Current Treatment of Nystagmus

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Opinion statement

Acquired and congenital nystagmus often causes decreased visual acuity as a direct result of the inability to maintain stable foveal vision. In addition, acquired nystagmus causes a disabling subjective sensation of movement of the visual world called oscillopsia. The eye movements themselves do not require treatment if the patient is asymptomatic. However, therapy is necessary if visual disability is present. Treatments based in pharmacologic mechanisms are preferred. There are few controlled treatment trials and therapeutic efficacy generally is sought in a trial and error approach, depending on the type of nystagmus present. Treatment with 3,4-diaminopyridine and 4-aminopyridine recently have been shown to be effective for downbeat nystagmus. Gabapentin, baclofen, and clonazepam also are useful in some patients with downbeat nystagmus. Baclofen is the therapy of choice for periodic alternating nystagmus. Gabapentin often is effective for acquired pendular nystagmus. Clonazepam and valproate also may be effective for acquired pendular nystagmus. Memantine now is available in the United States and is promising in the treatment of pendular nystagmus. Optical devices that negate the negative effects of nystagmus continue to undergo development research. These and other medical, surgical, and optical devices are potentially useful alone or in combination with other therapies.

Introduction

Advances in understanding of the neural networks involved in eye movement control and the consequences of disruption of these networks are fostering a transition from nystagmus treatments based on phenomenology to nystagmus treatments based on pathophysiologic mechanisms [1,2]. Pathologic nystagmus causes deterioration of visual acuity because of instability of retinal images and movement of images from the fovea to the peripheral retina. A second effect is oscillopsia, a sensation of movement of the visual world. To determine appropriate treatment for visual disability caused by nystagmus, accurate characterization of the nystagmus is essential.

It should be determined if the nystagmus is congenital or acquired. Quantitative eye movement recordings with the magnetic scleral search coil to clarify the waveform of the slow phases of the nystagmus may be necessary to determine this and to accurately characterize the type of nystagmus. Congenital nystagmus typically has an increasing velocity slow-phase waveform, in contrast to acquired nystagmus, which generally has a linear or decreasing velocity slow phase waveform (Fig. 1). Although patients with congenital nystagmus often have decreased visual acuity and may require treatment, acquired nystagmus results in greater patient disability as a consequence of oscillopsia and treatment options differ for congenital and acquired nystagmus. The presence of foveation periods during which the eye is still and directed toward the object of regard in congenital nystagmus allows for intermittent stabilization of the retinal image, permitting clear vision, and a null zone often is present in which the foveation periods are longer and nystagmus is minimal. Manipulation of this null zone is the premise of several surgical treatments for congenital nystagmus.

Acquired nystagmus should be characterized as jerk or pendular. Jerk nystagmus is defined by phases of unequal velocity (a slow phase and a fast phase) and
pendular nystagmus by phases of equal velocity. With jerk nystagmus, the underlying mechanism generating the abnormal eye movement results in an abnormal slow drift of the eyes away from the desired position (slow phase) and is followed by a rapid, corrective movement in the opposite direction (fast phase). Although the slow phase is the underlying abnormality, jerk nystagmus is named by the direction of its fast phase (slow drift of the eyes up followed by fast corrective movements down is downbeat nystagmus). If jerk, is the nystagmus right-beating, left-beating, torsional, downbeat, upbeat, or periodic alternating? Determination of the necessity of therapeutic intervention for nystagmus should include analysis of functional disability, whether or not oscillopsia is present, and visual acuity measurements. Ideally, scleral search coil measurements should be done before and after treatment for the most precise analysis of therapeutic response.

The goals of nystagmus treatment are to minimize oscillopsia and maximize visual acuity. In certain instances, this may be achieved by treating the underlying condition (treating epileptic nystagmus with antiepileptic medications or withdrawing a pharmacologic agent that is causing nystagmus); but more often, therapies directly aimed at improving vision are needed. Pharmacologic, surgical, and ocular device interventions are available. Unfortunately, few trials exist directly comparing the efficacy of the interventions and most evidence of efficacy for any given treatment arises from single case reports or small case series [3].

## Treatment of peripheral vestibular nystagmus

- Most nystagmus from acute peripheral vestibular impairment such as acute labyrinthitis or benign paroxysmal positional vertigo spontaneously resolves as central vestibular mechanisms compensate for the imbalance in the peripheral vestibular system [4]. However, if oscillopsia or the accompanying symptoms of vertigo are significantly debilitating, acute management with pharmacologic agents may be beneficial.

### Pharmacologic treatment

- In general, pharmacologic treatment should be used only for up to 48 hours to prevent delay in central compensation [2].

#### Diphenhydramine

- **Standard dosage**: 25 to 50 mg oral dose every 4 to 6 hours.
- **Contraindications**: Hypersensitivity to antihistamines.
- **Main drug interactions**: None.
- **Main side effects**: Somnolence, dizziness, dry mouth.
- **Special points**: None.

#### Promethazine

- **Standard dosage**: 12.5 to 25 mg oral dose every 4 to 6 hours.
- **Contraindications**: Hypersensitivity to antihistamines. Use caution in patients with narrow-angle glaucoma, prostatic hypertrophy, and liver disease.
- **Main drug interactions**: Central nervous system depressants and anticholinergic drugs.