

HYPERPHORIA AND CYCLOPHORIA

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Hyperphoria is a potential deviation of one eye upwards that becomes an actual deviation when the two eyes are dissociated and recovers when the dissociating factors are removed. In hypophoria, the deviation is downwards and, as hypophoria of one eye may be regarded as the same as hyperphoria of the other, the term 'hypophoria' is not in general use. Right hyperphoria is the same as left hypophoria. Occasionally, vertical heterophoria occurs in one eye only, which is usually found to be amblyopic.



Secondary hyperphoria

Aetiology

Hyperphoria is often present as a secondary condition and the primary causes should be considered before treating the hyperphoria. It may be secondary to the following.

Horizontal heterophoria

High degrees of comitant esophoria or exophoria are often accompanied by a small vertical component. In these cases, the treatment will be that which is appropriate to the primary condition but prism relief of the hyperphoria may help.

Incomitant deviations

Paretic conditions involving the elevator or depressor muscles may begin as hyperphoria and develop later into strabismus. It is important that this early sign of pathology should be detected. The sudden onset of intermittent vertical diplopia and/or other symptoms and the incomitant nature of the deviation, are the main diagnostic features. The most common cyclovertical incomitancy is a superior oblique underaction, so it is important to look carefully for cyclophoria in all cases of hyperphoria (Ch. 17). Congenital incomitant deviations are also frequently accompanied by a vertical element but symptoms are usually absent.



Unilateral high myopia (heavy eye syndrome)

Heavy eye syndrome involves anisometropia, usually with high myopia, and hyperphoria or hypertropia. The more myopic eye is hypotropic or hypophoric. The notion that the disorder results from a 'heavy' myopic eye is incorrect: the cause is an abnormally low muscle path of the lateral rectus in the involved eye (Yanoff & Duker 1999). The vertical deviation ranges from 2–25 Δ , although no association occurs between the amount of anisometropia and the amount of hypotropia. Elevation of the low eye may be limited. Frequently the head tilts to the side of the hypotropic eye, which may be compensatory to achieve single vision by the creation a of base-up prism effect before the hypotropic eye.

Tilted spectacles and anisometropia

If spectacles are incorrectly fitted or the frame becomes bent, a vertical prism element may be introduced, which will initially show as hyperphoria. Usually, the patient will adapt quite quickly to this abnormal prism and the hyperphoria will no longer be apparent. When this occurs, hyperphoria will be present when the glasses are removed or the spectacle frame is straightened. This will disappear after a few days.

Corrections for anisometropia may also produce hyperphoria when the eyes are not looking through the optical centres. Again, adaptation to this variable prismatic effect will usually occur after a few days of using the anisometropic correction but difficulties can arise with a correction for marked anisometropia, particularly where no glasses have been worn before (Ch. 11). Similarly, problems may arise if a refractive correction has changed markedly (e.g. after a cataract extraction operation).

A spectacle correction that has not been correctly balanced between the two eyes may also cause hyperphoria. The same applies to uncorrected anisometropia.

Primary hyperphoria

Primary hyperphoria is usually considered to be largely due to slight anatomical misalignments of the eyes and/or orbits or muscle insertions for which there is a physiological compensation. Usually, this type of hyperphoria is less than 3Δ , and it seldom causes symptoms. It has been shown that about 98% of symptom-free people will show some degree of hyperphoria after a period of prolonged occlusion of one eye, but this disappears after a few hours when the binocular vision is restored (Duke-Elder 1973, p 551). Vertical heterophoria is not associated with the convergence system in the way that applies to horizontal heterophoria, and this further suggests that anatomical factors play a larger part in its aetiology.

However, decompensation can occur in hyperphoria due to stress on the visual system or on the general wellbeing of the patient (Ch. 4).

Investigation

A routine eye examination should be carried out. The following points may be particularly useful in hyperphoria:

- (1) Symptoms, which can sometimes be very marked in hyperphoria, even where the degree of the heterophoria is low. They occur more frequently in middle age. Frontal headache, ocular discomfort or pain and blepharitis are the most common symptoms. Sometimes there is an anomalous head position, and other patients may report that vision is more comfortable if one eye is closed or occluded.
- (2) Motility test for incomitancy, which should always be undertaken with objective observation of the eyes and also noting the subjective response of the patient reporting any incomitant diplopia (see Ch. 2 for the routine and Ch. 17 for the diagnosis of incomitant deviations). If the clinical results suggest an incomitancy of recent onset, then the patient should be referred.
- (3) Refraction, which should give particular attention to the binocular balance of the spherical error between the two eyes. An unbalanced correction can sometimes be the cause of hyperphoria.
- (4) Compensation assessment, which should be made as described in Chapter 4. The cover test and fixation disparity tests will prove useful in making this assessment for hyperphoria.

Management

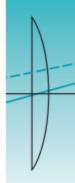
Removal of cause of decompensation

Care must be taken to explore the visual working conditions and any stress or ill-health that may be the cause of the decompensation. These should receive attention before other aspects of management.

Refractive correction

In some cases, the provision of a correction for previously uncorrected refractive error will alleviate the hyperphoria without any other treatment. Balancing the refractive correction is very important in hyperphoria.

In the case of marked anisometropia where no previous correction has been worn, a partial correction of the more hypermetropic eye may prevent disturbance by vertical prismatic effects when the patient is not looking through the optical centres of the lenses. The correction is reduced in the more hypermetropic eye until the vertical heterophoria is compensated when looking through the lenses a little above or below the optical centres. This can be judged by Turville's 'nodding test'. Traditionally the infinity balance septum is used and the patient is asked to raise and lower the head in a slight nodding motion until the reduced sphere does not create a change of level in the two letters. Nowadays, it is more common to carry out a version of this test with the patient slowly nodding while viewing the vertical Mallett fixation disparity test. This correction may be increased to a fuller prescription with subsequent glasses (see also Ch. 11).





Eye exercises

Orthoptic exercises to improve the vertical fusional reserves very seldom prove successful and do not seem to help in making the hyperphoria compensated. This is not surprising since vertical vergence may not be disparity-driven (Ygge 2000) and, unlike horizontal vergence, is not influenced by making an effort to track vergence changes (Stevenson et al 1997). However, one study (a non-controlled trial) indicated that it might be possible to change vertical fusional reserves with exercises (Luu et al 2000). When the hyperphoria is associated with horizontal heterophoria, orthoptic exercises to increase the horizontal fusional reserves will often result in the vertical heterophoria becoming compensated (Cooper 1988a).

Relieving prisms

Most primary hyperphoria can be readily relieved by weak vertical prisms. As explained above, the smallest prism that will neutralize the fixation disparity with a Mallett unit can be prescribed. Such vertical prism relief may also help any decompensated horizontal heterophoria (Sheard 1923; see Ch. 8).

Referral

Incomitant hyperphoria with intermittent diplopia of recent onset indicates the need for medical investigation. When there is a high degree of hyperphoria and congenital incomitancy that gives rise to intolerable symptoms, surgical relief is sometimes considered. Medical advice should be sought.

Dissociated vertical deviation

Dissociated vertical deviation (DVD) is a comparatively unusual anomaly that is also known as 'alternating sursumduction'. Although it could be mistaken for hyperphoria, the clinical appearance is not the same. It is usually seen during the cover test. When one eye is covered with an occluder or a dark filter it slowly deviates upwards, possibly by as much as $40~\Delta$. This differs from hyperphoria in that, whichever eye is covered, there is an *upward* movement of the eye behind the cover. When the cover is removed, the eye slowly recovers to the fixation position. The upward movement is not always equal in the two eyes and sometimes it can be absent in one eye, giving the appearance of a 'unilateral hyperphoria'. In all cases, if a neutral density filter bar is placed before the *uncovered* eye and the density of the filter is increased, the eye under the cover will slowly move down; when the density of filter is reduced, the covered eye moves slowly up again – the Bielschowsky phenomenon.

DVD is usually associated with a history of early onset esotropia. There is sometimes a cyclorotation of the occluded eye (Burian & von Noorden 1974, p 320).

When DVD exists without any other deviation or anomaly, there are usually no symptoms and no independent treatment is required. If it exists with other conditions, treatment appropriate to the primary condition can

be considered. Occasionally, patients with DVD complain that one eye deviates spontaneously and that this is noticed by other people. The condition rarely produces symptoms (Mallett 1988a) but if the condition is cosmetically unacceptable surgery is indicated (Kanski 1994).

Cyclophoria

It is doubtful if cyclophoria exists as a primary condition not associated with incomitant deviations. Many patients with long-standing cyclodeviations are asymptomatic because of sensory adaptations (von Noorden 1996, p 370).

The conventional view is that there is no motor cyclovergence (Kertesz & Jones 1970) and that cyclodeviations are mostly cyclotropia so that the differentiation between cyclophoria and cyclotropia is difficult to justify (von Noorden 1996, p 127). However, when measured with large field stimuli, 8° of motor cyclovergence has been demonstrated in normal subjects, who can also exhibit 8° of sensory cyclofusion, allowing the fusion of up to 16° of cyclodisparity (Phillips & Hunter 1999). Hence, in heterophoric patients a double Maddox rod test (p 286), which dissociates the eyes, will reveal more cyclodeviation than an associated test (e.g. Mallett fixation disparity test or double Bagolini lenses; p 287). Patients with cyclodeviations that had an onset in the first 6 years of life may develop torsional HARC (Ch. 12) and it has been suggested that this might prevent subjective torsion from being detectable even on dissociation tests, like the double Maddox rod test (Phillips & Hunter 1999).

Clinical Key Points

- Vertical heterophoria is very rarely unilateral: right hyperphoria is usually the same as left hypophoria
- Significant hyperphoria is likely to produce symptoms, particularly if there is a recent onset when vertical diplopia is likely
- Recent onset hyperphoria often results from an incomitancy which may indicate active pathology and requires referral
- The most common pathological cause of hyperphoria is superior oblique muscle palsy so cyclotorsional tests should be carried out
- Other causes of decompensated hyperphoria include an old hyperphoria decompensating and inappropriately prescribed or fitted spectacles
- Hyperphoria responds well to vertical prisms, but not to eye exercises
- Cyclophoria is probably always associated with incomitant deviations, and if these are long-standing then there may be sensory adaptations