Children and Technology: Esotropia because of excessive use of hand held devices.

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**Introduction:**

Since Sue Palmer published her book called *Toxic Childhood in 2006* [1] numerous publications appeared on the dangers associated with excessive use and exposure to hand held devices. Apart from all the social and psychological challenges today’s children are also facing significant physical deprivations. In the vision care domain we are seeing a significant increase in conditions such as near work induced transient myopia (NITM) and also acute acquired comitant esotropia (AACE). In 2016 Lee *et al.* [2]reportedon this unusual presentation of esotropia in children using smartphones for more than four hours per day. In my own practice I have seen 10 children between the ages of two and six years over the last six months presenting with AACE related to the excessive use of hand held devices.

**Aetiology and categorization of AACE:**

Acute acquired comitant esotropia is characterized by a sudden-onset eye misalignment with an equal angle of deviation in all fields of gaze and is generally associated with older children and adults [[3](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR1)]. Its prevalence remains unknown, but it is generally considered rare [4] but from my own experience it is certainly becoming more common. Although many cases are non-neurologic, comitance does not rule out the possibility of a serious, underlying neurological condition. Intracranial tumours, Chiari malformation, hydrocephalus and other neurological diseases may present with comitant esotropia with no clear neurological signs. According to Lee *et al* [2] three main types have been defined and later modified by previous investigators:

 1. Swan type: esotropia due to the disruption of fusion (precipitated by monocular occlusion or loss of vision in one eye);

 2. Burian-Franceschetti type: esotropia characterized by minimal hypermetropia and diplopia, often associated with physical or psychological stress;

 3. Bielschowsky type: esotropia that occurs in adolescents and adults with varying degrees of myopia, and shows equal deviation at distance and near fixation [[5](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR3), [6](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR4)].

The mechanism of Bielschowsky type AACE is thought to be uncorrected myopia with excessive near work (holding printed materials or sewing excessively close to the eye), resulting in an inability to maintain balance between the converging and diverging forces of the eye, and the subsequent development of increased tonus of the medial rectus muscles, leading to esotropia [[3](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR3)]. Other rare types of AACE have also been reported, such as refractive-accommodative type AACE, and AACE associated with accommodative spasm or intracranial diseases [[5](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR5), [6](https://link.springer.com/article/10.1186/s12886-016-0213-5#CR6)]. With AACE becoming more prevalent in earlier childhood, Buch [7] retrospectively reviewed records of all children referred with acute-onset comitant esotropia and diagnosed with AACE between 2000 and 2013 at the Eye Clinic, Rigshospitalet, Copenhagen, Denmark. She expanded the categorization into seven cause-specific types based on occurrence and the following definitions with respect to previous described types:

1. Type I, Swan, Occlusion-related AACE: monocular occlusion was the precipitating event.
2. Type II, Burian-Franceschetti, Idiopathic AACE: non-accommodative esotropia, in the presence of minimal refractive error of <+3 spherical dioptres and fusion. An obvious aetiological factor was lacking; however, this type was eventually precipitated by physical or psychological stress.
3. Type III, Acute accommodative AACE: hyperopia of ≥+3 spherical dioptres and fusion were present and eventually precipitated by physical or psychological stress.
4. Type IV: Decompensated AACE: Decompensated monofixation syndrome was regarded as the cause if the child postoperatively, after realignment of the eyes within 8 PD of orthotropia/esotropia, continued to suppress in the presence of anisometropia of ≥2.5 D and/or positive 4 PD base-out test at follow-up examination. Decompensated esophoria was the cause if the cover test at follow-up examination revealed esophoria after realignment.
5. Type V: Neurological AACE: Associated with intracranial disease.
6. Type VI: Cyclic AACE: Presence of non-accomodative, hypermetropic (<+3 spherical dioptres), normosensorial and idiopathic esotropia alternating with binocular single vision with gradually shorter intervals.
7. Type VII, Secondary AACE: Presence of different aetiologic disease entity, for example optic neuritis or ataxia telangiectasia.

Acute acquired comitant esotropia is distinct from common forms of childhood esotropia, such as infantile esotropia and accommodative esotropia, in the rapid tempo and typically later timing of onset; further, AACE is distinct from restrictive or paretic strabismus, which usually results in an incomitant angle of deviation that varies with the direction of gaze. As can be seen from the before mentioned categorizations, the underlying etiologies for AACE are broad but, in some cases, it may be associated with significant neurologic disease. It is therefore of paramount importance that any child presenting with AACE be referred to an Ophthalmologist and Neurologist to rule out any neurological disorder or any other disorder or trauma that may impact on the extra ocular muscles.

According to Lee *et al* [2] the children they examined presented with similarities but also significant differences if compared to the different categories described above. They found the children to present with refractive errors that ranged from various degrees of myopia to moderate hyperopia. All patients obtained 6/6 vision with spectacle correction and the esodeviation was similar for distance and near. This rules out the possibility of a Bielschowsky type of AACE. The absence of diplopia in some patients and the presence of only intermittent diplopia at distance fixation in others might, according to the authors, indicate that the dynamic hyper tonus of the medial rectus muscles and resultant development of esotropia progress slowly. They do admit that the exact mechanism by which comitant esotropia develops in myopic or moderately hyperopic patients, without previous manifest deviation, remains unclear and has yet to be determined. They did however found that if the children were refrained from using smartphones for as little as one month, the esodeviation decreased.

Working on video display terminals was shown by several authors [8,9] to induce abnormalities in accommodation and vergence, when compared to ordinary hard copy work. Therefore the authors [2] conclude that it is conceivable that excessive use of hand held devices at close reading distance and the resultant abnormalities in the accommodation and vergence systems of children with low fusional divergence capacity, can lead to dynamic preponderance of the medial rectus muscles, resulting in the development of manifest esotropia. Lee *et al* [2] did unfortunately not obtain any measurement on accommodative function or power. I believe this would have confirmed that this esotropia is the result of excessive strain on the accommodative- convergence system.

**Management of AACE induced by the use of hand held devices:**

From my own experience I have very little doubt that we will experience many more visual abnormalities related to the excessive use of hand held devices. It is not strange any more to see babies sitting with these devices. You can now also buy baby strollers with the bracket for a hand held device attached to it. Exposing children to these devices in the first two years of life when the visual system is still developing neurological and physical pathways will have serious consequences not only on the visual system but also on perceptual and neurological development. Parents must be made aware of the fact that instead of giving their children an advantage as they hope, they may just achieve the opposite. Several countries in the world are now looking at introducing mental health policies to protect their youth against excessive use of hand held devices [10] and are also developing so called Smartphone addition scales (SAS). Managing the time spent on these devices should form the cornerstone of any approach.

We are in desperate need of research to guide us to deal with children already presenting with AACE caused by these devices. Lee *et al* [2] suggested bilateral medial rectus recession as an option but my concern is that once the near point strain is being reduced that this may result in a consecutive exotropia. Since they admitted not investigating any accommodative functions I would like to see research in these areas. I have started to experiment with plus lenses [>1.50] for using on the devices and found that binocular vision was restored in all cases. There has also been a slow reduction in the angle of esotropia. I must stress again that all other possible causes for AACE must be ruled out before trying to restore binocular vision by optical means.

References:

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