
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 School of Medicine, 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 these determinations possible and thereby clarified the underlying ocular motor mechanisms responsible for many oscillations. Table 1 lists 47 types of nystagmus (two of which were not in the previous volumes) along with many other terms found in the literature to describe them; similarly, Table 2 lists 16 saccadic oscillations and intrusions (one of which has been renamed since the previous volume) with other descriptive terms. The tables evolved from those that appeared in previous biannual volumes on this subject.¹⁻⁵

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, some eye movements originally described with the word "nystagmus," are really saccadic oscillations. Most oscilla-

TABLE 1.
Forty-seven Types of Nystagmus*

Acquired "fixation"	Flash-induced	Pursuit after- induced
Anticipatory induced	flicker-induced induced	Pursuit-defect†
Arthrokinetic induced	Gaze-evoked deviational	Pseudospontaneous induced
somatosensory	gaze-aretic	Rebound
Associated induced	"neurasthenic"	Reflex
Stransky's	"seducible"	Baer's
Audiokinetic induced	"setting-in"	See-Saw
Bartel's induced	Horizontal	Somatosensory induced
Bruns'	Induced	Spontaneous
Centripetal	provoked	Stepping around apparent/real induced
Cervical	Intermittent vertical	somatosensory
neck torsion	Jerk	Torsional
vertebral-basilar artery insufficiency	Latent/Manifest latent monocular "fixation"	rotary
Circular/Elliptic/Oblique alternating windmill	unimacular	Unioocular
circumduction	Lateral medullary	Upbeat
diagonal	Lid	Vertical
elliptic	Miner's†	Vestibular
gyratory	occupational	a(po)geotropic/geotropic
oblique	Muscle-aretic	alternating current
radiary	myasthenic	Bechterew's
Congenital "fixation"	Optokinetic	caloric/caloric-after compensatory
hereditary	induced	electrical/faradic/galvanic
Convergence	"kinetic"	head-shaking
Convergence-evoked	"optic"	induced
Dissociated	optomotor	L-
disjunctive	panoramic	labyrinthine
Downbeat	"railway"	perverted
Drug-induced	sigma	pneumatic/compression
barbiturate	"train"	positional/alcohol
bow tie	Optokinetic after- induced	positioning
induced	post-optokinetic	postrotational
Epileptic ictal	reverse post-optokinetic	pseudocaloric
	Pendular	rotational/perrotary
	talantropia	secondary phase
	Periodic/Aperiodic alternating	
	alternans	
	Physiologic	
	end-point	
	fatigue	

*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.

TABLE 2.
Sixteen Types of Saccadic Intrusions and Oscillations*

Bobbing/Dipping	Saccadic lateropulsion
inverse bobbing	ipsipulsion
reverse bobbing	contrapulsion
Convergence-retraction "nystagmus"	Saccadic pulses/pulse trains
"nystagmus" retractoris	abduction "nystagmus"
Double saccadic pulses	ataxic "nystagmus"
(single/multiple) →	saccadic intrusions/oscillations
saccadic intrusions/oscillations	stepless saccades
Dynamic overshoot	Square-wave jerks/oscillations
"quiver"	Gegenrucke
Dysmetria	hopping nystagmus
Flutter	"lightning eye movements"
microflutter	myoclonus
Flutter dysmetria	saccadic intrusions/oscillations
Macro saccadic oscillations	Zickzackbewegungen
Myoclonus	Square-wave pulses (single/bursts) →
laryngeal "nystagmus"	Kippdeviationen/"Kippnystagmus"
"lightning eye movements"	"macro square-wave jerks"
pharyngeal "nystagmus"	"pendular macro-oscillations"
Opsoclonus	saccadic "nystagmus"
"dancing eyes"	saccadic oscillations/intrusions
"lightning eye movements"	Superior oblique myokymia
saccadomania	
Psychogenic flutter	
hysterical flutter	
hysterical "nystagmus"	
"ocular fibrillation"	
"ocular shuddering"	
psychological "nystagmus"	
voluntary flutter	
voluntary "nystagmus"	

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

tions were named before the benefits of accurate recordings. Quotation marks are used for those oscillations 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. Since these ambiguous or erroneous terms do not convey accurate information about the basic nature of the movement, their use is not recommended in scientific communications. 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 non-nystagmic oscillations (i.e., saccadic). The best names describe 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. The use of amplitude-related terms has

proven to be misleading when later work has uncovered the same oscillation at a different amplitude; terms like “micro,” “mini” or “macro” do not imply a different mechanism and should not be used in the primary name of an oscillation (see the suggestion to substitute “square-wave pulses” for the misleading “macro square-wave jerks”). This does not preclude their use in describing a specific case where the oscillation may be smaller or larger than is normally seen. There is a great deal of idiosyncratic difference in ocular motor oscillations, and low-amplitude oscillations seen in “healthy” subjects may actually be preclinical signs of later, larger oscillations.

NYSTAGMUS

The word “nystagmus” is derived from the Greek word νησταγμος meaning drowsiness, which in turn is derived from νησταξειν meaning to nod in one’s sleep. It should be noted that this nodding oscillation is generated and sustained by the slow downward drifting of the head; the upward head jerks are corrective (i.e., they serve to restore upright head posture). In keeping with this original definition, nystagmus is defined as follows: a biphasic ocular oscillation containing slow eye movements (SEM) 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 approximately equal in amplitude. It was once suggested that the term for pendular oscillations should be “talantropia” (from the Greek word ταλαντροπια for oscillating) to distinguish it from jerk nystagmus,⁶ but this terminology did not become popular. This section contains discussions of each nystagmus type in which significant work has been done in the past 2 years; they are in alphabetical order. For more detailed descriptions of other types, the reader is referred to the aforementioned previous volumes.

A study of the effects of sampling frequency, analog filtering, and A/D conversion bits on the accuracy of velocity determinations of fast and slow phases of nystagmus concluded that 400 Hz, 70 Hz, and 12 bits were the respective requirements of these variables.⁷ This is a complicated issue and there are problems associated with over-sampling as well as under-sampling that are not immediately obvious. It has been our experience that a 200-Hz sampling frequency, 100-Hz analog filtering and 12 bit A/D conversion is more than adequate for slow phase velocities; in fact, many times we cut the differentiator bandwidth to 21 to 24 Hz with no difference in slow phase velocities. For saccadic velocities, the higher sampling rate is required. Given the usual need to limit the size of computer data arrays per trial, it is probably better to sample the position signal at 200 Hz to include all of the frequencies of interest and later interpolate the data arrays to effectively double that sampling rate before differentiating.

Anticipatory Nystagmus

Anticipatory SEM are predictive movements that occur prior to either ramp or step target motions. When these movements are interspaced with saccades in the opposite direction, anticipatory nystagmus results.⁸ Boman and Hotson recently showed that with a continuously visible target, prior to the initiation of ramp motion, an anticipatory nystagmus was produced that restricted eye position and velocity errors. However, prior to ramp termination, this nystagmus was not produced and slow eye velocities changed by as much as 6 degrees/sec.

Circular/Elliptic/Oblique Nystagmus

Traccis et al. reported the successful treatment of an acquired pendular elliptical nystagmus in multiple sclerosis using isoniazid and base-out prisms.⁹ The drug abolished the nystagmus and relieved oscillopsia (OSOP) in two patients but was ineffective in the third in whom the base-out prisms were used. By careful questioning of these three patients, it was discovered that the OSOP associated with circular-elliptic nystagmus, unlike that of horizontal, vertical, diagonal, or torsional nystagmus, is in the *same* direction as the eye motion. Comparison was made to linear nystagmus where the retinal image motion is the same as eye motion and the perceived motion is opposite to the retinal image motion and to torsional nystagmus where the retinal image motion is in the opposite direction to eye motion and the perceived motion is in the same direction as the retinal image motion. In circular or elliptic nystagmus, both the retinal image and perceived motion are in the same direction as the eye motion. Therefore, a clockwise nystagmus (as seen from the *subject's* point of view) produces a clockwise OSOP and vice versa.

Congenital Nystagmus

We studied the accuracy and cycle-to-cycle repeatability of the foveation periods in congenital nystagmus (CN) since visual acuity is directly related to these indices.¹⁰ During a 5-second interval of fixation, the standard deviation of mean horizontal foveal position was ± 13 minarc (± 5 minarc vertical) and mean foveation time was 59 ms. There were 1-second intervals of fixation with standard deviations of 0 minarc. Position and velocity histograms reflected the increase in data about the zero position and velocity points caused by the foveation periods of the waveforms. We applied phase-plane analysis to the CN waveforms and demonstrated beat-to-beat overlap in the position and velocities during the foveation

periods. Contrary to the notion that CN was due to poor fixation reflexes, we concluded that the fixation reflexes in subjects with CN were remarkably strong and accurate despite the large oscillation always present. Even in albinos, where the fovea is not normal, CN foveation periods were found to approximate those of normal foveas.¹¹ In a study of various indices of CN waveforms, the highest correlations were found to be between: (1) foveation time and the maximum rate of the histogram indicating the rate of duration of the eye in each spatial position; (2) amplitude and intensity; and (3) mean slow-phase velocity and intensity.¹² Visual acuity correlated best with foveation time. Bedell et al. found foveation-period variations similar to ours (13 to 67 minarc horizontally and 8 to 20 minarc vertically).¹³ They attributed the vertical variation to crosstalk from the horizontal CN. Based on their finding of a correlation between the variations in the horizontal and vertical meridians in both idiopaths and albinos, they correctly concluded that CN is a *motor* and not a *sensory* disorder in *both* populations (see previous volumes for discussions of this issue). They further concluded that subjects with nystagmus might also exhibit *normal* ocular motor behavior under certain conditions; smooth pursuit or the vestibuloocular reflex (VOR) perhaps?

The accuracy, repeatability, and duration of the foveation periods are the most critical features of CN waveforms' effect on visual acuity. As a result of our studies of the dynamics of the foveation periods in CN, I developed a nystagmus foveation function (NFF) that should function as a sensitive indicator of the gaze angle of best acuity in an individual and a means of correlating waveform characteristics and acuity between subjects, something that CN intensity cannot do. This function is formed by the quotient of the product of foveation period per cycle and CN frequency in the numerator and the product of the standard deviations of the mean foveation-period position and velocity in the denominator. Preliminary data for one subject have demonstrated the direct relation between the value of the NFF and the gaze and convergence angles of best acuity. For a discussion of the importance of the foveation periods and the suppression of OSOP, see the section on oscillopsia in this chapter.

Abadi and Worfolk studied the relationship between visual acuity and the duration of low velocity in CN slow phases.¹⁴ They found a significant correlation between the duration of slow-phase velocities below 10 degree/sec and acuity. Although this was a somewhat cruder measure of the foveation periods, it does illustrate their importance in acuity. This paper contains velocity histograms of various waveforms and their effect on good foveation. Other researchers also found a correlation between acuity and foveation-period duration¹⁵ or variability.¹³ As expected, this latter study did not find a correlation in albinos, whose acuity is limited by afferent defects. One study of acuity and several waveform variables did not find a correlation between patients.¹⁶ This does not mean that correlations do not exist in specific individuals; experience has proven that damping CN and increasing foveation-period duration *will* improve acuity if it is not limited by afferent defects. The reason for the variability in the results of these studies is probably due to the intercorrelations of several variables and it is for this reason that the

NFF described earlier should yield better results. The NFF contains *all* the motor variables relevant to acuity. A study on the use of telescopic aids for low-vision patients (with and without nystagmus) showed that head motion was an important factor in preserving the stable retinal images necessary for good acuity.¹⁷

We studied the foveation periods of an individual with CN during smooth pursuit and VOR and demonstrated near unity gains for both over target velocities ranging from a few degrees per second to 215 degrees/sec and for rotations in the normal range.¹⁸ We also demonstrated, using phase-plane analysis, that both smooth pursuit and VOR were normal during foveation periods. By subtracting the target position and velocity from those of the eye, we reconstructed the retinal error phase planes during pursuit and VOR and showed them to virtually duplicate the phase plane of eye movement during fixation of a stationary target. Thus, by two unrelated methods, we proved that these subsystems were *normal* in CN and that hypotheses or models claiming deficits in either of them as the cause of CN were invalid.

In a study of smooth pursuit, VOR, and optokinetic nystagmus (OKN) in individuals with CN, Kurzan and Büttner confirmed the hypothesis that the measured waveforms are caused by a shift in the static neutral zone to a new position (the dynamic neutral zone).¹⁹ In agreement with our observations, they found that even low stimulus velocities cause large shifts in the neutral zone and higher velocities caused an increased shift. They also confirmed that this shift has no measurable latency. Given their verification of the neutral-zone shift, they concluded that smooth pursuit was *normal* and that retinal slip velocity is adequately utilized for the generation of smooth pursuit eye movements in individuals with CN. Lueck et al. studied a patient who presented with episodes of OSOP with smooth pursuit and OKN responses that exhibited nystagmus slow phases in the direction opposite to the stimulus.²⁰ Several different mechanisms for the etiology of this nystagmus were discussed; in my opinion, this was a rare form of CN that is related to those previously reported by Rosenberg et al. and discussed in the last volume.²¹ This patient exhibited a jerk left CN in far left gaze and when tracking from the right to the left. Thus, his neutral zone, which was in left gaze when fixating stationary targets, shifted to the right so that the jerk left CN was superimposed on leftward pursuit movements. This case is the missing link between normal CN (where the nystagmus is present while viewing stationary targets and the neutral zone can easily be seen to shift during pursuit) and those cases previously recorded (where there was no nystagmus during fixation of stationary targets and a shift in neutral zone with pursuit caused the nystagmus to become manifest). The pursuit-induced neutral zone shift was the explanation I offered for the mechanism involved in producing the CN in the two patients previously reported and that explanation is supported by this more recent patient. In a study of smooth pursuit in several cases of hereditary CN, Takahashi demonstrated smooth pursuit during the foveation periods in his subjects.²² The finding that there was a distinct difference in the CN of the male subjects from that of the female subjects during pursuit has neither been reported before nor noted in our data.

Finding an animal model for CN has proved to be an elusive goal. Tusa et al. described the strabismus and nystagmus found in monkeys due to early visual deprivations.²³ They studied three rhesus and three cynomolgus monkeys who had monocular eyelid suture within 24 hours of birth and reverse suture at age 25 days that was continued for another 25 days. At 50 days of age, when both eyelids were opened, the monkeys had 20 to 30 diopters of exotropia and nystagmus that persisted for the duration of the study (1 year). The cynomolgus monkeys developed a monocular 8 to 10 Hz pendular nystagmus in the eye sutured first; the rhesus monkeys developed a conjugate nystagmus with both jerk and pendular components. The slow phases often had increasing velocity profiles, and the rhesus monkeys had a superimposed latent component on the nystagmus. Certain characteristics of the nystagmus corresponded to human CN, others did not, and the difference in the two species of monkeys is troublesome. There are apparently species differences in the types of nystagmus that result from visual deprivation and extending this model to humans is difficult. More study of the nystagmus induced in these monkeys is required before some of these findings might be useful in understanding the mechanisms involved in CN in humans where visual deprivation is not the primary cause.

I have hypothesized that there are basically two oscillations that are the foundations for all of the CN waveforms: pendular and an increasing-velocity slow phase jerk nystagmus. The variations found in the waveforms described are a result of the individual's fixation subsystem attempting to maintain target foveation for as long as possible before the slow phase causes the eyes to accelerate away from the target. Thus, the foveation periods are created. A recent study of the evolution of CN waveforms in infants supports this hypothesis.²⁴ The authors found that CN often starts with a large triangular waveform at 1 to 4 months of age and evolves into a smaller amplitude, higher frequency pendular waveform that subsequently might give way to a jerk waveform by 7 months to 1½ years of age. Unfortunately, the low bandwidth of the electro-oculographic (EOG) signals may have prevented identification of small foveating or braking saccades. Despite this, the figures do indicate the possible presence of these small saccades in even the earliest waveforms. The time course of the waveform evolution was found to parallel that of the sensitive period of visual development. Thus, the attempt to see would develop and increase the nystagmus frequency along with foveation periods. After 6 months of age, the triangular waveforms are virtually nonexistent (in agreement with adult data) and replaced by pendular or jerk waveforms. The authors attributed this to the development of fusional convergence.

Also addressed in this paper was the issue of the name, "congenital," in CN. It is well recognized that many cases of CN do not appear until the first weeks or months of life although it must be remembered that several have been documented *at birth*. Eye movement recordings have shown that there are many forms of nystagmus present in infancy, so-called "infantile nystagmus." CN is one form of infantile nystagmus but so are latent/manifest latent nystagmus (LMLN), spasmus nutans, and others. Therefore, it would increase confusion to rename CN, a spe-

cific type of nystagmus, with the general description "infantile nystagmus." Neither the literal translation of CN nor the exact moment that CN appears is important here; what is important in differentiating CN from the other forms of infantile nystagmus. The argument that the term "congenital" is inappropriate has also been raised in describing congenital esotropia.²⁵ Actually, Gresty et al. documented six patients in whom CN emerged in later life.²⁶ The age of onset in these patients ranged from 12 to 30 years, and the nystagmus was proven to be CN by recordings. These patients make accurate ocular motility recordings indispensable in the diagnosis of nystagmus. They exhibited the null shift with pursuit and OKN that is a characteristic of CN. One of the subjects had a brother with CN, making his a hereditary CN that appeared late in life. The presence of foveation periods in these late-onset CN waveforms proves that the ability to suppress an acceleration of the eyes off target is part of our normal ocular motor arsenal and not something developed in early life by those with CN.

Several papers have appeared that emphasize the associated sensory defects found in individuals with CN.²⁷⁻²⁹ Some have stressed the high percentage of patients with CN and these defects, but this may reflect the authors' patient population more than the CN population in general. Whatever the numbers, two things are clear: (1) physicians do have to look for any sensory defects that may be present in an individual with CN and treat them; (2) no matter what the sensory defects are, the CN is still a *motor* instability and it remains erroneous to classify CN as consisting of sensory and motor subtypes or to state that CN is a disorder indicative of "a primary disturbance of the ocular motor or visual sensory systems," the primary disturbance responsible for CN is in the ocular *motor* system.

In some families, CN is hereditary, as can be seen by its presence in more than one member. However, when information about other members of the family is unavailable or if the individual with CN is the only known member of the family to have this disorder, one still cannot rule out heredity as a mode of transmission. Shallo-Hoffmann et al. studied the eye movements of family members of subjects with CN.³⁰ In each of five families, abnormalities of seemingly nonaffected members were demonstrated; in four, saccadic instabilities were found and in the fifth, a CN waveform. Increased frequency of square-wave jerks (SWJ) and square-wave oscillations (SWO) were seen in family members. This is a curious finding since CN is due to a SEM instability, not saccadic. Neither the reason why unaffected family members would demonstrate saccadic instabilities nor the relationship of such instabilities to CN is clear. The results of this study suggest that, in isolated cases of CN, the presence of saccadic instabilities in family members might be indicative of hereditary CN. Hereditary vertical pendular CN was documented in two sisters.³¹ Nystagmus has been reported with many other hereditary conditions, but these reports usually do not contain accurate eye movement records and preclude the identification of the nystagmus as CN.³²⁻³⁸ In one report of three patients with congenital absence of conjugate horizontal eye movements and nystagmus, recordings did show a pendular nystagmus in two of the three.³⁹

One area that has received much attention and is the result of some confused

ideas about the etiology of CN is the subject of individuals with albinism. Most, but not all, albinos also have CN. Apkarian et al. showed that *all* albinos exhibit asymmetry in the monocular visual evoked potential (VEP).⁴⁰ However, not all albinos demonstrate nystagmus and therefore the link between aberrant projections and organization of the ocular motor system in non-albinos with CN is in question. They found no crossed projections in a non-albino with nystagmus. Apkarian et al. devised a decisive test for human albinism and reported a case of albinism with LMLN.⁴¹ Apkarian and Spekrijse also reported another non-albino with nystagmus who had no crossed fibers.⁴² In fact, Apkarian has tested at least 14 patients with CN and has found *none* with optic pathway misrouting (personal communication, 1990). In a study of 18 albinos, 5 of whom had no noticeable nystagmus, all showed global stereopsis.⁴³ These findings are interesting when contrasted with the investigations in albino animal models where a paucity of binocularly driven cortical neurons is found in visual areas 17, 18, and 19. Other studies have also verified that albinos demonstrate VEP asymmetries whereas those with idiopathic CN do not.^{44, 45} Thus, since electrophysiologic evidence has proven that individuals with CN do *not* have crossed fibers, hypotheses and models that rely on crossed fibers and “reversed” pursuit have no basis in fact.

Rosenberg and Jabbari suggested that a horizontal grating stimulus might be used where the conventional check stimulus for VEP produces poor results due to horizontal nystagmus. In a paper on the recognition and management of albinism, Abadi and Pascal described twin girls who had similar CN waveforms but whose fast phases were in opposite directions. In this paper, the authors stated that all forms of albinism are characterized by nystagmus; this appears to be in contradiction to the studies mentioned earlier. As a final note on albinism and CN, it has been suggested that although acuity is primarily limited in albinism by retinal factors, reducing the CN can lead to an improvement.⁴⁶

We are beginning studies of the effects of afferent (cutaneous) stimulation of the ophthalmic division of the trigeminal nerve on CN, based on our observations of the effects of contact lenses.⁴⁷ Our preliminary results show this to be a robust response.⁴⁸ We documented 50% decreases in CN amplitude with pressure, vibration, or electrical stimulation. We hypothesized that the afferent stimulation was affecting the proprioceptive calibration of the extraocular muscles since these fibers travel in the trigeminal nerve. These results infer that there is a strong effect on eye movement by changing the proprioceptive bias to the extraocular muscles despite the absence of a classical stretch reflex.

Hatayama et al. examined several patients with and without base-out prisms.⁴⁹ Unfortunately, they used bitemporal EOG to record the eye movements. Four of the five patients showed a damping of the CN waveform during fixation in primary position. However, only three of the five showed an increase in acuity and one of the three was the patient in whom no damping of the CN was seen. The use of base-out prisms will usually damp CN and, more importantly, increase foveation time. However, to improve acuity, -1.00 spheres must be added to the refraction of pre-presbyopic patients to negate the effect of the accommodation accompany-

ing the prism-induced convergence. It was not clear if this spherical correction was added to the refraction of these patients. I would anticipate that, had this been done, the acuity would have increased in more of the patients. For those interested in accurate head posture measurement with minimal artifact, an apparatus has recently been described.⁵⁰ It is my opinion that head posture is not a good way to measure CN gaze-angle nulls preoperatively or postoperatively. There is too much error that can be introduced by the subject. It is best to fix the head and measure the CN at known gaze angles.

Castanera studied the length-tension diagrams of the medial rectus muscle after the Faden operation.⁵¹ He compared two techniques and found that primary length and spring constant were unchanged with a bridge suture technique whereas a marked stiffening appeared with the original technique. It would be interesting to know which of these techniques results in the greater damping of CN. Sandler et al. described the results of 26 patients with CN who underwent the artificial divergence operation.⁵² Seventeen patients showed significant improvement after the procedure; in three, the procedure had to be combined with the Kestenbaum procedure, and in 6 patients where the affect of artificial divergence (tested with a base-out prism) did not damp the nystagmus, the Kestenbaum procedure was used. All patients had good binocular function. This study suggests that artificial divergence alone or in combination with other procedures may have a significant damping effect on CN. In support of this, our recordings have shown that, in patients where both convergence and gaze angle induce nulls, the convergence null is almost always a better one. In a study in rhesus monkeys, Gamlin et al. found that there was some degree of co-contraction of the lateral and medial rectus muscles during convergence eye movements.⁵³ This finding may be the mechanism by which CN is damped during convergence, the co-contraction of the lateral and medial muscles would render them less susceptible to the signal driving the CN. Some CN patients exhibit a vertical or torsional head posture and there is very little in the literature concerning management strategies. A recent survey sent to members of the American Association for Pediatric Ophthalmology and Strabismus found that when surgery is indicated, a graded bilateral vertical rectus recession or combined vertical rectus recession-resection procedures are usually done.⁵⁴ Since most respondents saw no cases of torsional head postures, no procedure was advocated.

Biofeedback has been advocated as a possible treatment in CN. Mezawa et al. studied the changes in CN waveform associated with biofeedback.⁵⁵ They found that their subjects were able to voluntarily damp the CN (intensity decreased by 50%) and prolong the foveation time (increased by 180%). However, after completion of the training, no clear improvement in acuity was seen, although all subjects reported a subjective improvement in their vision when suppressing their nystagmus. A key to the effectiveness of this training appears to be in the answer to the question, can they suppress while attempting an acuity test? If suppression is only possible under certain conditions and these preclude real-life situations, biofeedback, although interesting, may not prove to be a practical therapy. The

value of electroretinography (ERG) in the evaluation of children with early onset nystagmus was recently examined.⁵⁶ The incidence of congenital achromatopsia in a group of otherwise undiagnosed children was 29%, with 40% being classified as having idiopathic CN. The authors concluded that there was a need to use ERG in children when the diagnosis was uncertain.

Since the last volume, in which I reviewed a paper from a group who used bi-temporal, alternating current-coupled EOG to take their data, several papers have appeared from this group using the same method.⁵⁷⁻⁶⁰ These papers contain fairly sophisticated analyses of poorly taken data and it is very difficult to separate out what can truly be inferred from their analyses and what is artifact. Specifically, the power spectra of various CN waveforms shown in these papers are *not* the complete spectra of these waveforms but only of their overly filtered data. Thus, the large amount of power in the range from direct current to 1 Hz is missing. This is *the most important* area of the waveform since it is produced by the foveation periods that we know are intimately related to acuity and the suppression of OSOP. Some of the correct descriptions of CN found in these papers (based on the work of others) are the result of high-bandwidth, direct current-coupled measurement techniques that could not have been discovered with their method. Therefore, it is disturbing to find statements denigrating both accurate descriptive analyses of CN waveforms and neurophysiologically based models in papers whose foundations are based on that knowledge. Despite many misstatements, these papers contain information that could, if properly interpreted, be useful. Unfortunately, it takes someone familiar with both areas (CN and spectral analysis) to be able to separate the good from the bad. I would strongly urge these workers to change their methods of recording eye movements to provide good input data for any future work if they hope to make meaningful contributions.

In one of their papers,⁵⁸ they correctly concluded that the model of Optican and Zee, that produced pendular waveforms from the same basic instability that caused the jerk waveforms, was incorrect. This was already known, based on observations of many patients and the independence shown between the two waveforms in those patients that had both; we concluded years ago that the pendular and jerk waveforms were independently caused. Their reasoning for the suggestion that the pendular CN waveform differs from that found in the dual jerk waveform is not supported by the many cases where the jerk waveform is variably present but the pendular waveform remains (this is also seen with convergence). Because of the differences in methods and the inability of their recording techniques to separate CN from LMLN, their two papers on the evaluation of surgery^{59, 60} are almost impossible to compare to previous work; some of their confusing findings are due, in my opinion, to their poor input data.

Much praise is given to the importance of spectral analysis in these papers, but I found nothing new or that could not be easily predicted by anyone knowledgeable about frequency-domain analysis. We have looked at the total spectra (direct current to 100 Hz) of CN waveforms in the past and they merely confirmed what we expected based on simple observation of the waveforms. All analysis methods

have something to add to our understanding, but none give all the answers; the total reliance by these authors on spectral analysis is misplaced and their expectation that better models will arise from that approach rather than from models closely tied to known neurophysiology ignores the early history of bioengineering modeling.

Convergence Nystagmus

A case of convergence nystagmus associated with an Arnold-Chiari type 1 malformation was presented. The nystagmus appeared in the absence of fixation and was provoked during the Valsalva maneuver and neck flexion and extension as well as being attenuated on deep inspiration.⁶¹ The authors postulated that this nystagmus was due to a combination of mechanical distortion and abnormal transmission of cerebrospinal fluid pressure to the aqueductal region. The figures shown in the paper were of a pendular convergence nystagmus that could be quite asymmetric between the eyes.

Convergence nystagmus at approximately 1 Hz has been described in several cases of Whipple's disease.⁶² The authors suggested that the nystagmus be called "pendular vergence oscillations," but since this eye sign is convergence nystagmus by definition, there is no compelling reason for the less specific term "oscillations." The association of this convergence nystagmus with discharge of the masticatory muscles has given rise to the vague term "oculo-masticatory myorhythmia." Perhaps the best compromise here would be, "oculo-masticatory convergence nystagmus" to identify the oscillation as a nystagmus and use the prefix to indicate a connection with masticatory discharges.

Convergence-Evoked Nystagmus

Two cases of convergence-evoked nystagmus were reported to have occurred in spasmus nutans.⁶³ The compressed time base of the recordings shown in the paper make it difficult to determine if the nystagmus was truly disconjugate as has been reported for spasmus nutans.⁶⁴ The case description refers to the nystagmus as pendular and conjugate which would be more indicative of CN than spasmus nutans. Despite the clinical description, the two figures appear to show disconjugate nystagmus in each case. Oliva and Rosenberg presented a review of 17 patients with acquired convergence-evoked nystagmus.⁶⁵ Cases showing both vertical and horizontal nystagmus were included. A wide variety of neurologic diagnoses and other findings were included for each patient; some of these findings were not related to the nystagmus.

Dissociated Nystagmus

A peculiar form of dissociated nystagmus (divergence nystagmus) was reported with congenital adduction palsy and synergistic divergence.⁶⁶ On attempted adduction, the affected eye moved into an abducted position causing extreme divergence and simultaneous divergence nystagmus resulted. This was a jerk nystagmus with the fast phases beating temporally in both eyes. Attempted right gaze caused the affected left eye to go further to the left and optokinetic and vestibular inputs resulted in an inverse nystagmus of the affected left eye. The authors concluded that, similar to Duane's retraction syndrome, this developmental anomaly was characterized by absence of the abducens nucleus and subsequent innervation of the lateral rectus muscle by the inferior branch of the oculomotor nerve. In addition to the nystagmus, flutter was observed during intense fixation.

A dissociated nystagmus was also reported as a common sign in patients with human immunodeficiency virus (HIV).⁶⁷ Here, it was thought that the medial longitudinal fasciculus was affected and the nystagmus was indicative of the development of internuclear ophthalmoplegia (INO). If that was the case, then these oscillations were saccadic pulse trains and not nystagmus. The tracings shown resemble the so-called abduction "nystagmus" of INO; rebound nystagmus and gaze-evoked nystagmus were also seen in these patients.

Downbeat Nystagmus

Downbeat nystagmus that appeared during eye closure was reported in two patients with dizzy spells.⁶⁸ The nystagmus only appeared when the eyeballs were depressed to the midline position during eye closure. Since there were no other remarkable neurologic findings, the nystagmus was probably related to the vestibular symptoms. Weissman et al. reported downbeat nystagmus in an infant that resolved within the first year of life.⁶⁹ The nystagmus was observed by 6 weeks of age and the patient preferred to keep her head in a chin-down position. The slow phases of the nystagmus were predominantly of constant velocity, and it was suggested that the nystagmus was caused by an immaturity of central connections associated with the vertical canal pathways. Recent reports have also linked downbeat nystagmus with dolichoectasia of the vertebrobasilar artery,⁷⁰ multiple sclerosis,⁷¹ occult breast carcinoma,⁷² and alternating skew on lateral gaze (bilateral abducting hypertropia).⁷³ An EOG study of downbeat nystagmus has documented some of the uncommon features that may be encountered.⁷⁴ The use of alternating current-coupled EOG confounds the slow phases, although the time constant of 10 seconds is long enough to distinguish increasing velocity exponential slow phases from linear; one case did have the former. Pursuit, optokinetic, and vestibular responses were obtained and correctly interpreted as being confounded by the

presence of the spontaneous nystagmus rather than being defective and the cause of the nystagmus, as has been the misinterpretation applied to other forms of spontaneous nystagmus when tested under these conditions. Possible mechanisms responsible for downbeat nystagmus were reviewed, but no conclusion was reached because of the variable nature of the downbeat nystagmus in these patients.

Drug-Induced Nystagmus

A review article by Abel and Hertle documented the effects of psychoactive drugs on ocular motor behavior.⁷⁵ The authors grouped the drugs in seven categories based on their primary functional effects. Two papers reported downbeat nystagmus induced by ingestion of lithium.^{76, 77} Williams et al. studied two patients in whom lithium was administered for psychiatric illness. Several months after stopping the lithium, one patient showed a resolution of his nystagmus but the second did not. They concluded that several months of abstinence may be necessary for improvement. Halmagyi et al. studied 12 patients in whom lithium was administered. Of the six that were able to reduce or stop taking lithium, only two showed a resolution or improvement of the downbeat nystagmus. Some patients from both studies also showed gaze-evoked horizontal nystagmus in addition to the downbeat nystagmus.

Bow tie nystagmus is a drug-induced oscillation consisting of upbeat nystagmus combined with alternating horizontal saccades that are synchronized with the vertical fast phases. The resulting two-dimensional motion traces a bow tie, hence the name. The authors who first recorded bow tie nystagmus after their subject smoked tobacco have isolated the cause to the nicotine and suggested that perhaps "nicotine" nystagmus might be a better name than their original term, "bow tie."⁷⁸ I prefer bow tie for two reasons: (1) there are many drugs that induce nystagmus and creating a new name for each would unnecessarily add to an already large collection of terms; (2) bow tie describes the nystagmus correctly without linking it to any of the many possible drugs that might produce the same nystagmus. As I have urged in these chapters, new names should be descriptive of either the oscillation or, where known, the neurophysiologic mechanism.

Epileptic Nystagmus

A combination of vertical and horizontal gaze deviation and nystagmus associated with epilepsy was reported in a comatose patient with a left hemisphere subdural hematoma.⁷⁹ The vertical eye deviation and nystagmus are unusual since seizure activity usually causes a contralateral eye deviation in nystagmus. We now

have the second and third accurate recordings of the epileptic nystagmus in the literature. In one, the slow phase component was found to be linear,⁸⁰ and in the other, the slow phases were both linear and of decreasing velocity.⁸¹ These are in contrast to the first recorded case of epileptic nystagmus where a gaze-evoked, decreasing velocity slow phase was found.

Gaze-Evoked Nystagmus

Gaze-evoked nystagmus (GEN) is a jerk nystagmus, not present in primary position, elicited by attempted maintenance of eccentric eye position. GEN may have a linear or a decreasing-velocity exponential slow phase; the latter has sometimes been referred to as gaze-aretic nystagmus but, unless it is due to a nerve (gaze-aretic) or muscle (muscle-aretic) paresis, the GEN terminology is preferred. Distinction should be made between nystagmus that is truly gaze-evoked and that which is gaze-modulated. Many types of nystagmus vary with gaze angle but are present in primary position and are not GEN. Gaze-modulated nystagmus is a jerk nystagmus, present in primary position, that is increased by lateral gaze. The slow phases may be linear, decreasing-velocity or increasing-velocity exponentials. Examples of gaze-modulated nystagmus are vestibular nystagmus (VN), LMLN, and CN. Both VN and LMLN vary in intensity with gaze angle according to Alexander's law; the nystagmus increases as gaze is directed in the direction of the fast phases. CN increases as gaze is directed away from the null and, if the null is near primary position, CN may be mistaken for GEN (especially in the absence of a patient history or in a case of head trauma seen in the emergency room). These types of nystagmus increase with lateral gaze but, since their genesis is not due to gaze off primary position, they are not types of GEN. There is overlap between GEN and gaze-modulated nystagmus. Nystagmus that is modulated according to Alexander's law may be classified type I, II, or III, depending on the field of gaze in which the nystagmus first appears. Thus, a type I nystagmus (appearing only in lateral gaze) may easily be mistaken for GEN. There are both normal and pathologic types of GEN as well as of gaze-modulated nystagmus. The normal types of GEN are physiologic (end-point) nystagmus and rebound/centripetal nystagmus; the normal type of gaze-modulated nystagmus is induced VN. The pathologic types of GEN are those due to drugs or alcohol gaze nystagmus, gaze-aretic or muscle-aretic nystagmus, rebound/centripetal nystagmus, and Brun's nystagmus; pathologic types of gaze-modulated nystagmus are alcohol position nystagmus, periodic alternating nystagmus, LMLN, and CN.

Medulloblastoma has been linked with GEN and upbeat nystagmus⁸² and both horizontal and vertical GEN were found in familial paroxysmal ataxia.⁸³ In a study of three patients with central nervous system (CNS) lesions and markedly decreased time constants of the VOR, GEN was also found.⁸⁴

Induced Nystagmus

Information about diurnal variations of induced nystagmus in healthy and sick subjects has now been collected.⁸⁵ The authors demonstrated circadian rhythms of vestibular responses, and in some variables, significant differences were found between healthy and sick subjects.

Latent/Manifest Latent Nystagmus

In an exhaustive study of smooth pursuit and the optokinetic response (OKR) in LMLN, Dickinson and Abadi came to the following conclusions: (1) both pursuit and OKR could be symmetric in LMLN for both binocular and monocular viewing; (2) the asymmetric patterns of response often reported in LMLN result from either shifts in the zone of minimum intensity oscillation or from nonstimulus-specific increases in the LMLN.⁸⁶ Their careful work revealed that, when properly evaluated, there is no nasal-to-temporal (N-T) asymmetry in the smooth pursuit of subjects with LMLN and therefore, the hypothesis that an N-T smooth pursuit asymmetry is the *cause* of LMLN is invalid. As with CN, conventional measures of slow-phase velocity do not reveal the true performance of either pursuit or the OKR and the apparent asymmetries recorded are, in fact, merely reflections of the spontaneous LMLN oscillation and *cannot* be causative factors in the genesis of this condition. In another study on the N-T asymmetries of pursuit in strabismic amblyopes, correction for the mean velocity of the slow phases eliminated the observed asymmetry in the nonamblyopic eyes.⁸⁷ Although the authors of this study never mentioned that their subjects had LMLN, their description of the eye movements made it clear that they did. Thus, we have two studies that yielded the same conclusions: there is no N-T asymmetry in the smooth pursuit or OKR of subjects with LMLN.

Leigh et al. found that one of four patients with monocular loss of vision exhibited LMLN.⁸⁸ In addition, upbeat nystagmus was measured; gaze-evoked nystagmus was not found in these patients. In a patient with bilateral congenital blindness, they found nystagmus with horizontal and vertical components and a wandering null point; they attributed this to an abnormal neural integrator. Steinbach et al. studied spatial localization monocularly in each eye of eight patients undergoing unilateral strabismus surgery.⁸⁹ In most cases, they found that the changes in spatial localization produced by the surgery on the operated eye paralleled those found in the other eye both in magnitude and duration. Thus, unilateral surgery was found to have central effects on egocentric localization. Since the surgery forced a recalibration of one eye's position (the operated eye), a change was forced on the calibration for localization even in the unoperated eye. From this study, we can infer that the strabismus that *always* accompanies LMLN might also

cause a change in the calibration necessary for spatial localization and therefore result in a shift in central egocentric localization. This paper, as well as others I have reviewed in previous volumes, provide strong support for the hypothesis that LMLN is the direct result of a shift in egocentric localization and not of asymmetry in any of the ocular motor subsystems.

Zubcov et al. studied four patients with dissociated horizontal deviation.⁹⁰ All had amblyopia and asymmetric MLN and used convergence to reduce their nystagmus when viewing with the eye having the more severe nystagmus. They concluded that an asymmetric nystagmus blockage syndrome can be found in MLN. By comparing the clinical descriptions of each patient with the nystagmus shown in eye movement recordings, the authors also reemphasized that clinical observation alone is often inadequate and ocular motor recordings are necessary for accurate diagnosis. Their findings also confirm that it is the angle of convergence and not accommodation that is related to the amplitude of MLN (also of CN).

In a study of VEP in patients with early monocular loss of vision, jerk nystagmus in the sound eye was found in 7 of 16 patients.⁹¹ Since no ocular motor recordings were made, LMLN could not be verified. In a retrospective review of the charts of 119 children with infantile esotropia, it was found that 14 (12%) also had high-frequency, low-amplitude torsional nystagmus.⁹² Ten of the 14 were said to also have MLN superimposed on the torsional nystagmus and the relatively larger MLN masked the underlying torsional nystagmus. The occurrence of torsional nystagmus in association with LMLN is not uncommon and has been reported by several authors. A recent study of LMLN in infantile esotropia misidentified the nystagmus as "abduction nystagmus."⁹³ The author recognized LN upon occlusion but failed to realize that what was seen in lateral gaze was the Alexander's law increase of MLN.

The effects of both surgical and optical treatment on eight cases of LMLN have revealed that MLN may be converted to LN as a result of the treatment.⁹⁴ Five of the patients had their tropia corrected surgically, and their visual acuity improved as a result of this conversion. Of the three patients who received optical treatment, in one the MLN converted to LN with improved vision and in the other two the MLN decreased in intensity and the acuity of one of these patients improved. It thus appears that surgical or optical alignment of eyes can decrease the intensity of MLN and improve binocular visual acuity. Several of these patients had both CN and MLN. The authors concluded that there was a close relationship between the level of visual acuity and the intensity of MLN. In one case, although there was MLN, the patient appeared clinically not to have any strabismus. The authors allowed for the possibility that this patient did have a small-angle tropia that was not detected because of the high amplitude of the MLN. In previous volumes, I have discussed how small tropias can be detected by properly calibrating each eye individually (while the other eye is behind cover) so that when the cover is removed, small tropias in the non-fixating eye can be easily measured.

Simonsz reported on the effect of prolonged monocular occlusion on LN in the

treatment of amblyopia.⁹⁵ Whereas before occlusion the slow-phase velocity was lower when the better eye fixated, after occlusion it was lower when the worse eye fixated. He concluded that occlusion of the better eye in children with amblyopia and LN should be prescribed in days per week not in hours per day.

Optokinetic Nystagmus

Howard and Simpson developed a procedure by which OKN gain could be measured as a function of the binocular disparity of the stimulus to test the hypothesis that a linkage exists in higher mammals between the optokinetic system and the stereoscopic system.⁹⁶ They found that the gain of OKN was inversely proportional to binocular disparity. This supported their previous hypothesis that the main reason for the evolution of a cortical component to OKN is to allow animals with foveate eyes and stereoscopic vision to deal with complex motion signals that are generated by linear motion through a three-dimensional world. Another interesting finding about OKN is that it can exhibit the same predictive behavior that is known to occur in smooth pursuit.⁹⁷ Thus, the "primitive" OKN subsystem may actually be intimately linked with the "higher" pursuit subsystem; they may even both be part of one SEM subsystem responding to different inputs. It has long been my opinion⁹⁸ that there is only one SEM control subsystem and that the subsystems exhibited in models (including my own) for smooth pursuit, OKR, or VOR merely reflect the mathematical differences in signal processing of the various inputs to this unitary motor subsystem.

In another study of OKN in monkeys, it was found that although they tried to suppress OKN movements, the monkeys exhibited slow movements of low amplitude directed oppositely to the moving OKN stimulus while they were fixing a stationary object.⁹⁹ These SEM were apparently induced by a perceived motion of the stationary target in a direction opposite to the moving OKN background. When they exhibited optokinetic after-nystagmus (OKAN), it was always determined by the actual background motion and not by the perceived target motion.

Several papers investigated direction asymmetries of OKN. Van den Berg and Collewyn found no evidence for asymmetry in the horizontal direction but a clear preference for upward stimulus motion.¹⁰⁰ They found the decrease in OKN gain as a function of stimulus velocity was steeper for vertical than horizontal motion, and the eyes were nearly perfectly yoked for vertical OKN but not during horizontal OKN. Tracking in the nasal direction had a higher gain (by about 4%) than in the temporal direction. This difference was attributed to motor rather than sensory origin. The finding of a vertical OKN asymmetry in man is in disagreement with past reports where it was found not to be significant but in agreement with findings of vertical OKN asymmetries in rhesus monkeys, squirrel monkeys, and cats. In squirrel monkeys, the vertical OKN asymmetry was increased after bilateral sac-

culectomy.¹⁰¹ It was also found that the asymmetry of vertical OKAN is more dominant than of OKN.

Vestibular habituation in the squirrel monkey also affects OKN.¹⁰² Repeated clockwise vestibular stimulations caused an increase of counterclockwise OKN responses. It had been previously thought that prolonged OKN stimulus affected VOR gain but the reverse was not true; in these experiments, OKN responses were not affected for the first 5 days but thereafter there were clear and repeatable increases. Since the OKN response was back to baseline by the time of the next day's test, the authors concluded there was either no or a short-term storage.

In a study of the characteristics of saccades, the fast phases of OKN were found to be as fast as "foveating" saccades.¹⁰³ This term, introduced to distinguish foveating from braking saccades in CN waveforms, has now been extended to normal saccades that achieve target foveation.

Periodic/Aperiodic Alternating Nystagmus

An interesting case of intermittent unidirectional nystagmus that might be related to periodic alternating nystagmus (PAN) was recently described.¹⁰⁴ It consisted of an intermittent jerk right nystagmus occurring every 45 to 90 minutes and lasting 20 to 40 seconds; during this nystagmus, OSOP was perceived. At other times, no nystagmus could be elicited and the patient's neurologic examination was normal. The amplitude of the nystagmus followed a sinusoidal time course. The authors speculated that the nystagmus might represent dormant PAN or another type of dormant nystagmus appearing intermittently due to episodic migrainous brain stem ischemia. One paper claimed to demonstrate an alternating nystagmus appearing with infectious mononucleosis.¹⁰⁵ Unfortunately, the figure shown had no identification of the zero position for either eye and the methods did not describe whether the EOG signal was alternating current- or direct current-coupled, nor was the bandwidth of the system given. The only thing evident from the figure was that there was a conjugate oscillation that had both fast and slow phases. The figure did not demonstrate which of the two phases was the initiating phase and therefore we cannot determine whether this was nystagmus or saccadic pulse trains.

The effects of baclofen on PAN in both monkey and man raises the question of whether its beneficial effect lasts while the drug is continued.¹⁰⁶ In a 1-year study of the effects of baclofen in a patient with PAN, it was found that, although initially the PAN was abolished, continued treatment resulted in only a partial suppression. The authors concluded that baclofen does not seem to be a real therapeutic drug for PAN. The figures in this paper suffer from the same deficiencies found in the previous paper. PAN has recently been associated with administration of primidone/phenobarbital.¹⁰⁷ With withdrawal and discontinuation of these medica-

tions, the cycle length, velocity, and magnitude of the PAN diminished and then disappeared. Periodic alternating (ping-pong) gaze has been reportedly induced by deliberate overdose of tranlycypromine.¹⁰⁸ This condition resolved spontaneously, leaving no lasting impairment. This is the first report of ping-pong gaze resulting from drug therapy or toxicity.

Physiologic Nystagmus

Eizenman et al. recently published the results of a study of the three types of physiologic (end-point) nystagmus: (1) sustained; (2) unsustained; and (3) fatigue.¹⁰⁹ They found that the onset of end-point point nystagmus is determined by the velocity of the slow-phase drift and investigated the factors affecting drift velocity: target eccentricity, visual feedback, and fatigue. Visual feedback served to decrease drift velocity; mean drift velocity increased during the fixation period (the increase was more pronounced for larger fixation eccentricities), and mean drift velocity increased for larger fixation eccentricities. They found that when the drift velocity was less than 0.3 degrees/sec, no end-point nystagmus occurred. For drift velocities between 0.3 and 1 degree/sec, either sustained or unsustained end-point nystagmus was observed and for drift velocities above 1 degree/sec, either sustained or fatigue end-point nystagmus was apparent. The authors included the results of their study in an integrated model of ocular motor control that included saccadic, pursuit, and optokinetic subsystems. Their model demonstrated that the reduction in drift eye movement velocity during fixation of a visual target as compared to attempted fixation in the dark was mainly due to their smooth pursuit subsystem. Since the subject is not actually pursuing a moving target, it is more appropriate to describe the subsystem responsible for this reduction in drift velocity as part of the SEM subsystem that is operating during fixation rather than the part that is operating during smooth pursuit.

Pseudospontaneous Nystagmus

Blessing and Kommerell found that the small light sources of Frenzel's glasses can induce a Purkinje's figure on the retina and that this stabilized retinal figure may elicit an artificial pseudospontaneous nystagmus.¹¹⁰ Since this might be mistaken for spontaneous vestibular nystagmus, they advocated the use of glasses that cannot induce a Purkinje's figure. They found that 22 of 55 normal subjects were able to produce pseudospontaneous nystagmus when instructed on how to induce a Purkinje's figure.

Rebound Nystagmus

Shallo-Hoffmann et al. recently studied both the characteristics of physiologic end-point nystagmus and rebound nystagmus.¹¹¹ With sustained lateral gaze of 40 degrees and 50 degrees, end-point nystagmus almost always appeared immediately and was sustained for 15 to 25 seconds. In five of their subjects who exhibited end-point nystagmus, they measured rebound nystagmus whose magnitude was always less than the end-point nystagmus and decayed over a 5- to 10-second time period. An important finding of this study was that rebound nystagmus can be evoked in healthy subjects even when a fixation target, in a fully lit room, is present; it had previously been thought that darkness was necessary for rebound nystagmus.

A new syndrome has been suggested based on three members of a family with motion sickness and rebound nystagmus.¹¹² The patients seemed to have a type I VN, saccadic pursuit, defective OKN slow phases and failure of VOR suppression. The authors suggested the name "familial vestibulocerebellar dysfunction."

See-Saw Nystagmus

Nakada and Kwee have analyzed the role of visuo-vestibular interaction in the pathogenesis of see-saw nystagmus.¹¹³ They reviewed all known cases of see-saw nystagmus in the literature and concluded that see-saw nystagmus is an ocular oscillation brought about by an unstable visuo-vestibular interaction control system. They postulated that it was the nonavailability of retinal error signals to the inferior olivary nucleus, due to disruption of chiasmal crossing fibers, that caused the system instability. Although a model was not presented in this paper, a mathematical and control system analysis is available as a supplement by request.

Torsional Nystagmus

Morrow and Sharpe reported on three patients with lateral medullary syndrome who exhibited torsional nystagmus.¹¹⁴ The slow phases of the torsional nystagmus included increasing, decreasing, and constant velocity. In addition, torsional pulsion of saccades was observed. The authors concluded that torsional nystagmus was due to an imbalance of central projections from the anterior and posterior semicircular canals and the otolith receptors. Unlike another report on torsional nystagmus published in the same year and reviewed in the last volume,¹¹⁵ these authors chose to define torsional nystagmus from the point of view of the *observer* rather than the *subject*. Since all other eye movements are defined from the point of view of the subject and since such a definition makes interpretation of OSOP

due to torsional, circular, or elliptical movements easier, I would urge that all future reports and recordings of these types of nystagmus adopt the Kestenbaum convention (i.e., from the point of view of *subject*). Thus, someone with a clockwise torsional slow phase (and counterclockwise quick phase) would experience a counterclockwise OSOP and presentation of a clockwise OKN stimulus would result in clockwise slow phases (counterclockwise fast phases) of OKN.

We reported a patient with a combination of torsional, see-saw, and bow tie nystagmus.¹¹⁶ The patient had brain stem anomalies that appeared to have disturbed central vestibular pathways.

Unilateral Nystagmus

Pritchard et al. studied ten patients who had long-standing visual loss and monocular vertical oscillations.¹¹⁷ These oscillations varied from 3 to 50 degrees with frequencies of 0.12 to 5 Hz.

The authors were particularly interested in the waveform characteristics of the vertical oscillations. Their recordings revealed that several subjects appeared to have two waveforms superimposed on, and interfering with, one another. In these patients a low-amplitude, high-frequency waveform was superimposed on a larger amplitude, low-frequency waveform. Since these were unilateral oscillations, they were probably not due to artifact. I agree with the authors that these phenomena deserve further study and suggest that perhaps another, more accurate method of recording eye movements might be used to study the interaction between the two frequencies.

Upbeat Nystagmus

Two recent papers investigated possible mechanisms responsible for upbeat nystagmus. Ranalli and Sharpe reviewed reports of upbeat nystagmus with pathologically verified lesions and concluded that upbeat nystagmus was due to bilateral damage to the ventral tegmental pathway (an upward VOR pathway) in pontomedullary disease.¹¹⁸ Harada et al. investigated upbeat nystagmus in humans and cats.¹¹⁹ They presented a case with a discrete vascular lesion in the lower dorsal brain stem involving the hypoglossal nucleus and adjacent structures and, in addition, they presented the results of experiments in cats where the floor of the fourth ventricle was destroyed electrolytically. From their data, they concluded that a prepositus hypoglossal lesion could result in a tonic imbalance for eye position and eye movements of the vertical axis and thereby cause upbeat nystagmus in both humans and cats. Traccis et al. described a rare case of upbeat nystagmus due to cerebellar astrocytoma.¹²⁰ Their patient developed upbeat nystagmus with increasing velocity slow phases. Surgical

removal of the tumor resulted in a virtual disappearance of the upbeat nystagmus. Another report of upbeat nystagmus accompanied a focal hemorrhagic lesion of the left brachium conjunctivum, the anterior vermis, and the anterior superior left cerebellar hemisphere.¹²¹ This nystagmus was suppressed by a contralateral head tilt, and the authors postulated that the nystagmus was inhibited by the otolith-ocular reflex.

Vertical Nystagmus

Baloh and Yee studied the records of 106 patients with spontaneous vertical nystagmus that had been evaluated over the past 10 years.¹²² They found correlations between downbeat nystagmus and lesions involving the caudal midline cerebellum and with upbeat nystagmus and lesions of the central medulla. They concluded that, since the vestibular system is the main source of tonic input to the ocular motor neurons and since both the up and down VOR pathways separate beginning at the level of the vestibular nuclei, asymmetric involvement of these pathways could explain spontaneous vertical nystagmus. Thus, both upbeat and downbeat spontaneous nystagmus would, by their analysis, be types of central vestibular nystagmus resulting from an imbalance in central VOR pathways. They did not offer explanations for the changes in these types of nystagmus with lateral gaze or convergence.

Vestibular Nystagmus

An interesting study examined the effects of temperature on the magnitude of fixation suppression of the VOR.¹²³ Using both warm and cold caloric vestibular stimulation, the authors found that the warm media elicited more nystagmus and significantly less fixation suppression than the cool media. This effect worsened as a function of subject age. The authors concluded that separate normal upper limits for fixation and suppression need to be determined for different temperature caloric testing and age should be factored into these norms. A related study was of the test-retest repeatability of caloric responses.¹²⁴ Twenty-nine subjects were evaluated over a 6-month period and it was found that for any given subject, there was a reasonably reliable repeatability over time for testing of young and old subjects. Intersubject variability was greater in older subjects. In a study of the effects of linear acceleration, it was found that the induced L-nystagmus was modifiable.¹²⁵ Similar to the VN produced by rotation, L-nystagmus could be suppressed or augmented by appropriate visual or imaginary stimuli.

Brandt wrote an extensive review article on positional and positioning vertigo and nystagmus.¹²⁶ The review contains both theoretical mechanisms and clinical

signs as well as management of many different types of positional and positioning nystagmus. It is well worth reading for the clinician who sees patients with vestibular symptoms. Positional vertigo is usually treated with various exercises meant to repeatedly elicit the vertigo.¹²⁷ Various maneuvers can be used to elicit vertigo, and this study found that since the specific maneuvers differed from patient to patient, prior testing of each patient is necessary before prescribing adequate exercises.

A recent paper reported vestibular symptoms arising from vascular compression of the eighth cranial nerve.¹²⁸ Magnetic resonance imaging documented a tortuous basilar artery compressing the eighth cranial nerve on the involved side. In another study, a review of 84 patients with vertigo resulted in a conclusion that the vestibular labyrinth is selectively vulnerable to ischemia within the vertebrobasilar system.¹²⁹ Positional vertigo also resulted from surgical trauma.¹³⁰ This patient developed vertigo due to trauma to the labyrinth during surgery for partial excision of the upper jaw for squamous carcinoma. Asymmetry in vestibular nystagmus was documented in congenital ocular motor apraxia.¹³¹ Along with the asymmetry of VN was an asymmetric OKN. The neural and anatomical reasons for these asymmetries are not presently well understood. Head-shaking nystagmus was noted in 37 of 108 patients referred for caloric testing.¹³² However, these authors found that head-shaking nystagmus and canal paresis were insensitive predictors of either hearing loss or of each other; they concluded that head-shaking nystagmus is not as powerful a test as canal paresis in detecting lesions of the eighth nerve.

Unilateral labyrinthectomy in six rhesus monkeys resulted in spontaneous VN where the slow phases could be increasing then decreasing in velocity.¹³³ The authors of this study concluded that peripheral vestibular lesions can alter the function of the ocular motor neural integrator. Labyrinthectomy also impaired the velocity-storage component of the OKN system. The initial reduction in the time constant of velocity storage only partially recovered. A model with bilateral central vestibular connections, including an inhibitory vestibular commissure, suggested that changes in commissural gains either played no role at all or at most only a secondary role in the restoration of static vestibular balance. In a related study,¹³⁴ the influence of visual experience on vestibular compensation was investigated. The authors found that visual experience after labyrinthectomy was essential for recovery of VOR gain but not for the resolution of spontaneous VN.

A study of 70 patients with acoustic neurinomas found a highly significant linear relationship between the tumor size and the caloric side difference.¹³⁵ For tumors larger than 20 mm, disturbed pursuit movements or GEN were frequently found. Vestibular nerve section, which is used in the management of Meniere's disease, creates a state of dysequilibrium which usually resolves. However, 6 patients have been reported who had persistent spontaneous VN after this procedure.¹³⁶ Torsional (rotatory) recovery nystagmus has been identified as an important localizing sign in endolymphatic hydrops.¹³⁷ The descriptions of torsional nystagmus in the paper were based on the observer's point of view rather than the subject's.

For those interested in epidemiology, studies on benign paroxysmal positional vertigo and Meniere's disease in Japan are available.^{138, 139}

SACCADIC INTRUSIONS AND OSCILLATIONS

Nonnystagmic ocular motor oscillations and intrusions represent solely saccadic or saccadically initiated instabilities. Table 2 shows 16 varieties of saccadic oscillations and intrusions that have been characterized in the literature by the other terms shown, including 10 which erroneously contain the term "nystagmus." This section contains discussions of recent studies of saccadic oscillations and intrusions; they are in alphabetical order. For detailed discussions of these and other types of saccadic intrusions and oscillations, see previous volumes on the subject.¹⁻⁵

Flutter

Low-amplitude flutter has been described in two cases and given the designation "microflutter."^{140, 141} In one, cerebellar degeneration was the diagnosis. Ashe et al. studied five cases with microflutter and hypothesized that microflutter was not merely a case of low-amplitude flutter but a mechanistically different oscillation.¹⁴² Their suggestion that the term, "microsaccadic flutter" is more "specific" than microflutter confuses specificity with redundancy; *all* flutter is, by definition, *saccadic* and this attempt to drive home that point is overkill. More difficult to resolve is the question of mechanism. The authors used a modification of their original model for flutter that included the local, resettable neural integrator first proposed by Abel et al.¹⁴³ to support the suggestion that different mechanisms were involved. They demonstrated that flutter might be caused by an increased delay of the feedback signal from the local integrator plus a lowered tone signal in the pause cells and that microflutter might be due to a lowered tone signal alone. They did not use the model to show that both could not be caused by some other single deficit. Since *both* of these are speculative mechanisms based on a putative model of the saccadic pulse generator itself and since these specific, subtle changes are more easily made on a model than in that small area of the brain stem where these cells and their connections reside, it is premature to conclude that the mechanism for microflutter is significantly different from that of flutter. The former may merely be the precursor of the latter or reflect a smaller disruption to that area of the brain stem. In keeping with the guidelines at the beginning of this chapter, microflutter is included as a subtype of flutter in Table 2.

Opsoclonus

Opsoclonus was first described in 1913¹⁴⁴ and again in 1927.¹⁴⁵ In addition to the many disorders that have since been associated with opsoclonus, we can now add organophosphate poisoning,¹⁴⁶ thymic carcinoma,¹⁴⁷ Epstein-Barr virus infection,¹⁴⁸ hyperosmolar stupor,¹⁴⁹ and hypertension.^{150, 151} Also, a case of opsoclonus-myoclonus (“dancing eyes, dancing feet”) was reported secondary to cocaine usage.¹⁵² The episode was self-limited over 4 weeks and had not reappeared after 1 year.

Saccadic Lateropulsion

Two cases with leftward saccadic pulsion during vertical saccades were studied with EOG.¹⁵³ The patient with Wallenberg’s syndrome exhibited lateropulsion, whereas the patient with the proximal type of the superior cerebellar syndrome had contrapulsion.

Saccadic Pulses/Pulse Trains

Gamlin et al. studied the effects (in rhesus monkeys) on convergence and conjugate eye movements of lidocaine-induced unilateral INO.¹⁵⁴ While a thorough discussion of the many findings of this study are beyond the scope of this chapter, relevant findings were: (1) uniocular vertical nystagmus with unyoked fast phases inferred independent operation of the individual saccade generators; and (2) abduction “nystagmus” was actually caused by adaptive saccadic behavior. This latter finding is yet another supporting the classification of this sign as a saccadic oscillation. A report of INO in tuberculous meningitis failed to appreciate this distinction.¹⁵⁵

Square-Wave Jerks/Oscillations

Shallo-Hoffmann et al. studied the occurrence of square-wave jerks (SWJ) in healthy subjects under different conditions.¹⁵⁶ They concluded that SWJ were normal with the eyes closed, in darkness while looking straight ahead, and while fixating a target in a lighted room. More than 16 SWJ per minute while fixating or 20/min under the other conditions should be considered abnormal according to these authors. A difference between this and a previous report¹⁵⁷ on the number of

SWJ occurring in darkness was attributed to the specific instruction, “hold the eyes still” given in the other study. In a related study, Shallo-Hoffmann et al. reported the characteristics of SWJ over a broad age range.¹⁵⁸ Their results showed no relationship between SWJ frequency and age.

The occurrence of both SWJ and square-wave oscillations SWO has been documented in two patients with acquired immunodeficiency syndrome.¹⁵⁹ The SWO had a frequency of up to 5 Hz.

Square-Wave Pulses (Bursts/Single)

When “macro square-wave jerks (MSWJ)” were first recorded and named, they were of greater amplitude than SWJ and were recognized as being caused by a different mechanism since their pulse width was not the saccadic interval (200 ms) characteristic of SWJ.¹⁶⁰ Nevertheless, the name “macro square-wave jerks” was chosen. This name had its origin in “pendular macro-oscillations,” which had been used to describe these eye movements at the bedside. Eye movement recordings showed that they were saccadic and not pendular, so MSWJ was chosen despite the mechanistic difference from SWJ. Since that time, despite several papers restating the differences,^{161, 162} this doubly erroneous name (they are *not* always large and they are *not* SWJ) has continued to mislead both the scientific reader and investigator. As one of those responsible for this name, I am herein proposing that we drop “macro square-wave jerks” from the literature and use the more correct description “square-wave pulses (SWP)” for this low-pulse-width saccadic intrusion (less than the 200 ms intersaccadic latency). When bursts of these SWP appear on a strip chart recording made at normal paper speeds, the oscillation looks like narrow square pulses separated by the saccadic latency; at fast paper speeds, no saccades appear “square” but despite this, the term is well known and gives an immediate and correct picture in the mind of the reader. An example of how the term “MSWJ” may have caused confusion is in the inclusion of two patients (numbers 2 and 3) in the category of SWJ in a 1982 paper on SWJ.¹⁶³ The authors recognized that the short latency between the initiating and return saccades differed from SWJ but because of the implications of the term “macro,” the low amplitude of the intrusions may have kept them from considering MSWJ; these patients’ intrusions are defined by their timing not their amplitude and should be classified as SWP (at the time, MSWJ). The characteristics of SWP and other similar oscillations are summarized in Table 3.

Superior Oblique Myokymia

Morrow et al. recorded superior oblique myokymia (SOMY) in an idiopathic case and in a case of an astrocytoma involving the midbrain tectum.¹⁶⁴ Monocular

TABLE 3.
Characteristics of Square-Wave Instabilities*

Characteristic	SWJ	SWO	SWP	MSO
Amplitude (degrees)	0.5–5†	0.5–5†	4–30	1–30
	Constant	Constant	Variable	Increasing then decreasing
Time course	Sporadic/bursts	Bursts	Bursts/sporadic	Bursts
Latency (ms)	200	200	50–150	200
Foveation	Yes	Yes	Yes	No
Presence in darkness	Yes	Yes	Yes	No

*SWJ = square-wave jerks; SWO = square-wave oscillations; SWP = square-wave pulses; MSO = macro saccadic oscillations.
†Amplitudes occasionally may reach 10 degrees.

bursts of tonic and phasic intorsion and depression and miniature oscillations were found. In both patients, the torsional and vertical movements were synchronous with similar waveforms, but the torsional movements were about twice as large as the vertical. The waveforms of SOMY consisted of either a slow sustained tonic intorsion and depression or a phasic intorsion; each was followed by a decreasing velocity return. Occasionally there were small torsional-vertical oscillations superimposed on the tonic deviations. The tonic amplitudes were 2 to 3 degrees of intorsion and 1 to 1.5 degrees of depression. The phasic amplitudes were 0.5 to 1 degree and the small oscillations were less than 0.5 degree at up to 20 Hz. Thus, in the torsional plane, SOMY consists of either an intorsional saccade followed after some time by a decreasing velocity return (tonic) or an intorsional saccadic pulse (phasic). In addition, bursts of small pendular oscillations can be superimposed on the tonic intorsions. These movements are also seen in the vertical plane. The symptoms stopped in the second patient after tumor resection. An EOG recording of the pendular horizontal and vertical components of SOMY in a patient's right eye also appeared.¹⁶⁵ They looked to be in phase, resulting in a diagonal movement; the torsional component was said to be clockwise (presumably from the observer's point of view).

Ruttum and Harris reported on the results of surgery in superior oblique myokymia.¹⁶⁶ They found that when superior oblique tenotomy or tenectomy were used, OSOP recurs in approximately 50% of the patients; incomplete transection of the tendon, residual attachments, or postoperative adhesions were given as possible reasons for the failures. Repeat surgery with a superior oblique myectomy and trochlectomy via an anterior orbital approach was successful in one such patient.

OSCILLOPSIA

Since the identification in the early 1970s of the foveation periods in CN waveforms, their importance in visual acuity was obvious. All therapies for CN have

stressed reduction of the nystagmus waveform intensity with the hoped-for result that the foveation-period durations would be increased. What was less appreciated was the importance of stable, on a beat-to-beat basis, foveation periods in the suppression of OSOP in subjects with CN. This was partially due to the early hypothesis that OSOP was suppressed in CN by efferent copy of the CN waveform.¹⁶⁷ Based on the premise that the best way to study OSOP in healthy subjects is to understand its suppression in those with CN, we have begun to study this phenomenon. Inherent in this premise is the hypothesis that the mechanisms used by subjects with CN to suppress OSOP do *not* represent specially developed abilities but rather result from the application of the normal capabilities present in the ocular motor system. If this hypothesis is true, the subject with CN becomes an excellent model for the study of healthy subjects who acquire OSOP due to neurologic deficits in later life and, once the mechanism used by subjects with CN is understood, we should be able to therapeutically apply this knowledge to subjects with acquired OSOP.

We studied the waveform changes in a subject with hereditary CN who experienced intermittent OSOP after an episode of loss of consciousness.¹⁶⁸ Using the same type of phase-plane analysis employed to study foveation periods in CN, we found that his normal CN fell into a foveation window defined by 0 ± 0.5 degrees and 0 ± 4 degrees/sec limits on eye position and velocity, respectively. However, when he complained of OSOP, he exhibited a different waveform that *never* entered this foveation window. Thus, when the foveation periods fell within this foveation window on a beat-to-beat basis, he was able to suppress OSOP and when his waveform did not allow foveation periods to fall within the window, he could not suppress OSOP. We performed the same analysis during retinal image stabilization (RIS) and the results did not change. We concluded from this that RIS by itself was insufficient to suppress OSOP and further that the mechanism that prevents OSOP in subjects with CN requires the ocular motor stability provided by the CN foveation periods; without it, OSOP is not suppressed even during RIS. Thus, we infer that all one needs to suppress OSOP is a short period of time during which the subject can foveate a target of interest with a low retinal slip velocity on a repeatable basis. In subjects with acquired oscillations, ways must now be found to alter the oscillations in such a way to achieve these periods of stable target foveation.

In a study of human horizontal VOR, it was found that high-acceleration stimuli resulted in OSOP.¹⁶⁹ The gaze disturbance that resulted in OSOP was attributed to the latency of the VOR, which was found to be 6 to 15 ms. The instability of gaze during locomotion has also been studied.¹⁷⁰ Two patients with bilaterally deficient vestibular function complained of OSOP during walking. It was found that during sitting and standing their gaze was as stable as in healthy subjects, but during walking in place the gaze velocity was double that of healthy subjects. Their VOR compensated well during active head motions but not during locomotion; this was attributed to the predictable nature of active head movements. The authors concluded that testing of such patients should include perturbations similar to those

that occur during locomotion. Anticonvulsant therapy also affects the VOR and can cause OSOP.¹⁷¹ In a study of eight patients with epilepsy receiving anticonvulsants who had recurrent visual disturbances (diplopia and OSOP) in both the horizontal and vertical planes, it was found that both the vergence and version mechanisms (smooth pursuit and gaze-holding) were affected. Spontaneous vertical nystagmus and up-down asymmetry of the VOR were found; the former caused OSOP with a stationary head and the latter during vertical head motion. OSOP has also been reported in a case of otolith Tullio phenomenon.¹⁷² This sound-induced OSOP results from paroxysms of the ocular tilt reaction. When the Valsalva maneuver was performed, both the tonic eye movements and OSOP induced were opposite in direction to those produced by the sound, reflecting the push-pull nature of the otoliths. Sound stimulation elicited a paroxysmal ocular tilt reaction with both phasic and tonic components. Initially there was rapid phasic clockwise (reported as counterclockwise with respect to the observer) rotary-upward deviation that was followed by a similar tonic effect as long as the sound stimulation lasted. Switching the sound off produced a rapid return of the eye to primary position.

A first-person account of OSOP secondary to Wallenberg's syndrome by an experienced observer is worth reading.¹⁷³ The accompanying paper describes the eye movements associated with the OSOP.¹⁷⁴ Clear examples of dysmetria and SWO were recorded in this patient. An up-to-date chapter on the management of OSOP is available.¹⁷⁵ In it are descriptions of new optical treatments to alleviate OSOP. Acquired nystagmus causing OSOP has also been treated with botulinum.¹⁷⁶ Two patients were treated and OSOP diminished with an increase in acuity that lasted for 3 to 18 weeks.

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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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