CASE REPORTS

Congenital nystagmus: rebound phenomenon following removal of contact lenses

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Abstract

Symptoms resulting from congenital nystagmus can be significantly reduced by wearing corneal contact lenses. A 90 minute therapeutic trial with contact lenses was performed on a 20-year-old affected patient and produced a beneficial effect. Upon removal of the lenses however the patient showed a transient rebound phenomenon with oscillopsia lasting about 20 minutes. This phenomenon, although it might be expected in theory, has apparently not previously been observed either because it is rare or because in most patients it is not clinically apparent. The purpose of this report is not to discourage treating patients with congenital nystagmus by means of contact lenses, but rather to draw attention to the occasional occurrence of such a rebound phenomenon and to discuss its theoretical significance.


It is well established that alteration in visual function resulting from congenital nystagmus can be reduced by fitting corneal contact lenses. We performed a successful therapeutic trial on a 20-year-old affected woman. When the contact lenses were removed however the patient experienced transient oscillopsia. To our knowledge such a rebound phenomenon has not previously been reported in this condition.

Discussion

In individuals with congenital nystagmus vision remains close to normal unless additional abnormalities, such as albinism, are present. However visual function can be disturbed when the nystagmus is increased as a result of anxiety or fixation effort – for example, with poor ambient illumination or when attempting to fixate using lateral gaze because of an eccentrical null point. Rarely alteration in vision of affected patients presents as oscillopsia. This has been reported to occur in various circumstances – for example, while artificially stabilising images upon the retina. Changes in nystagmus waveform might account for the occurrence of oscillopsia in these individuals.

Numerous techniques have been suggested for improving the visual capabilities of affected patients. These include prisms, surgery, auditory biofeedback, and corneal contact lenses. Currently there is a strong trend towards attempting to treat such patients with contact lenses, and these often result in dampening of the congenital nystagmus. This phenomenon might be due to sensory feedback from movement of the edge of the lens against the inside of the lids, since topical anaesthesia was shown to result in suppression of the beneficial effect of contact lenses in this condition.

When first fitted with contact lenses our patient experienced relief from her visual disturbance. Upon removal of the lenses she showed a transient episode of oscillopsia. This pattern of events strongly suggests a rebound phenomenon, which is a common event occurring when a factor influencing the biological balance is suppressed, and results from the dynamic nature of the homeostatic balance process.

Rebound phenomena have been reported in normal individuals, including in ocular motor
control mechanisms, as exemplified by the observation of rebound nystagmus in healthy subjects. Rebound phenomena are amplified in certain conditions such as cerebellar dysfunction, as demonstrated by the Holmes rebound test, or by the occurrence of marked rebound nystagmus in patients with cerebellar disorders.

Thus, in our patient, relieving the dampening effect of contact lenses on congenital nystagmus might have induced a rebound phenomenon consisting of a transient increase in nystagmus intensity and/or a change in the foveation period. The observation of such events in our patient, but not in other treated patients, was probably due to the rarity of the phenomenon. However, the phenomenon may in fact be common, but usually occurs with minimal intensity and thus escapes notice. This appears to happen in the case of rebound nystagmus occurring in normal subjects. With congenital nystagmus intra-individual variation in the intensity of rebound phenomena may be due to a number of factors, including psychological ones, especially as anxiety has been shown to influence the amplitude of congenital nystagmus.

Other possible causes of the induction of oscillopsia in our patient were also considered, including those related to refractive changes or behavioural disorders. However they were not considered plausible. Indeed, changes in nystagmus did not result from an increase in fixation effort due to alteration in refractive correction because the contact lenses used in this patient were plano. The possibility that the patient’s complaints were not organic in nature cannot be excluded, but there was no indication to favour such a hypothesis. If the patient was hysterical in character this would more probably have shown up while she was wearing contact lenses not after their removal.

Recording the eye movements both before and immediately after removal of contact lenses might have provided additional information. However our patient’s refusal to undergo further attempts at treating the nystagmus with contact lenses precluded such an evaluation. In any event the variation which usually occurs during the recording of congenital nystagmus in relation to anxiety or fixation efforts, as well as the transient nature of the rebound, may make it difficult to record such a phenomenon.

Our report is not intended to discourage clinicians from attempting to treat patients suffering from congenital nystagmus with contact lenses since this treatment appears to be effective in many cases. Moreover, the rebound phenomenon which we describe is apparently rare and, when it occurs, is not a serious or long-lasting side-effect of the treatment. Our purpose is rather to draw attention to the possible occurrence of rebound in congenital nystagmus following removal of contact lenses, and to its theoretical significance.

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