more pronounced with rightward saccades than leftward, but was present with both. The author suggested that the oscillation recorded was different from flutter dysmetria and should be called ''macroflutter,'' but this was unconvincing. Patients with multiple sclerosis may exhibit flutter dysmetria along with dysmetria and square wave jerks.[141] The occurrence of flutter or flutter dysmetria associated with caloric nystagmus was correlated to prediction of the outcome of patients admitted in a comatose state.[142] The authors stated that the poor outcome of ten patients who exhibited these oscillations associated with the quick phases of caloric nystagmus could be indirectly related to the pathophysiology of the oscillations. Unfortunately, the recordings shown are from bitemporal, accoupled EOG of low bandwidth and no quantitative calibration of eye movements in degrees was shown. Therefore, nothing can be said about the movements of either eye and the possible dissociation between the eyes; this despite the disclaimer that visual inspection was used to detect dissociated eye movements. Such claims simply do not hold up when accurate recordings of both eyes are made.

A new form of flutter dysmetria induced by blinks has recently been reported.[143] Blinks resulted in saccades with large dynamic overshoots in a patient with cerebellar degeneration. The authors explained their findings with the use of a model of the saccadic pulse generator that was sensitive to a blink-related neural signal at the level of the pause cell. The model predicted blink-induced flutter in normal subjects, which was later found.

Macro Saccadic Oscillations

A recent longitudinal study of a patient who initially had macro saccadic oscillations (MSO) and one year later showed saccadic dysmetria, suggests that both are due to elevations in the gain of the corrective mechanism associated with saccades.[144] The authors noted that the patient made undershoots for large target displacements and overshoots for small target displacements with relatively the same percentage as normal subjects. From this they concluded that the main saccadic system was normal and a miscalculation of the corrective saccade was responsible for the dysmetria. A review article by Zee relates dysmetria and MSO to various defects in the cerebellar control of eye movements.[145] In addition to the saccadic disabilities, the author discusses gaze-evoked nystagmus, rebound nystagmus, and oscillopsia.

Macro Square-Wave Jerks (Bursts/Single)

An interesting case of sustained blepharoclonus associated with bursts of macro square-wave jerks (MSWJ) have recently been reported.[146] With eyes open, the patient exhibited long periods of MSWJ. Voluntary eye closure resulted in rhythmical contractions of both obicularis oculi, which were synchronous with the MSWJ. The patient exhibited the cerebellar signs of dysmetria and dysfunction of speech as well as motor control of the trunk and limbs. The authors suggested that the same lesion in the cerebellar system was responsible for both the blepharoclonus and the MSWJ.

Opsoclonus

A case has recently been reported where opsoclonus has been associated with hyperosmolar nonketotic coma.[147] This is the first case of opsoclonus in a metabolic disorder. The opsoclonus was described as mainly in the horizontal

plane, but with rotary and vertical components; no recordings were made. Pathology revealed an adenocarcinoma in the tail of the pancreas with multiple metastasis to the liver; no structural lesions were found in the brain on both macroscopic and microscopic examination. The authors speculated on some possible metabolic causes for opsoclonus in the absence of brain-stem or cerebellar pathology.

Saccadic Lateropulsion

A new observation is lateropulsion away from the side of the lesion (contrapulsion) that has been reported by Ranalli and Sharpe.[148] They described a patient with a unilateral disorder of the rostral cerebellum who exhibited ipsilateral limb ataxia and saccadic contrapulsion of vertical saccades. Also, contralateral saccades were hypermetric and ipsilateral saccades, hypometric; this is equivalent to the findings in patients with ipsipulsion. The authors attributed the contrapulsion to imbalanced cerebellar outflow. Saccadic lateropulsion has recently been reported in a case of right juxtapontobulbar cerebellar infarct involving the flocculo-nodular lobe.[149] Given the above findings of contrapulsion with a cerebellar lesion, it is unfortunate that no eye movement recordings were presented; the authors reported that there were no vertical, cerebellar, or ocular motor symptoms. The patient showed an isolated axial lateropulsion *toward* the affected side. The authors discussed possible mechanisms of this previously unreported syndrome.

Saccadic Pulses/Pulse Trains

An article on saccadic intrusions in INO has provided additional evidence that the so-called abduction ''nystagmus'' of INO is, in fact, a saccadic pulse train (SPT).[150] The authors recorded patients who had spontaneous saccadic pulses (SP) of abduction in one or the other eye while fixating straight ahead. These patients had bilateral INO; three had multiple sclerosis and one had a brain-stem infarction. The sporadic SPT were recorded in either eye, but the initiating saccades were always in the abducting direction. The presence of these saccadic oscillations in patients with INO supports the hypothesis that in lateral gaze, the abducting eye is actually exhibiting SPT while the adducting eye is attempting to reach the target via hypometric saccades. The authors went on to suggest that these SPT are due to something extrinsic to the medial longitudinal fasciculus. Strongly supportive of this latter hypothesis is a recent article on pseudointernuclear ophthalmoplegia induced by surgical paresis of the medial rectus muscle.[151] After surgically weakening the medial rectus muscle in five patients, SPT were recorded that were indistinguishable from the abduction ''nystagmus'' of INO. Patching experiments

clearly revealed the SP to be due to increased gain in the abducting eye in an effort to overcome the limitations placed on the adducting eye. Thus, whether a central or peripheral deficit causes limitation in adduction, the SP that result are due to increased gain in that direction; as a result, the abducting eye overshoots the target. A recent article found that slowing of the adducting saccade was the most sensitive indicator of INO, with the dissociated abduction ''nystagmus'' being another good indicator.[152] In their 21 patients, limitation of the adducting eye was seen much less frequently.

Safran et al. described two patients with dorso-mesencephalic lesions who showed adduction overshoot and abduction undershoot and instability of ocular fixation.[153] Their tracings seem to indicate that the instability of fixation as well as the adduction overshoot consist of SP in the adducting direction. Thus, we have the same occurrence in adduction that is normally found in abduction in patients with an INO. This again supports the hypothesis that an increase in gain in the direction of weakness is the underlying cause for the occurrence of SP and SPT.

Square-Wave Jerks/Oscillations

It is well known that square-wave jerks (SWJ) are a very nonspecific saccadic eye sign. A recent article reviewed the literature on SWJ and reached the same conclusion.[154] Square-wave jerks have been found in Wernicke-Korsakoff's syndrome,[155] Parkinson's disease,[156] and Friedrich's ataxia.[157] Square-wave jerks, unfortunately identified merely as saccadic intrusions, have been reported in cases of reading disability.[158, 159] Some of these patients appeared to have square-wave oscillations (SWO) in addition to SWJ. It was refreshing to read a letter to the editor that took exception to the practice of using the general term ''saccadic intrusions'' when in fact the authors were specifically referring to SWJ.[160] The paper in question dealt with schizophrenia and the common occurrence of SWJ in schizophrenics that has been confused with a pursuit defect. The author of the letter correctly pointed out that what was shown were SWJ and that it would certainly simplify nomenclature of eye-movement disorders if they were properly identified as such; it might also link schizophrenia to a larger cluster of neurologic disorders in which SWJ appear. While it is true that the speed of the chart recorder can reduce the ''squareness'' of SWJ, at most normally used chart speeds, they do appear square. Despite this curious objection, the term is well known and accepted in the literature and the equating of the more generic term ''saccadic intrusion'' with SWJ can only confuse the reader. One then must search the paper to identify which of the many saccadic intrusions the authors are talking about; invariably it is SWJ. This type of thinking would justify using the word ''nystagmus'' to describe any or all of the 43 different types of nystagmus; while this would be technically correct, it would hardly be useful.

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