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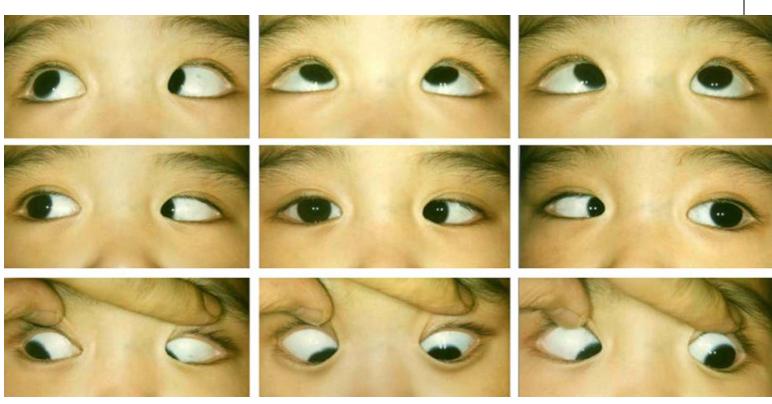


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(Prism-diopters are represented by a delta Δ ; eg, $50\Delta = 50$ prism-diopters)





~50∆ of comitant esotropia



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Huge ET in right gaze

Moderate ET in primary gaze

Almost no ET in left gaze

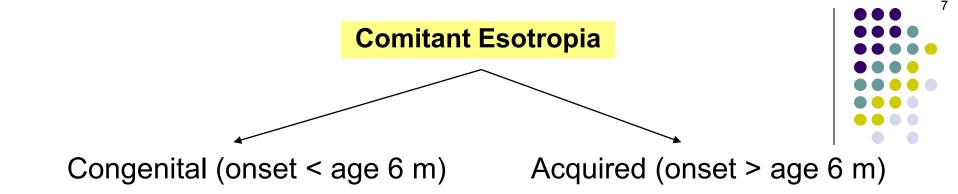
Incomitant ET 2ndry to right CN6 palsy



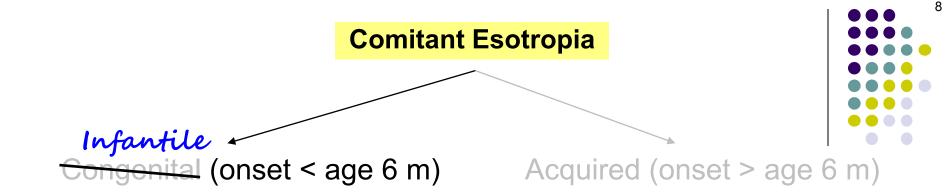
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This slide-set will focus on comitant ET. There is no gender predilection for comitant ET; however, there is a racial one: Incidence in White and Black infants is roughly equal, and both are higher than that for infants of Asian descent.



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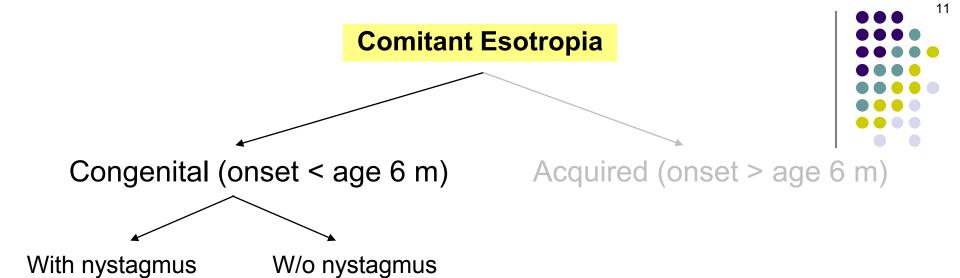
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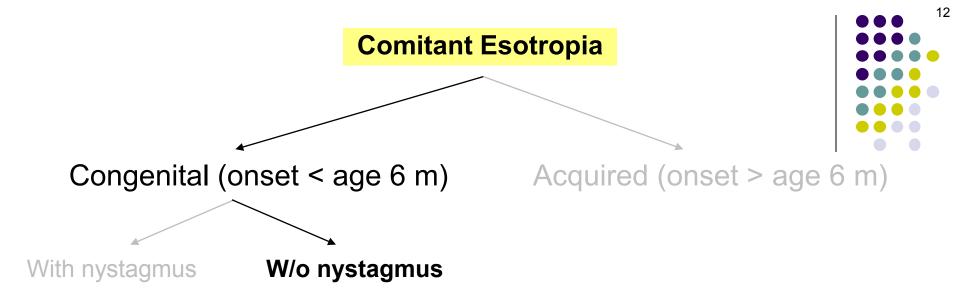
It's important to note here that brief strabismic episodes are commonly seen in the first few (especially two) months of life. Further, it's not uncommon for the same infant to manifest short periods of both ET *and* XT; this is referred to as *ocular instability of infancy*.

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It's important to note here that brief strabismic episodes are commonly seen in the first few (especially two) months of life. Further, it's not uncommon for the same infant to manifest short periods of both ET and XT; this is referred to as ocular instability of infancy. However, if the ET is 1) present after age 2 months; 2) constant; and/or 3) large, it probably represents a congenital ET.



We divvy congenital ET into two groups: Those presenting *with* nystagmus, and those presenting *without* it

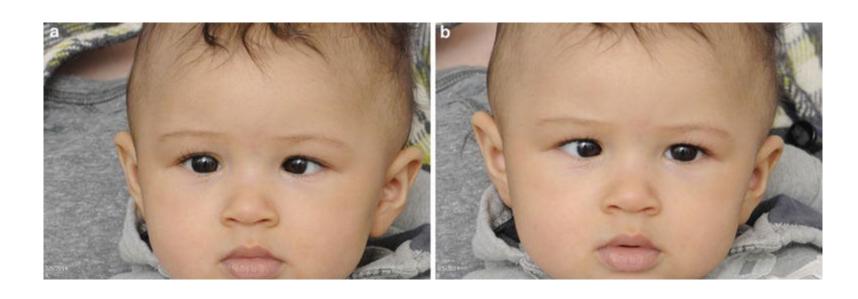


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ET with cross-fixation

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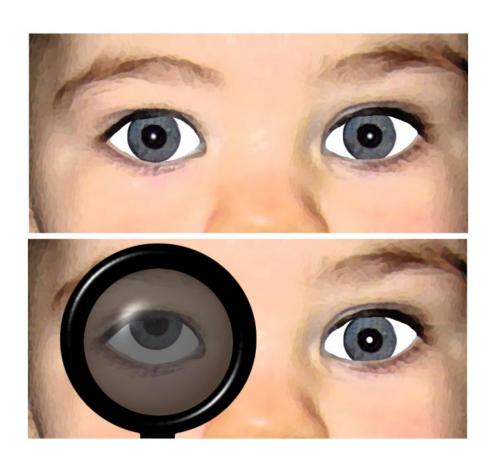
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Another common finding in these infants is *dissociated vertical deviation* (DVD), the phenomenon in which one eye slowly elevates and extorts. This may occur spontaneously (*manifest* DVD), or only when the eye is occluded (*latent* DVD).





(Latent) DVD





For more on DVD, see slide-set P7 (see also the set concerning Sherrington's and Hering's law, FELT3)

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Management of congenital ET w/o nystagmus is usually surgical via bilateral medial rectus recession. It is generally agreed that surgery should occur before the child's 2nd birthday if possible. High-grade stereopsis is not a realistic outcome to expect; rather, *monofixation syndrome* is the hope-for outcome.

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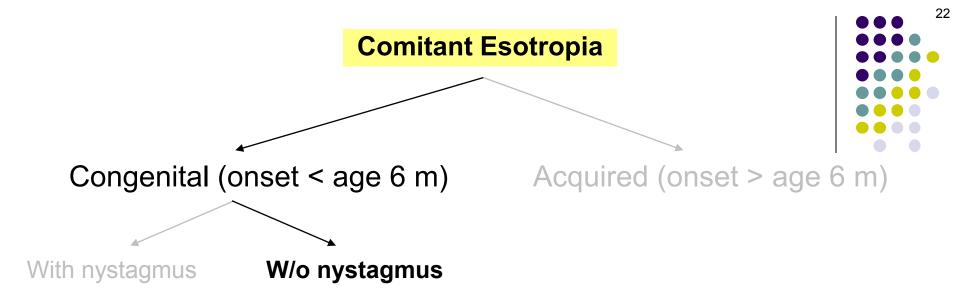
In order to both prevent diplopia and provide some degree of binocular cooperation, the immature visual system responds to strabismus with some combination of three adaptations:

Suppression

Anomalous retinal correspondence (ARC)

Monofixation syndrome

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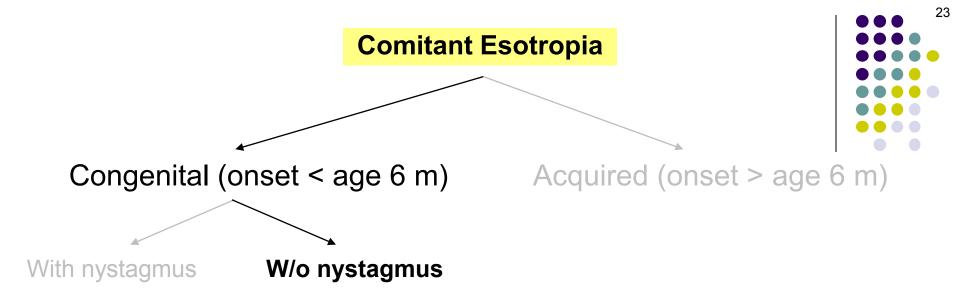
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Monofixation syndrome

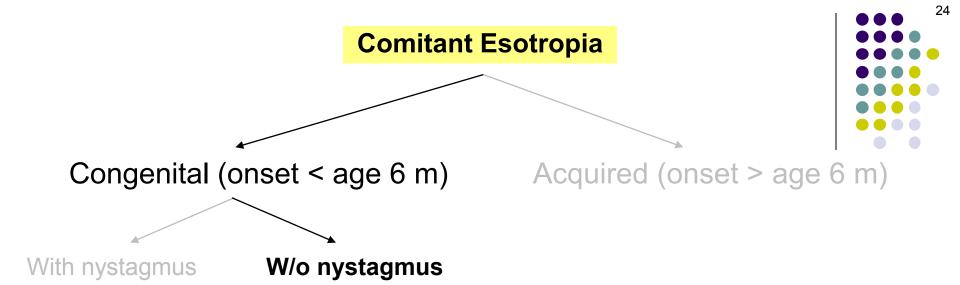
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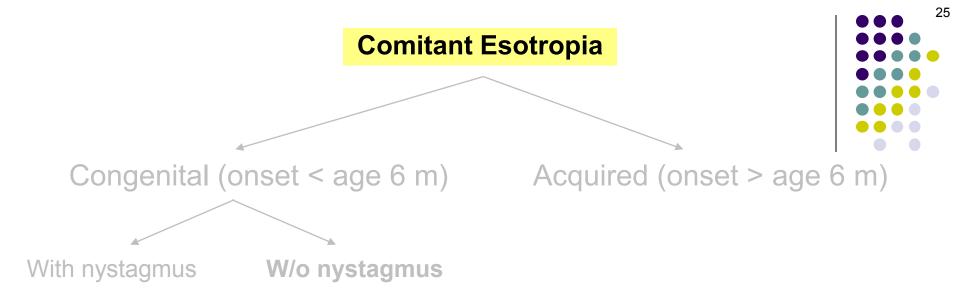
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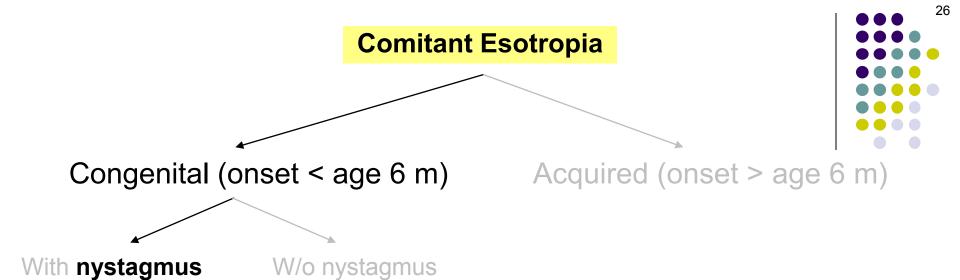
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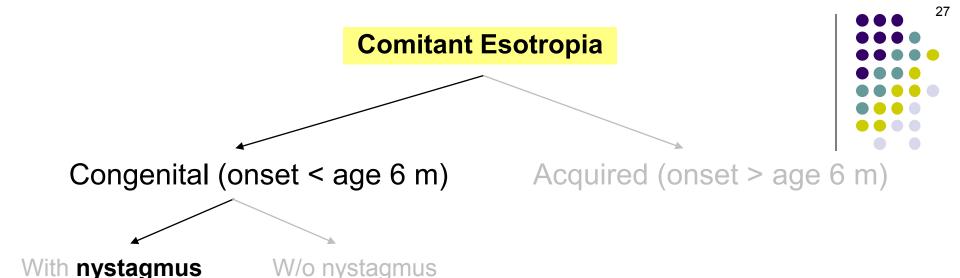
For more on sensory adaptations to strabismus, see slide-set P14

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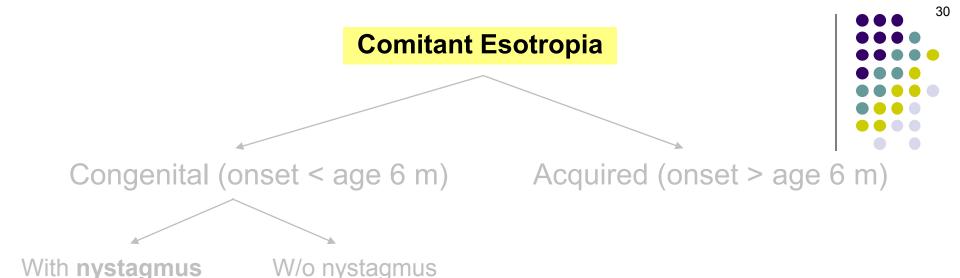


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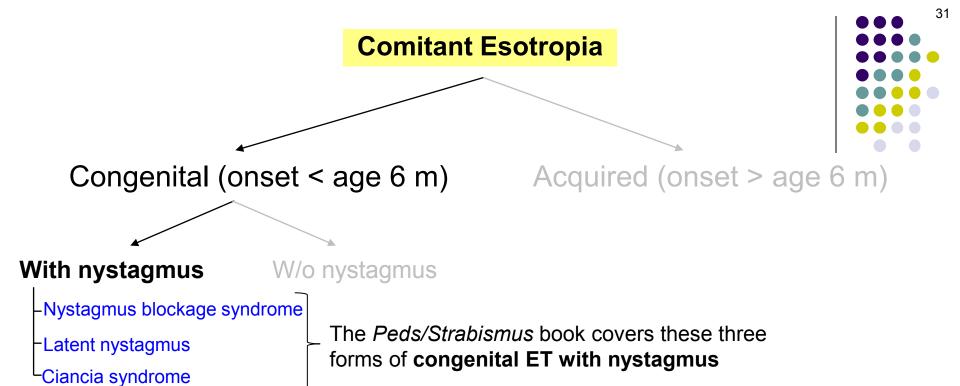
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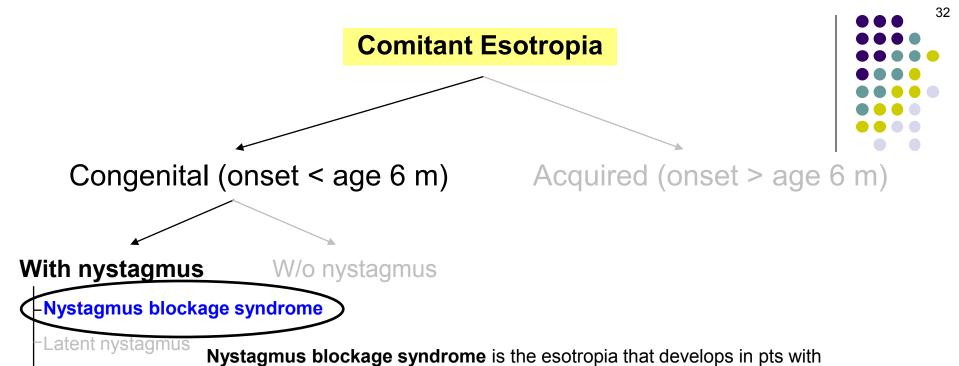


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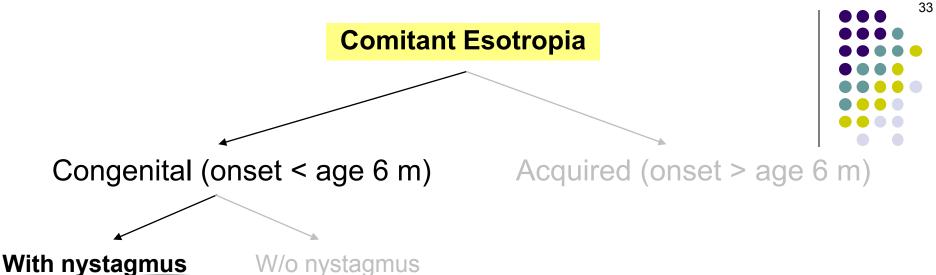
The oscillation of the eyes. The oscillation





congenital motor nystagmus (CMN).

Ciancia syndrome

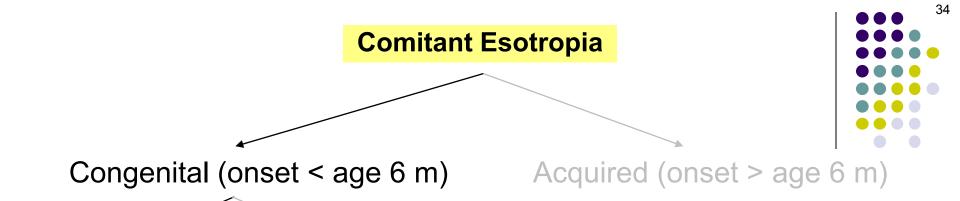


-Nystagmus blockage syndrome

Latent nystagmus

-Ciancia syndrome

Nystagmus blockage syndrome is the esotropia that develops in pts with congenital motor nystagmus (CMN). CMN is a form of nystagmus that arises in the first few months of life. Unlike most forms of nystagmus, it is not secondary to either vision loss or CNS pathology— it just kinda 'is.'



With nystagmus

W/o nystagmus

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Congenital (onset < age 6 m)

Acquired (onset > age 6 m)

With nystagmus

W/o nystagmus

-Nystagmus blockage syndrome

Latent nystagmus

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So why does CMN lead to esotropia? Because early in life, the visual system 'realizes' that the nystagmus is minimized (and thus acuity is maximized) when the eyes are converged.

With nystagmus

W/o nystagmus

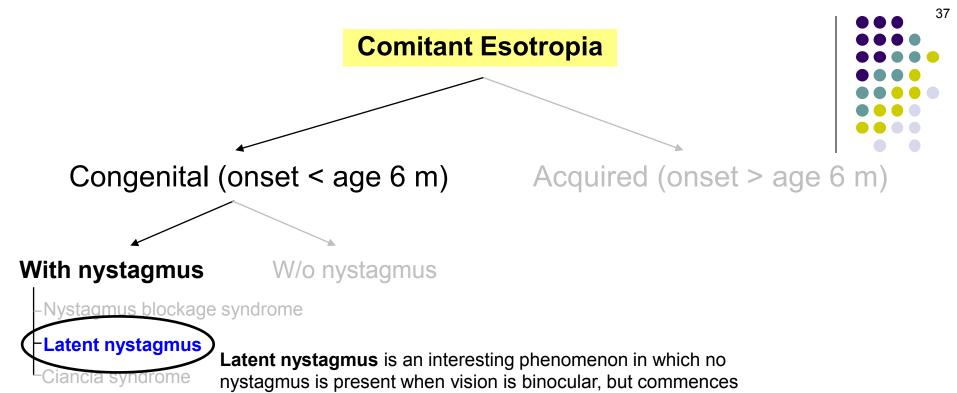
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So why does CMN lead to esotropia? Because early in life, the visual system 'realizes' that the nystagmus is minimized (and thus acuity is maximized) when the eyes are converged. Because of this, the system learns to adopt and maintain an esotropic posture.



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With nystagmus

W/o nystagmus

-Nystagmus blockage syndrome

Latent nystagmus

Ciancia syndrome

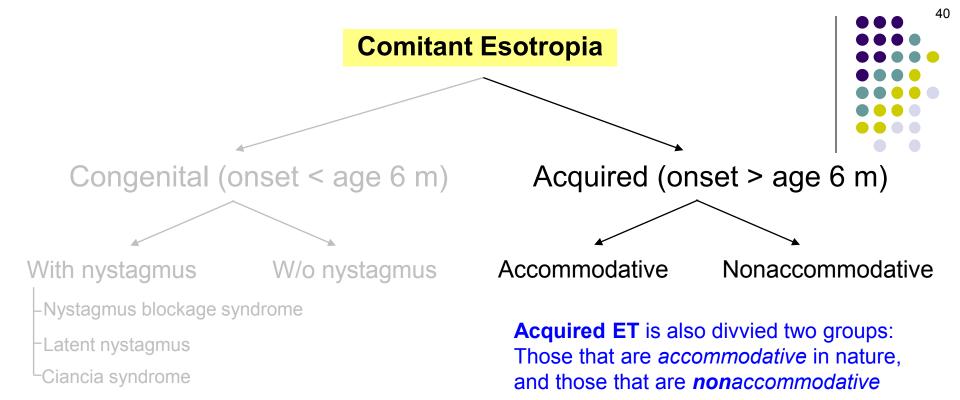
Ciancia syndrome has two features that give it away: The first is, the ET is very large—usually 50Δ or more. Second, the nystagmus intensifies if the pt attempts to abduct either eye.

Comitant Esotropia





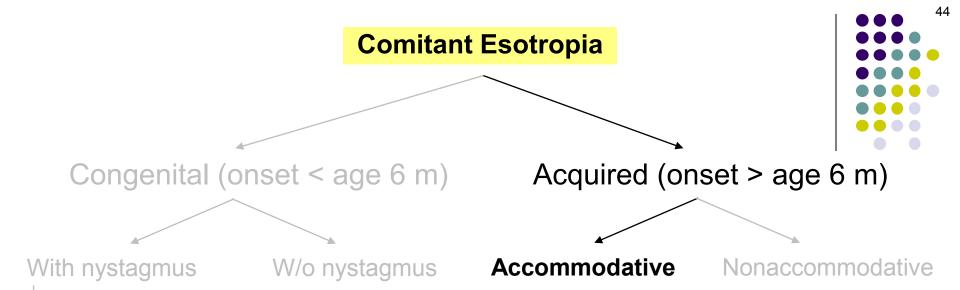
Ciancia syndrome



Accommodative esotropia refers to ET stemming from activation of the *accommodative* (aka *near*) *reflex*. Recall that the accommodative reflex has three components: *accommodation*, *miosis*, and *convergence*. Recall further that *accommodation* refers to the cranking in of lenticular 'plus' power to overcome hyperopic blur.

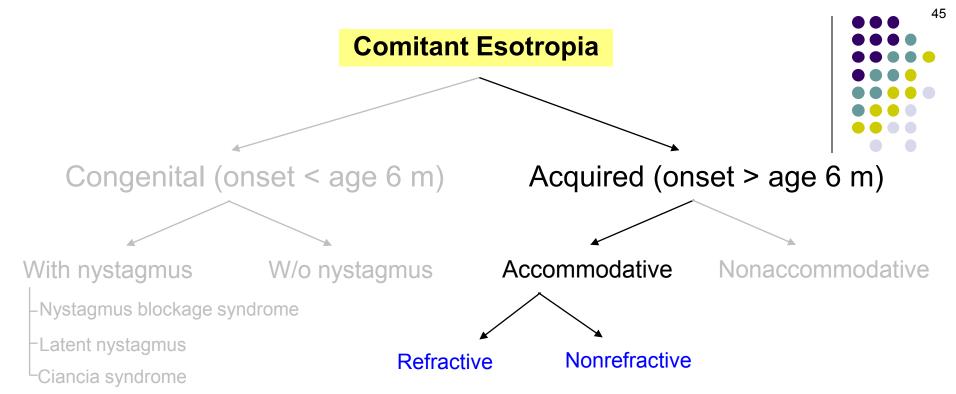
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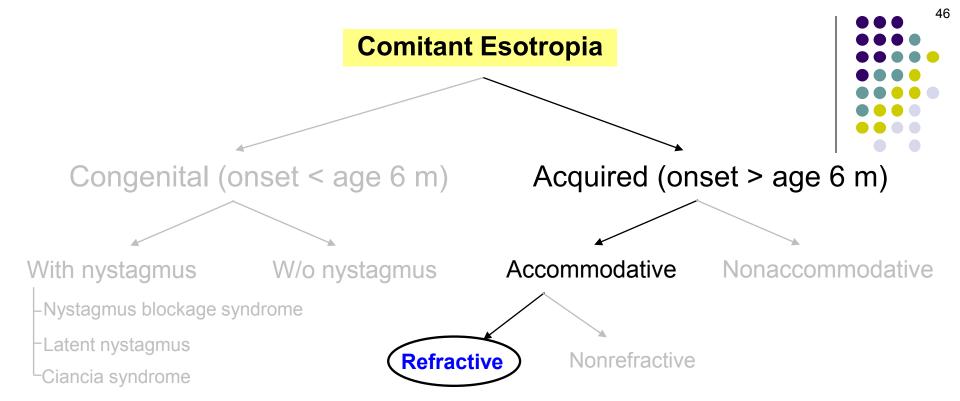


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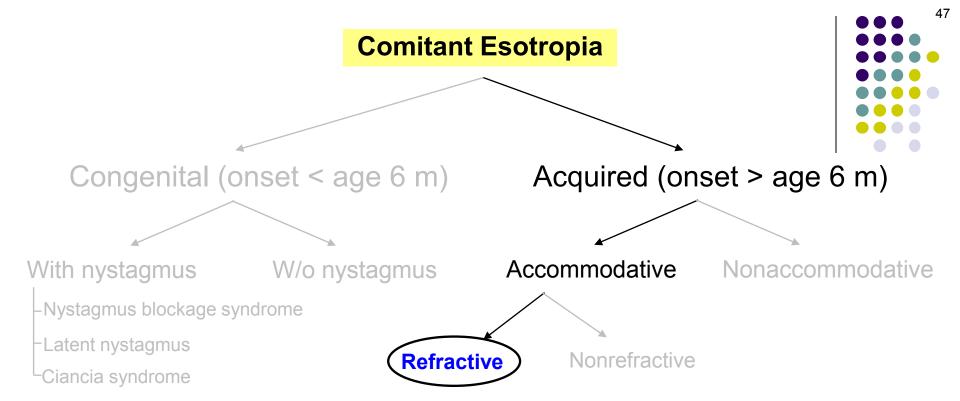
Accommodative ET typically arises around age 2-3 years. It is intermittent initially, eventually progressing to become constant. The child will often complain of diplopia at first, but stops after developing a facultative suppression scotoma. (A *facultative* suppression scotoma is one that is active only while the eye is deviated.) Amblyopia is common.



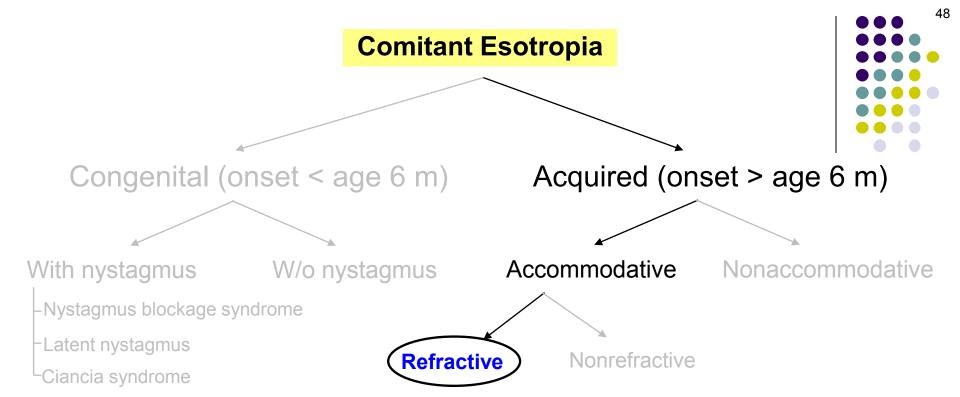
Accommodative ET is further divvied into two forms: *Refractive*, and *Nonrefractive*



Refractive accommodative esotropia is pretty straightforward: It is due primarily to being a high hyperope (average ~4D). For these kids, the severe accommodative exertion required to overcome their hyperopia stimulates so much convergence (via the near reflex) that their divergence inputs get swamped, and their eyes turn in.

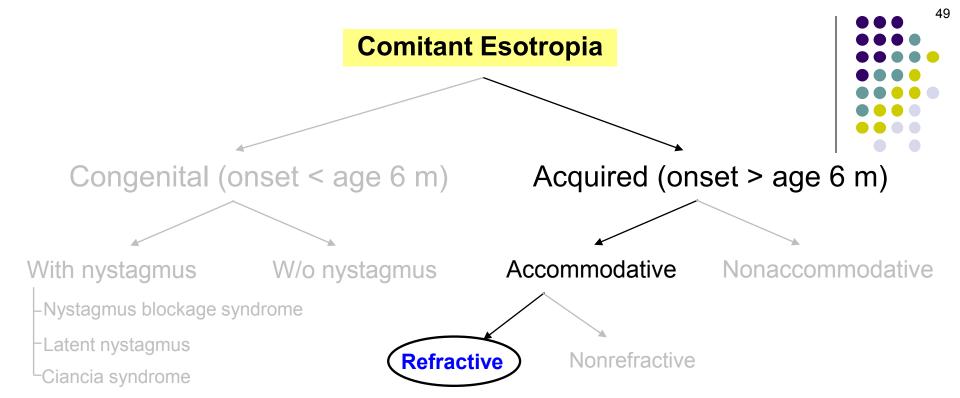


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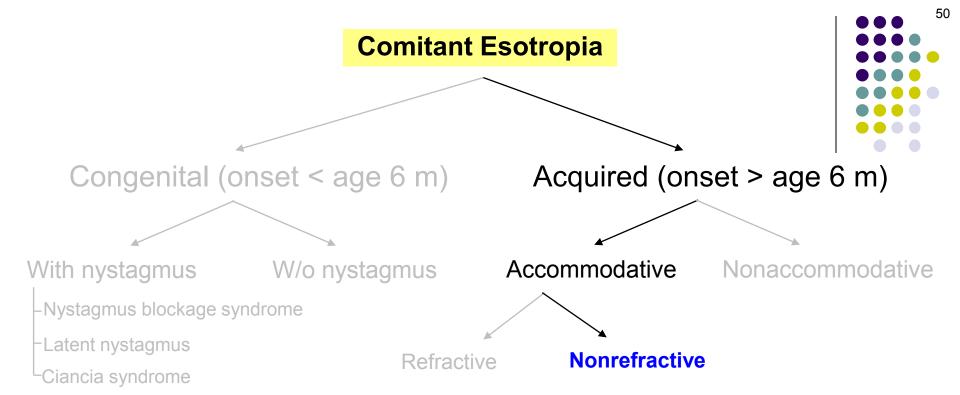
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Refractive accommodative ET is managed by prescribing the full cycloplegic refraction, which should eliminate the accommodation \rightarrow convergence \rightarrow ET chain at the source. If residual ET' is present, a bifocal should be employed.



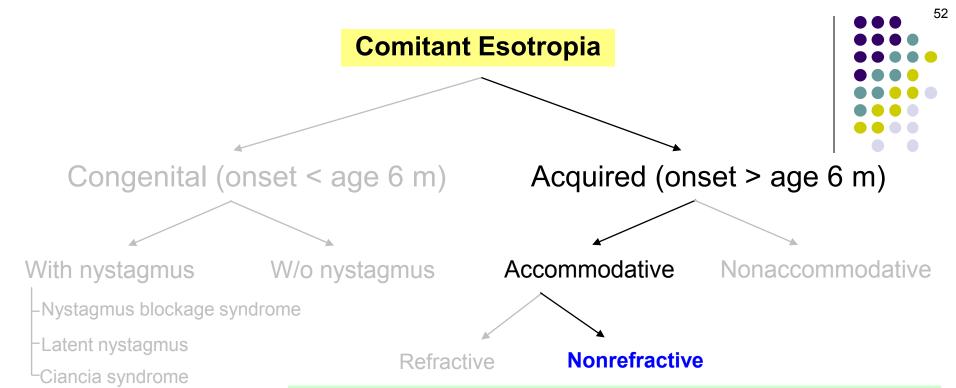
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The mechanism underlying *nonrefractive* accommodative esotropia is less straightforward. As in its refractive cousin, nonrefractive accommodative esotropia is secondary to convergence induced by accommodation for which divergence inputs are insufficient to keep the eyes straight.

The mechanism underlying *nonrefractive* accommodative esotropia is less straightforward. As in its refractive cousin, nonrefractive accommodative esotropia is secondary to convergence induced by accommodation for which divergence inputs are insufficient to keep the eyes straight. However, the underlying issue is not one of high hyperopia per se, but rather of a clinical issue called a *high* AC/A ratio. Let's unpack this important concept.



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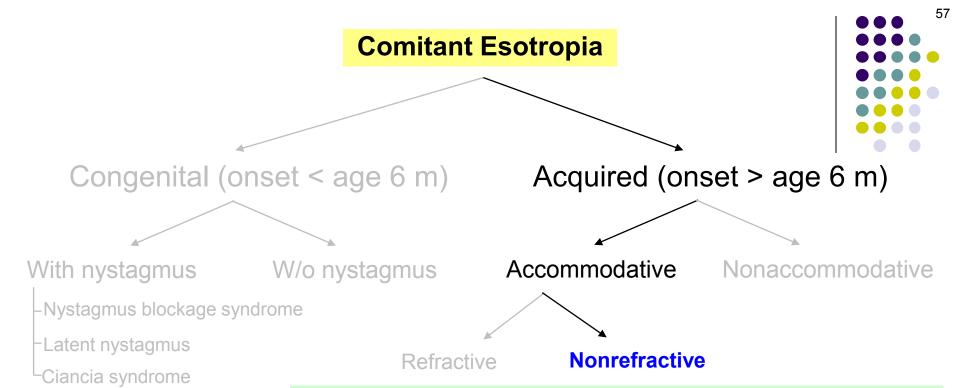
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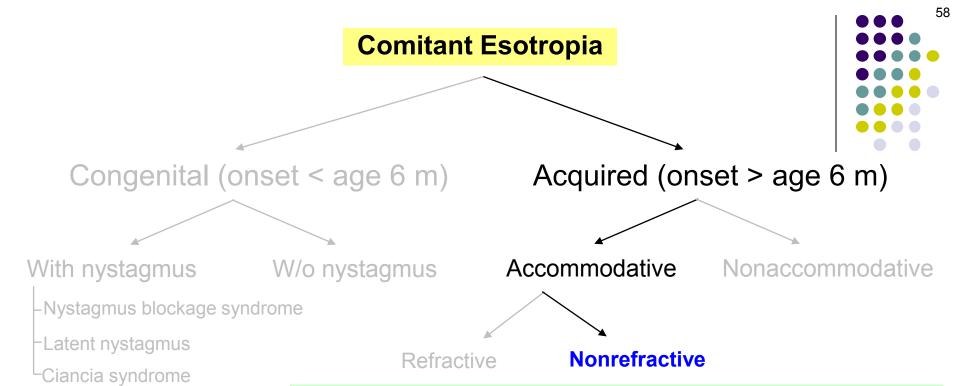
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Recall that a key feature of refractive esotropia (ie, ET secondary to high hyperopia) was that the magnitude of the ET was roughly equal at distance vs near, ie, ET = ET'.



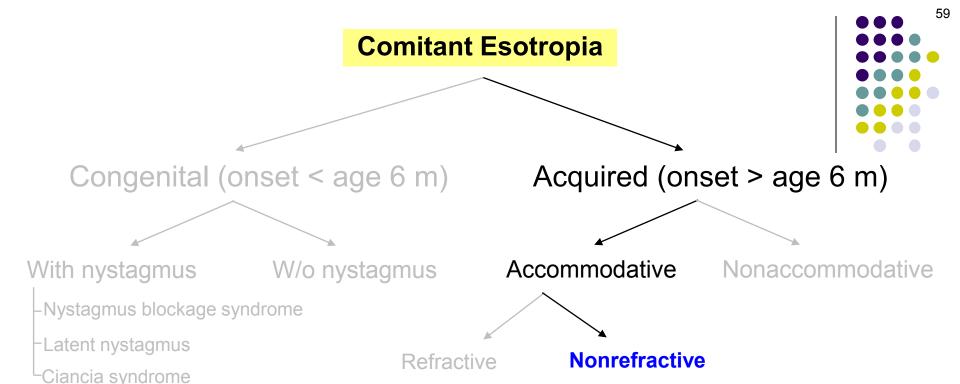
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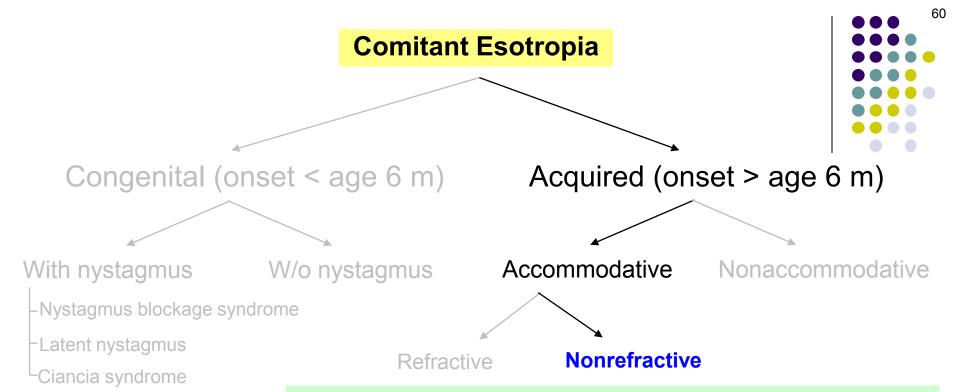
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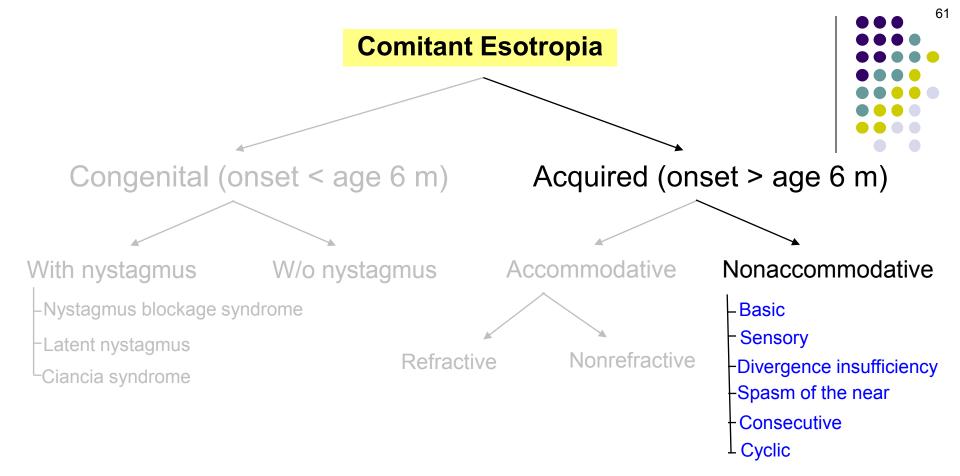
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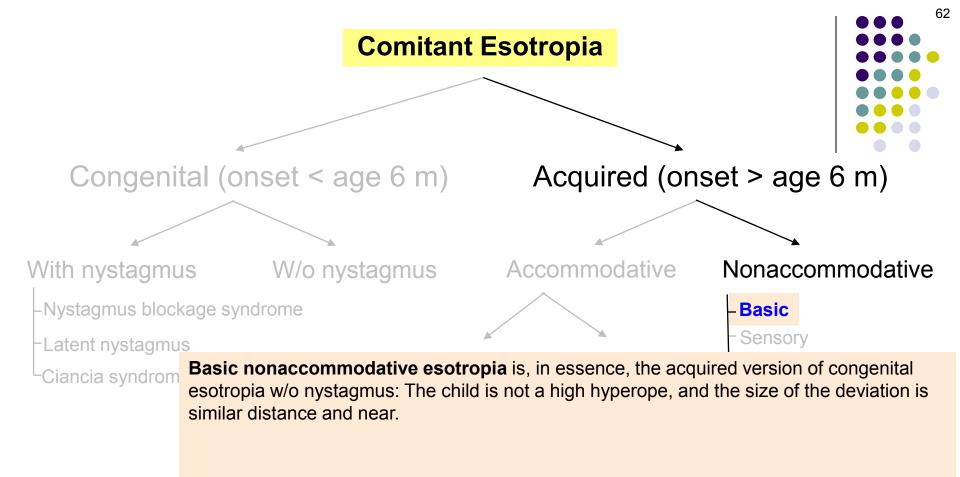
thumb: ET' will be at least 10∆ greater than ET.

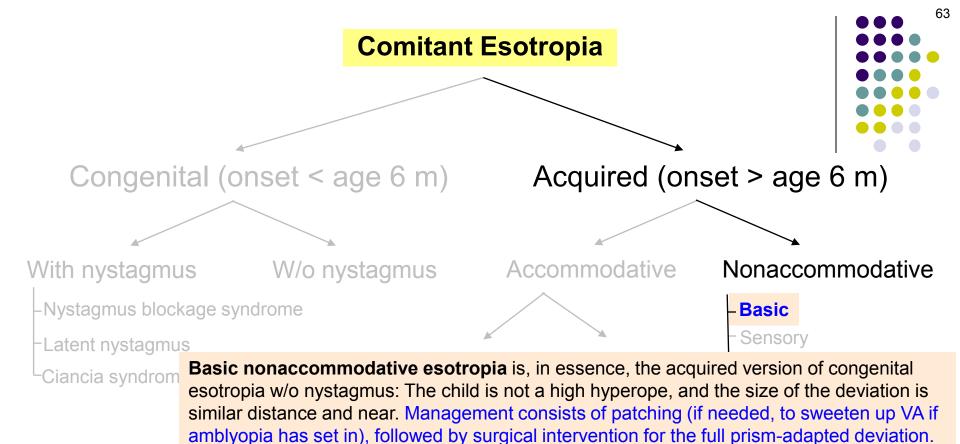
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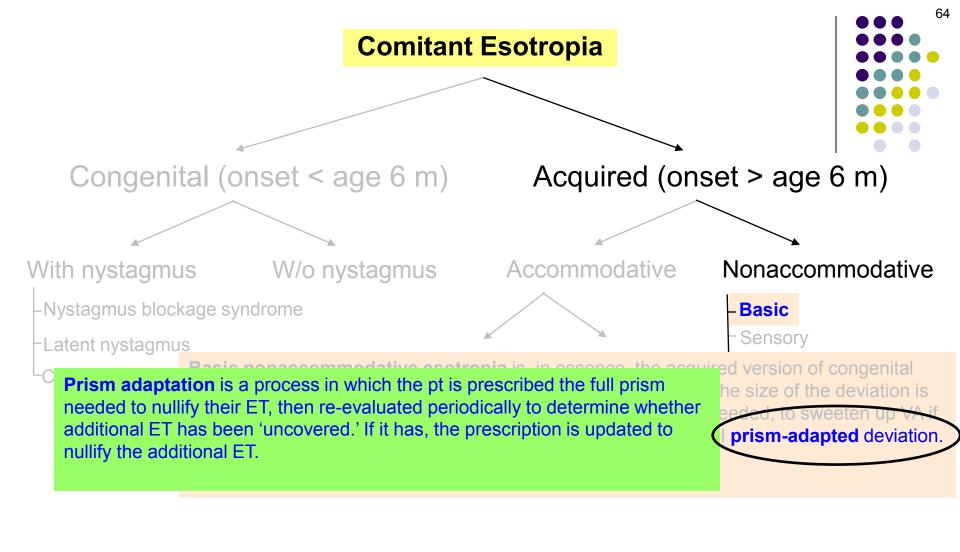
How best to manage high AC/A ratio accommodative esotropia is unsettled as of this writing. Some specialists elect observation so long as the child's eyes are pretty straight at distance. Others argue that bifocals are worth a try, provided you ensure the kid actually uses the add appropriately. Surgical correction can be countenanced, but is labeled "controversial" by the *Peds* book. While the ideal outcome would obviously be resolution of the ET at both distance and near, most specialists could live with a small amount (<10 Δ) of ET at near. the more the child accommodates, the greater their ET will be. It follows that, because hear vision

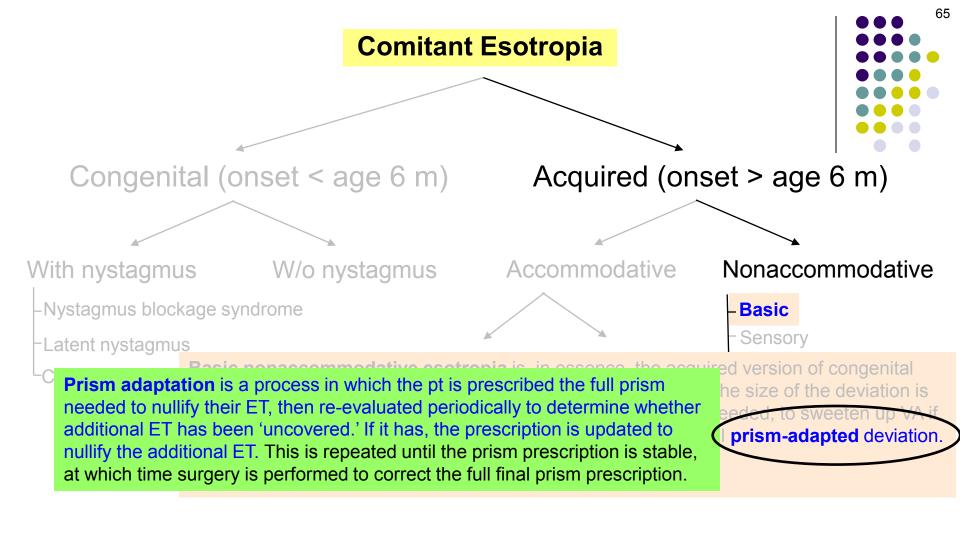


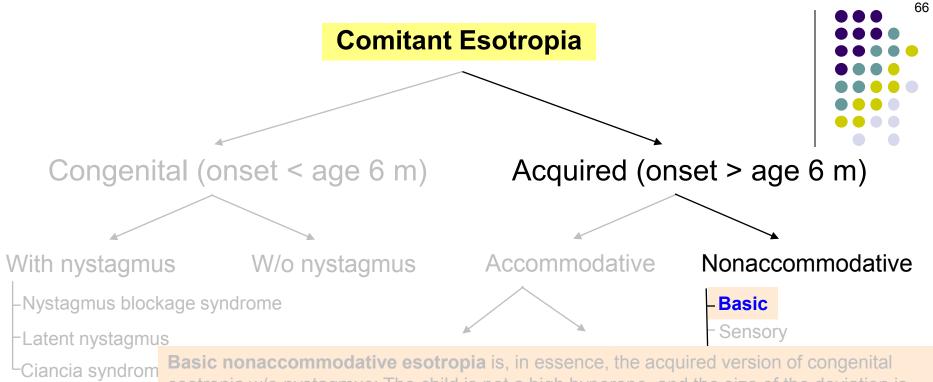
The *Peds* book discusses several forms of acquired nonaccommodative esotropia.





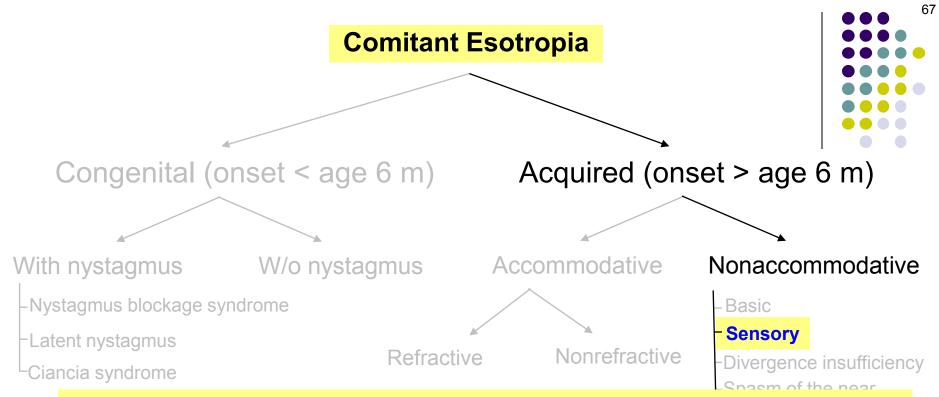




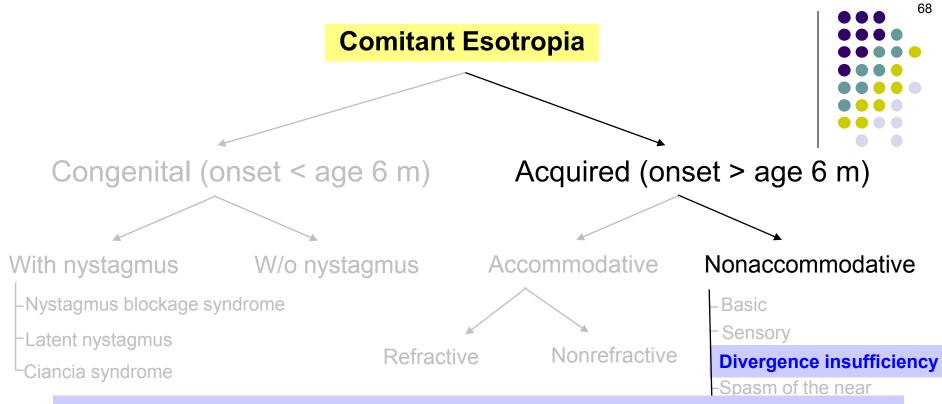


esotropia w/o nystagmus: The child is not a high hyperope, and the size of the deviation is similar distance and near. Management consists of patching (if needed, to sweeten up VA if amblyopia has set in), followed by surgical intervention for the full prism-adapted deviation.

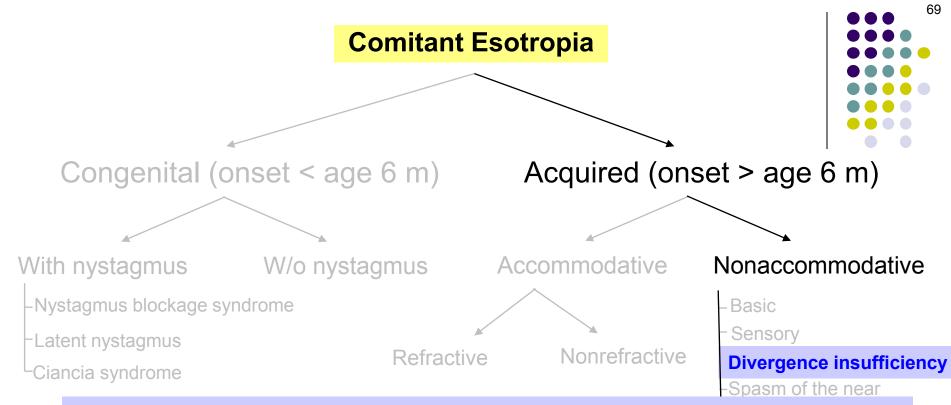
If there is anything hinky about the presentation (eg, any neuro signs/symptoms; face turn; c/o HA), imaging should be obtained.



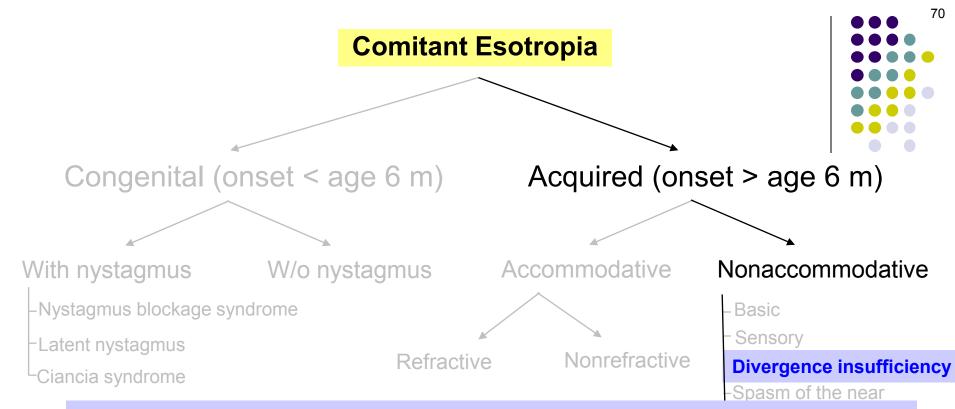
Sensory (aka *deprivational*) **nonaccommodative esotropia** develops in response to monocular vision loss. Common causes include cataracts, corneal clouding, and retinal or optic nerve disorders. The lack of symmetric visual stimulation leads to amblyopia, followed by a breakdown in fusion.



Earlier in this slide-set we mentioned supranuclear divergence inputs that prevent overconvergence. In **divergence insufficiency**, a lack of robustness on the part of these inputs allows the eyes to turn in a bit, resulting in a modest esotropia.



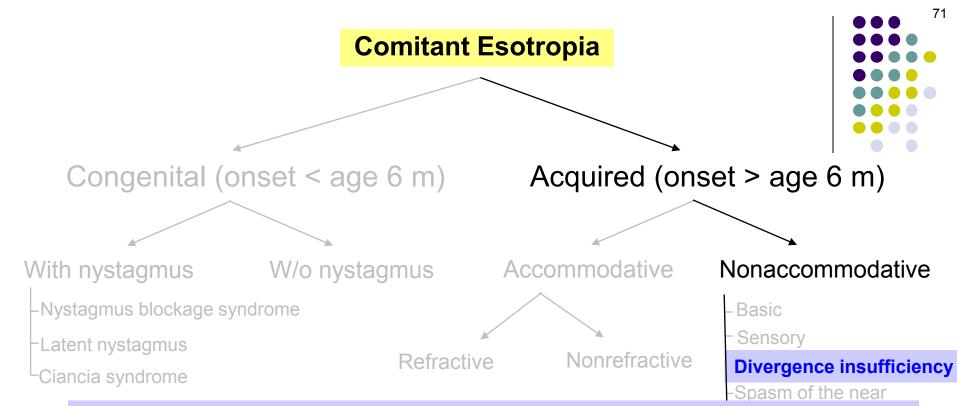
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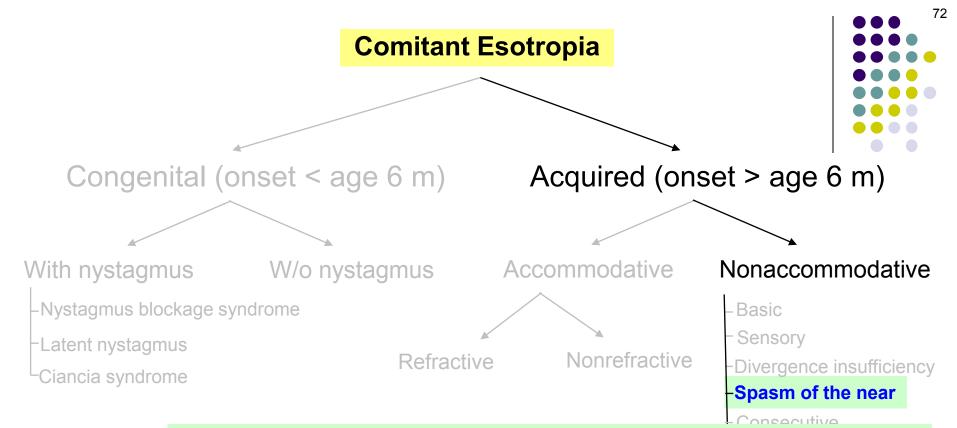
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Note that these conditions can be differentiated on the basis of the relative magnitude of the esotropia as a function of whether it is measured at distance vs near:

Accommodative refractive esotropia: ET ≈ ET'
Accommodative nonrefractive (high AC/A ratio) esotropia: ET < ET'
Divergence insufficiency: ET > ET'

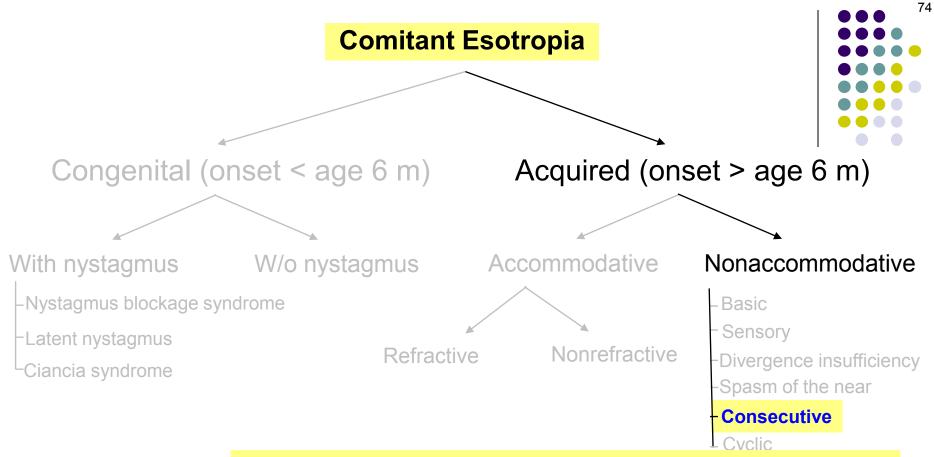


Earlier in this slide-set we mentioned supranuclear divergence inputs that prevent overconvergence. In **divergence insufficiency**, a lack of robustness on the part of these inputs allows the eyes to turn in a bit, resulting in a modest esotropia. The classic presentation is that of an esotropia that is present at distance, but not at near. The most common form of this develops in older individuals—hence its alternative name, *age-related distance esotropia*. In some pts, imaging reveals age-related structural changes to the EOMs or orbital ligamentous support structures. Prisms, Botox injection, and surgery have all proven safe and effective interventions.



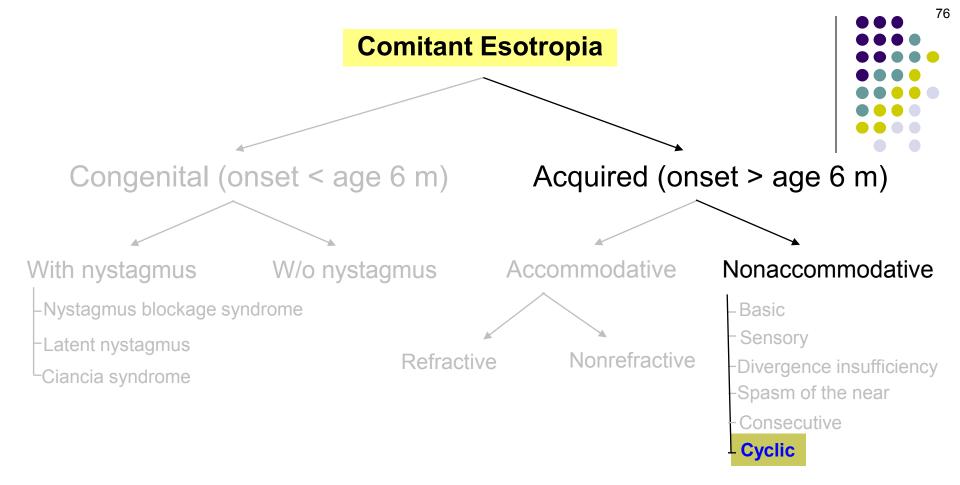
Spasm of the near (aka *convergence spasm*) is almost always a functional response to psychosocial stressors. All three components of the near triad (convergence, miosis and accommodation) can usually be demonstrated. The esotropia may alternate with periods of orthotropia. Abduction will be poor or absent when the eyes are tested simultaneously, but full when tested monocularly.

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Consecutive esotropia refers to esotropia that develops in someone with a history of exotropia. In almost all cases, consecutive esotropia is post-surgical, ie, it represents an apparent overcorrection in someone who underwent strab surgery for exotropia.

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Cyclic esotropia is a rare disorder in which a comitant ET is present intermittently, usually every other day. The typical pt is pre-school age. Surgical correction of the maximum observed ET is the treatment of choice.

Comitant Esotropia



That's it! Go through this slide-set a couple of times (at least) until you feel like you have a handle on it. When you're ready, do slide-set *P5*, which covers this material in a Q&A format (and more detail).