This review reports on recent advances in understanding nystagmus and other involuntary eye movements. Advances in quantitative evaluations of eye movements using oculography, computational model simulations, genetics, and imaging technologies have markedly improved our understanding of the pathophysiology of involuntary eye movements, as well as their diagnosis and management. Patient-initiated capture of eye movements, especially when paroxysmal, and the online transfer of these data to clinicians would further enhance the ability to diagnose involuntary eye movements.

**Key Words** eye movement, nystagmus, saccadic oscillation.

**INTRODUCTION**

Ocular oscillations mainly comprise nystagmus and saccadic intrusions/oscillations.\(^1\)\(^-\)\(^10\) Nystagmus refers to repetitive and involuntary to-and-fro eye movements that are initiated by a slow drift of the eyes away from the object of interest.\(^8\) Nystagmus should be distinguished from other oscillatory eye movements, especially saccadic intrusions and oscillations.\(^5\)\(^,\)\(^6\)\(^,\)\(^8\) Unlike in nystagmus, the initial abnormal eye movement is a saccade in saccadic intrusions/oscillations.\(^5\)\(^,\)\(^6\)\(^,\)\(^8\)

Eye movements are generated by signals transmitted from the eyes and inner ears to structures located in the brainstem and cerebellum, where they are closely monitored and modulated.\(^4\) Human eye movements include saccades, smooth pursuit, the vestibulo-ocular reflex (VOR), optokinetic eye movements, vergence, and fixation, and each such subclass of eye movements is generated and modulated by its own neural network.\(^8\) The eye-velocity signals generated for each eye movement are integrated in the neural structures (neural integrators) in order to hold the gaze steady in the required position.\(^11\) Dysfunction of these structures and the resultant imbalance or instability in each subclass of eye movements may generate involuntary eye movements. Nystagmus may occur when the subsystems involved in slow eye movements (smooth pursuit, the VOR, optokinetic eye movements, and vergence) or the gaze-holding mechanisms are impaired. In contrast, saccadic intrusions/oscillations may be ascribed to instability of the networks involved in generating saccades.\(^12\)

Identifying and characterizing nystagmus and saccadic intrusions/oscillations provide valuable information about lesion sites and pathophysiology.\(^5\)\(^,\)\(^6\)\(^,\)\(^8\)\(^,\)\(^10\) The present review provides an update of the major findings in nystagmus and other oscillatory eye movements since 2017.
GENERAL FINDINGS

Peripheral disorders
The presentation of significant spontaneous nystagmus is associated with hospitalization for ≥5 days and nystagmus persisting at 1 month after discharge (with a cutoff of 12.1°/s) in patients with vestibular neuritis (VN). Two of three patients with delayed endolymphatic hydrops showed a typical horizontal-torsional nystagmus beating to the healthy side, whereas the third showed initial upbeat nystagmus with a slight torsional component that evolved into horizontal-torsional nystagmus beating to the healthy side. Initial upbeat nystagmus might reflect inhibition of the superior semicircular canal in the involved ear.

Central disorders
In Wernicke's encephalopathy, spontaneous upbeat nystagmus often changes into downbeat nystagmus (DBN) during eccentric gazes and convergence, or after vestibular stimulation. Furthermore, initial upbeat nystagmus may evolve into permanent DBN. Hypothetical explanations for these transitions and their evolution include directional vulnerability to thiamine deficiency of the vertical gaze-holding networks in the dorsomedial medulla, and impaired processing of otolith information. Differentiating neuromyelitis optica spectrum disorder (NMOSD) from multiple sclerosis (MS) is important for both ensuring appropriate treatments and minimizing irreversible neural damage. When the brainstem is involved, the presence of horizontal gaze-holding nystagmus with bilateral lesions involving the medial vestibular nucleus and nucleus prepositus hypoglossi favor a diagnosis of NMOSD over MS. Recording eye movements revealed gaze-holding nystagmus and superimposed pendular nystagmus in five of seven patients with MS and ocular oscillations. The gaze-holding nystagmus showed all the features of a leaky integrator, while the pendular nystagmus presented with all the features of an unstable integrator. Simulations using a computational model aided in conceptualizing the underlying pathophysiology of these abnormal eye movements in a common disorder. Quantitative evaluation of eye movements using video-oculography revealed pendular nystagmus and ocular bobbing in a comatose patient with a pontine hemorrhage, with the authors proposing three hypothetical models to explain these eye movements that involved the inferior olive, neural integrator, and omnipause neurons.

Patients showing positivity for serum anti-GQ1b antibody may present with an acute or fluctuating vestibular syndrome (vertigo, nystagmus, and ataxia) without ophthalmoplegia. They may show various patterns of nystagmus, including spontaneous, gaze-holding, and central positional nystagmus (CPN). Patients with lateral medullary infarction may show persistent nystagmus with dizziness/imbalance. Transcranial magnetic stimulation of the cerebellum may improve dizziness that remained during the chronic phase of lateral medullary infarction along with the disappearance of ipsilesional nystagmus and a decrease in the horizontal VOR gain bilaterally.

In Charles Bonnet syndrome (CBS), visual hallucinations occur after visual impairments, and they might be caused by spontaneous activities in the extrastriate visual cortex. Two patients with CBS and periodic alternating nystagmus reportedly experienced visual motion hallucinations synchronous with the nystagmus. This may be explained by ocular proprioceptive inputs from the extracocular muscles projecting to either the extrastriate areas processing visual scenes, or the higher-order visual cortical areas involved in analyzing motion signals across the entire visual fields.

Evaluation
Genetic testing for infantile nystagmus and DBN may improve diagnoses. Implementing home-based vestibular event monitoring by patient-initiated capture of ictal nystagmus could help in detecting nystagmus during vertiginous attacks and in the differential diagnosis of three of the most commonly encountered causes of episodic vertigo: vestibular migraine (VM), Meniere's disease, and benign paroxysmal positional vertigo (BPPV).

SPONTANEOUS NYSTAGMUS

Infantile nystagmus
Oscillopsia refers to the illusory motion of the visual environment, and is rare in infantile nystagmus syndrome (INS). However, new-onset oscillopsia in a patient with early-onset eye oscillations is not uncommon in routine clinical practice. It is often associated with other changes in the central nervous system, the eye, or the ocular motor system, such as uncompensated strabismus, medication alterations, stroke, age-related cerebrovascular disease, and dementia. In a 74-year-old male with INS, the presence of an epiretinal membrane (ERM) produced an unexpected focal area of torsional/vertical oscillopsia in the portion of the visual field that corresponded to distortions from the ERM. The oscillopsia improved after removing the ERM, which suggests that suppression of the oscillopsia in INS requires undistorted vision across the visual fields.

Predicting future visual function is important in children with infantile nystagmus. A recent longitudinal cohort study found that handheld optical coherence tomography was useful for predicting future visual acuity in infantile nystagmus. Future visual acuity was predicted better by structural grad-
ing than by quantitative segmentation and preferential looking. Individuals with infantile nystagmus can read at normal or near-normal speeds. It is recommended that patients with infantile nystagmus should read horizontally oriented text in order to obtain the best reading performance.36

Downbeat nystagmus
DBN is a common form of acquired central fixation nystagmus that is often associated with other cerebellar signs. Despite its distinctive clinical features, the mechanisms of DBN remain to be elucidated.28 A recent genome-wide association study found a significant association between DBN and variation in the fibroblast growth factor 14 (FGF14) gene located on chromosome 13. FGF14 is expressed in Purkinje cells (PCs), and its reduction leads to decreases in the spontaneous firing rate and excitability of PCs, which are compatible with the pathophysiology of DBN.29 In addition, mutations in the FGF14 gene cause episodic ataxia type 937 and spinocerebellar ataxia type 27 (SCA27).28 FGF14-associated phenotypes also highlight the overlap between progressive and episodic ataxias, similar to those observed in mutations involving CACNA1A.37 SCA27 is a typical childhood-onset disorder that presents with tremor, gait ataxia, parkinsonism, depression, and anger outbursts. Given the genetic relationship between SCA27 and DBN, idiopathic DBN could be a late-onset and milder manifestation of SCA27.28 While DBN is typically observed in dysfunction of the lower cerebellum, the association of DBN with motor neuron diseases indicates the need to look for neuromuscular disorders in patients with this type of nystagmus.36-40 SCA38 is characterized by DBN, intermittent strabismus, and hearing loss in addition to gait ataxia, and is associated with lower total scores on the Scale for the Assessment and Rating of Ataxia (SARA) and higher levels of docosahexaenoic acid, which should also be validated for DBN.41 There are rare cases of DBN being observed during attacks of Meniere’s disease, which are probably due to asymmetry in the vertical VOR or saccular dysfunction.42 Thus, Meniere’s disease should be considered in recurrent audiovestibulopathy and icctal DBN.

Seesaw nystagmus
While the pathogenesis of pendular seesaw nystagmus remains elusive, dysfunction of the visuovestibular mechanisms that control eye movements may play a pivotal role.43 Pendular seesaw nystagmus has been described most frequently in large parasellar tumors, and has been attributed to either the effects of commonly associated visual field defects or secondary midbrain compression.44 Based on a patient with paraneoplastic seesaw nystagmus and opsoclonus due to breast cancer and antineuronal nuclear autoantibody type 2, increased excitability of the excitatory burst neurons in the interstitial nucleus of Cajal and resultant reverberations of the reciprocally excitatory circuit was proposed as the mechanism underlying pendular seesaw nystagmus.44

Windmill nystagmus
Windmill nystagmus is characterized by a clocklike rotation in the direction of nystagmus.4 Like in other abnormal eye movements observed in the blind, windmill nystagmus is explained by a lack of visual feedback from the image motion on the retina that stabilizes the velocity-storage mechanism or gaze-holding network. Given that windmill nystagmus was observed in association with subacute progressive vision loss for 3 months, the time span required for the development of impaired gaze-holding or velocity-storage mechanism may be shorter than thought previously.45 Furthermore, windmill nystagmus has been observed in a patient with paraneoplastic cerebellar dysfunction but normal vision, which suggests that this form of nystagmus can occur in the absence of any vision loss.46

Divergence nystagmus
There are rare cases of divergence nystagmus being observed in patients with lesions involving the dorsal pons and midline cerebellum, while convergence or convergence-retraction mostly indicates an upper midbrain lesion.47 Divergence nystagmus supports that divergence is generated by active neural innervation rather than by passive relaxation after convergence.

TRIGGERED NYSTAGMUS
Rebound nystagmus
Rebound nystagmus is a common cerebellar sign that is observed when the eyes return to the primary position following a period of prolonged eccentric gaze-holding. Even in healthy subjects, rebound nystagmus may be evoked after extreme (±40°) eccentric gaze, which is modulated by the presence of vision.48 The slow phase of rebound nystagmus is directed toward the former eccentric gaze position and the rapid phases away from it.49 Since the initial velocity of rebound nystagmus is correlated with the velocity decay of gaze-holding nystagmus, rebound nystagmus may be explained as gaze-holding nystagmus relative to a new set point with the lowest eye drift.50 Thus, it might be easier to detect rebound nystagmus when the patient is instructed to look at a target at an eccentric position (e.g., 10°) opposite to the prior eccentric gaze.50

Positional nystagmus
Even though CPN is much less common than BPPV, it is important to distinguish these two conditions.51 The Bow and
Head-shaking nystagmus

Head shaking in the horizontal plane at 2 to 3 Hz for 10 to 20 s may induce nystagmus in central as well as peripheral vestibular lesions. Head-shaking nystagmus (HSN) is also frequently found in patients with HC-BPPV, with the nystagmus mostly beating to the lesion side in geotropic HC-BPPV and to the healthy side in apogeotropic HC-BPPV. HSN in BPPV may be explained by altered endolymph or cupular dynamics due to otolithic debris in the semicircular canal.
peripheral or central vestibulopathy, and it may be vigorous. Given that the time constant of HSN in these patients (=12 s) is larger than in those with VN, VM, and Meniere's disease, this disorder appears to represent a distinct entity. This may be due to a hyperactive and asymmetric velocity-storage mechanism that gives rise to intermittent attacks of spontaneous vertigo, especially when the marginal compensation of the underlying pathology is disrupted by endogenous or exogenous factors.

**Vibration-induced nystagmus**

Skull vibration stimulates both the otolith and canal structures, and instantaneously triggers nystagmus by inducing or augmenting a preexistent vestibular asymmetry. Vibration-induced nystagmus may be observed in up to 20% of healthy controls, and may be useful for screening children with hearing loss for vestibular asymmetry when combined with other tests of vestibular function.

**Other types of triggered nystagmus**

Hyperventilation instantaneously increases the randomness of the waveforms, but with only a small change in the intensity of nystagmus. The increase in waveform randomness can have detrimental effects on function. Sound- or pressure-induced horizontal nystagmus is a rare finding in bilateral vestibular paresis, but associated elevations of the summation of the waveforms, but with only a small change in the intensity of nystagmus. The increase in waveform randomness can have detrimental effects on function. Sound- or pressure-induced horizontal nystagmus is a rare finding in bilateral vestibular paresis, but associated elevations of the summation of the waveforms, but with only a small change in the intensity of nystagmus. The increase in waveform randomness can have detrimental effects on function.

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In conclusion, careful evaluation of nystagmus and saccadic intrusions/oscillations would provide valuable information on the lesion location and the involved pathomechanism. Recent advances in quantitative evaluations of eye movements using oculography, computational model simulations, genetics, and imaging technologies have markedly improved our understanding of the pathophysiology of involuntary eye movements, as well as their diagnosis and management. Patient-initiated capture of eye movements, especially when paroxysmal, and the online transfer of these data to clinicians would further enhance the ability to diagnose involuntary eye movements.

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