The Relationship Between Accommodation and Convergence

There are cases where you have a standard tightly controlled accommodative/vergence insufficiency and accommodation is the 'leader' in helping the poor convergence over the 'finishing line' of Panums area. But in what anomalous circumstances if any, does convergence lead a weak accommodation?

This is a brilliant question, which no one has asked before and I have not considered.

**Accommodation /Convergence insufficiency (most common, say 60%)**

You have expressed how accommodation can be used to assist poor convergence very well. I think there is almost a requirement of accommodation itself to be poor where there is convergence insufficiency, so the increased enervation going to accommodation is reflected in the increased enervation to the medial recti.

**Accommodation Insufficiency (30%)**

Where accommodation is weak with normal convergence (accommodation insufficiency), plus lenses on their own will correct it.

**Convergence insufficiency (10%)**

If accommodation is normal it cannot be used to control a convergence insufficiency so in the absence of orthoptic exercises it has to be corrected with prism. The arguments against eye exercises in what is regarded at the most susceptible orthoptic condition to therapy are:

- They only work for as long as there are given
- They can hurt
- Poor compliance
- The effect may vary with general health or fatigue

So therefore in what anomalous circumstances, if any, does convergence lead a weak accommodation?
My guess is that vergence sends a fixed amount of enervation to accommodation via the proprioceptive receptors in the medial recti. The need to accommodate on a near object is not nearly as great as the need to converge on it; poor convergence causes diplopia. Poor accommodation just causes blur, so at least you can judge where the object is in terms of position and distance, which is the survival imperative. If the convergence was required in any way to lead accommodation it would induce diplopia, which would immediately put the brake on this as a mechanism to control accommodation.

The reality is that the effect of convergence on accommodation is very difficult to show scientifically, because is it difficult to devise a convergence target which has no effect on the accommodation.

Is the principal of Schoolvision, the introduction of Plus power and Base In prism initially just to give a massively overworked and very tight accommodation and convergence a 'holiday' to repair themselves (medial recti to gain tone again by resting)?

The problem may be that the medial recti have lost the incentive to do anything and rely totally on enervation coming from the accommodation vergence reflex.

When the accommodation is corrected and the compensatory prism introduced, the medial recti now have an achievable target, and will be given the incentive to work a little harder (not rest). This is one of the reasons why patients get headaches initially with their new corrective lenses; some muscle working harder and some weakening off. I am beginning to find that teaching the eyes to see properly, where convergence leads accommodation, will reduce prism requirement. Be cautious about removing the prism altogether, the eyes may be rested but the underlying weakness remains.

**Compensatory Prism in Pure Accommodation Insufficiency**

In pure accommodation insufficiency there can be a secondary convergence insufficiency when corrected. This is due to a loss of tone in the medial recti to reduce the secondary esophoric effect. If the medial recti are otherwise healthy their tone will return and you will be able to remove the base-in support in due course (say 3 months).

Is it true that the accommodation and convergence, because they are working so hard for years, in effect become weak, as the massive constant enervation causes muscle paresis, like any muscle in the body?
A Holiday for Tired Muscles?

I do like the concept of a holiday and I am sure this is why some people feel brave enough to leave their correction off. Sometimes the symptoms return and they have to start wearing their spectacles again. I think it is also true to say that the risk of muscle paresis (pulled muscle) is greater in these patients (I know that from personal experience), which will trigger an episode of acute pain.

However this may not be due to weakness but over exercise. It could be that two tight mechanisms become stronger in this locked state, leading to latent hyperopia and accommodate spasm and far better accommodation is some presbyopes than you would expect.

We may find that presbyopes who hardly seem to need reading specs (in fact pride themselves on this and the amount of money they save) are ok with the occasional menu, but generally hardly read at all for any extended period. They tend to ask other people to tell them what the small writing is on the back of the cereal packet.

A Parallel with Dyspraxia

An interesting parallel with a locked visual system is dyspraxia where antagonistic muscle groups in the limbs are locked for fear of bumping into things. A free flowing body movement can come from a well-balanced visual system, which is able to judge the distance and position of an oncoming hazard.

Dyspraxic children can take on a chunky appearance because of hypertrophic muscles. You could even argue that their less sensitive visual systems and over developed muscles would predispose them to sports like rugby where visual demand is super ceded by the need for bulk and strength, especially in the forwards. This predisposition would be enhanced by the motivating frustrations of dyspraxia in the formative years, usually coexisting with dyslexia (being bullied, underachieving academically) and then realizing as they leave school "yes, I am quite strong and I quite fancy a bit of revenge, in an organized and legitimate way of course."