Interdisciplinary Symposium on
Pediatric Ophthalmology 2018
Physiology and biomechanics of the eye

Theory. Physiological and biomechanical features of the interconnected functioning of the systems of accommodation, and aqueous humor production and outflow systems. Hypotheses and executive mechanisms of the growth of the eye's optical axis in the metabolic theory of adaptive myopia and in the theory of retinal defocus

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The fundamental principle of development of all biological systems

Minimization of the energy consumption to perform the required work.

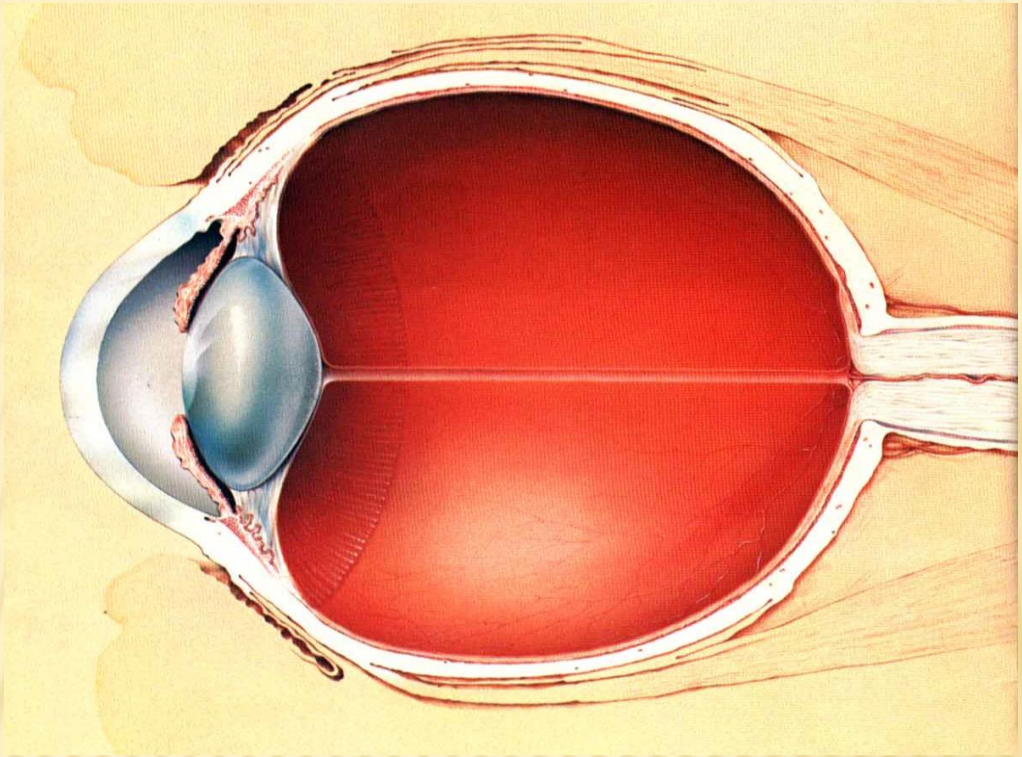
Therefore, the development of the anatomical structure in nature always goes on the way of energy conservation.

And below we will see how this key principle is performed in the eye.

Part 1.

Eye how interconnected system

The main drawback of the detection system in humans



In the lens there are no nerve endings!

Therefore, the brain is not able to understand what changes have occurred in the lens with age. To reverse presbyopia the brain will try to image insert some corrections on the retina using a system of accommodation. But this system has a limited range.

The ciliary muscle is the servant of the three gentlemen: accommodation, production and outflow

- Obviously, at the functional level in a coherent work of these systems lies the objective contradiction.
- For example, to increase the outflow of ocular liquid in the trabecular pathway, we must strain our ciliary muscle.
- But the same moment seen away the Tiger , we will need to relax the muscle ciliary.

Which commands will be implemented?

- Of course, the accommodation, because your life depends on it!

The first and the main principle of the eyes work

Absolute priority of the accommodation management system over the management of outflow system

Regulation of the outflow pathways with the help of ciliary muscle

Norm

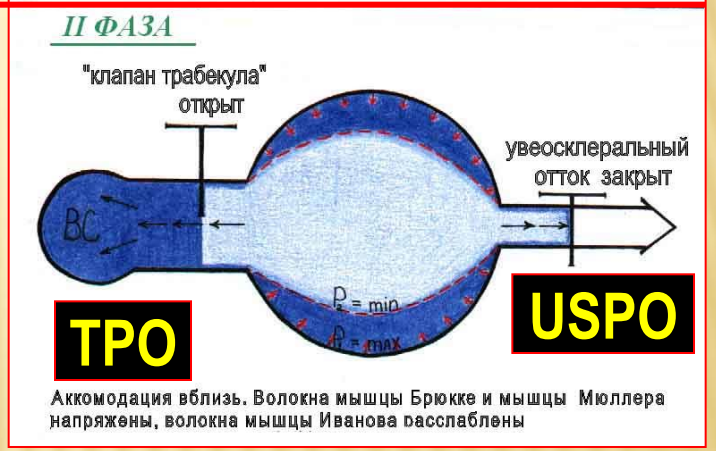
No accommodation:

The trabecular pathway **(TOP) is closed**

Norm

Near accommodation:

The Uveoscleral pathway **(USOP) is closed**



In fact the ciliary muscle acts as a switchman of the pathways, and the uveoscleral outflow pathway is closed only when we are to looking at objects located near us

Regulation of the outflow pathways through the ciliary muscle

In the works for the exploration of the human eye accommodation *in vivo* using modern high resolution biomicroscope Stachs O. et al.(2002,2003) brought out the most important clinical fact:

- The scleral spur moves posteriorly only when the tone of the ciliary muscle is above average and has not shifted posteriorly with a lower tone.

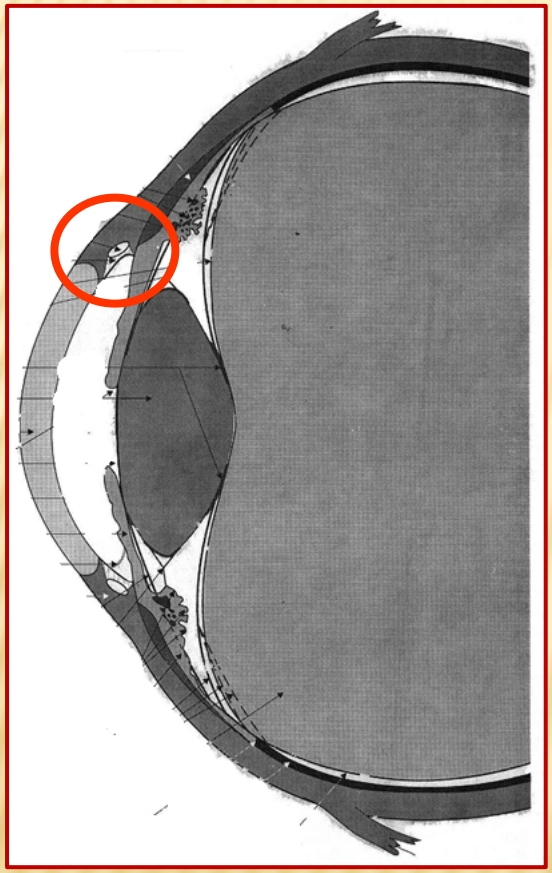
NB! Mechanism of TOP is included it is in moments of near vision, discovering this path as an additional outflow

Functioning of the outflow pathways of aqueous humor depending on the phase of accommodation

Name of outflow pathways of intraocular fluid	The phases of accommodation and the ciliary muscle tonus		
	The «completely near» phase of accommodation. Ciliary muscle tonus is maximal.	The «partially near» and «partially afar» phases of accommodation. Ciliary muscle tonus is average.	No accommodation. The tone of the muscle ciliary is minimum.
Trabecular outflow pathway through trabeculae (TPO)	Opened	Closed	
Uveoscleral outflow pathway through the matrix of the ciliary muscle and then through the sclera (USPO)	Closed	Maximally opened	Partially opened

In fact, the ciliary muscle regulates the intensity of the outflow pathways of intraocular fluid

The trabecular poutflow athway is an additional way of outflow, which allows the eye to work for a long time at near sight



We now can understand why a person and four species of highly evolved monkeys developed an additional trabecular outflow pathway.

After all, in most representatives of the animal world there is only one way the outflow – the uveoscleral pathway.

In the evolution of our ancestors work at close range have become more and more important

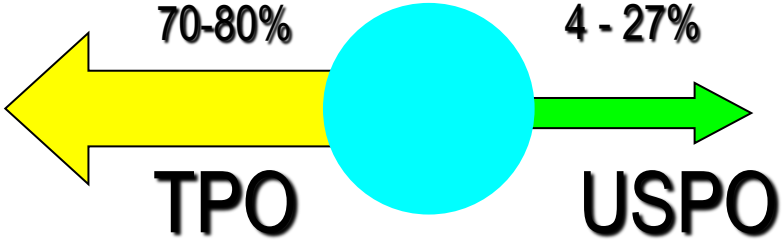
And at this point, the USOP is closed!

Part 2.

The sclera feeding mechanisms

Interaction the trabecular pathway outflow (TPO) with the uveoscleral pathway outflow (USPO) in norm and glaucoma

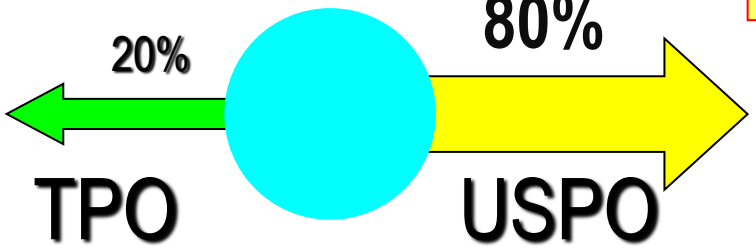
Normal



S. Düke-Elder (1968),
A. Bill & I. Phillips (1963,1975),
A.P. Nesterov (1976)

A. Bill (1989): USPO in norm = 35-60%

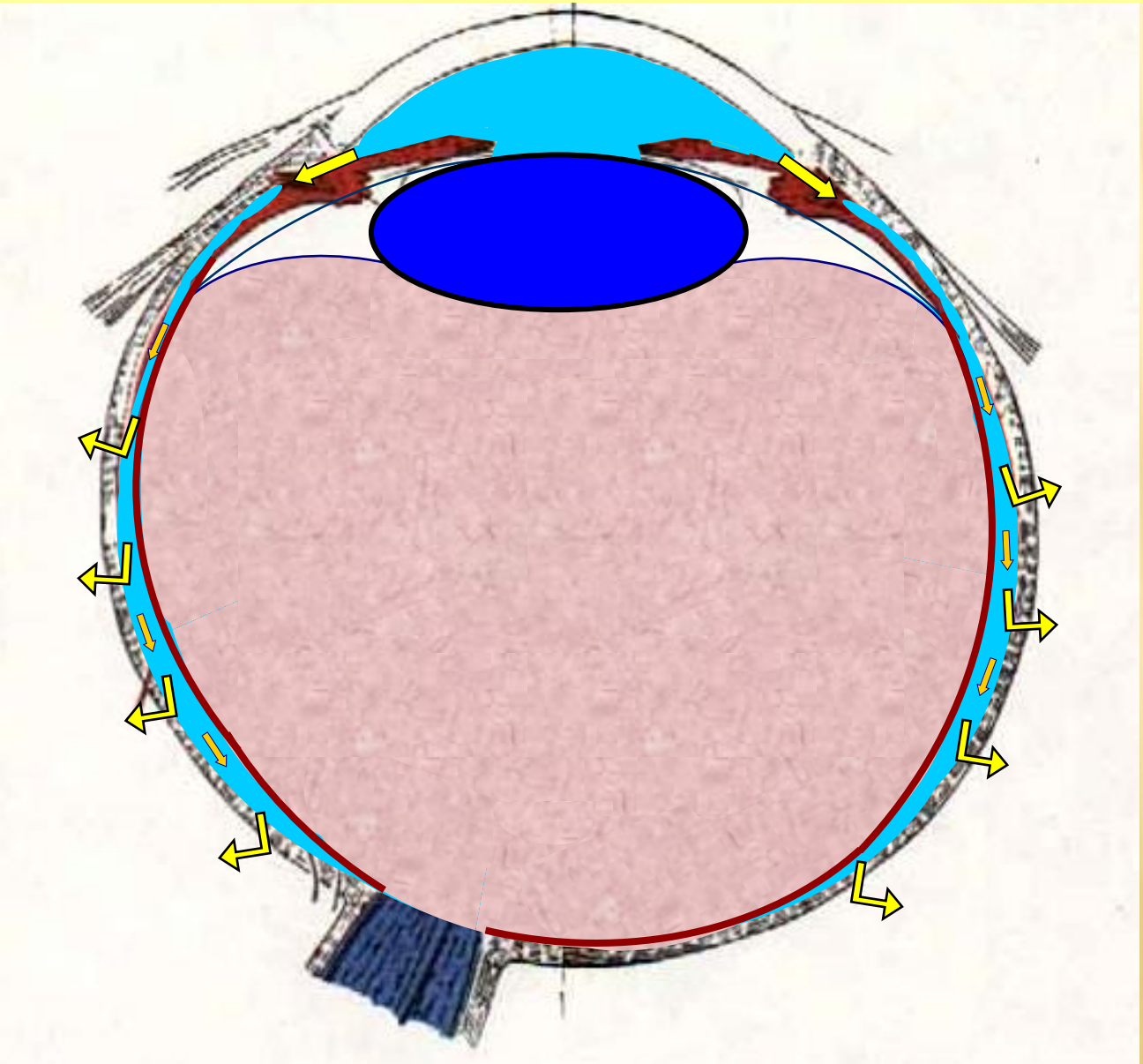
Glaucoma II - III:



N.V. Koshych (1998)

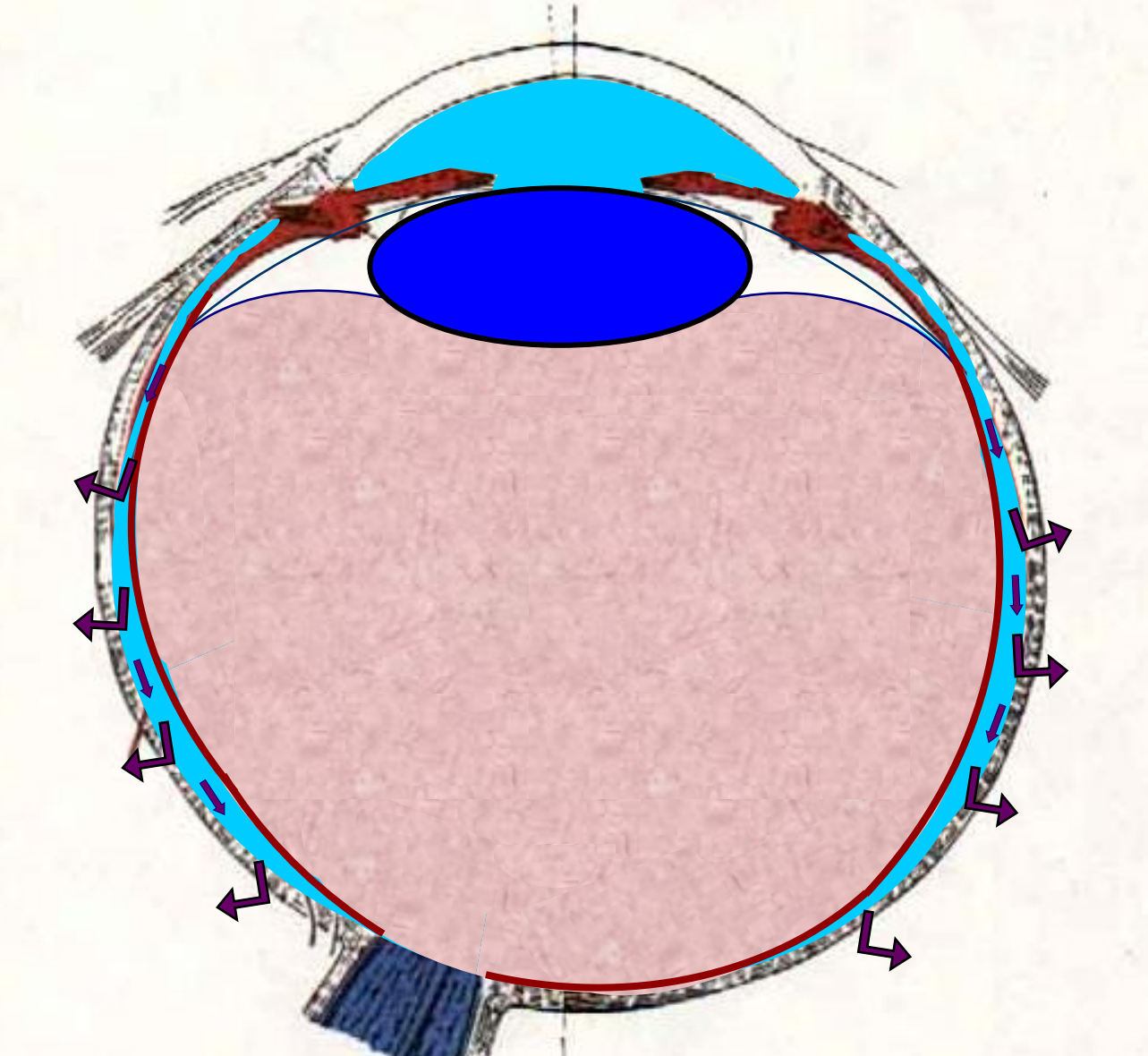
These data suggest that USPO is the main pathway outflow !

The delivery of ingredients for collagen in the middle and posterior parts of the sclera in mammals is provided to a large extent, through the USOP



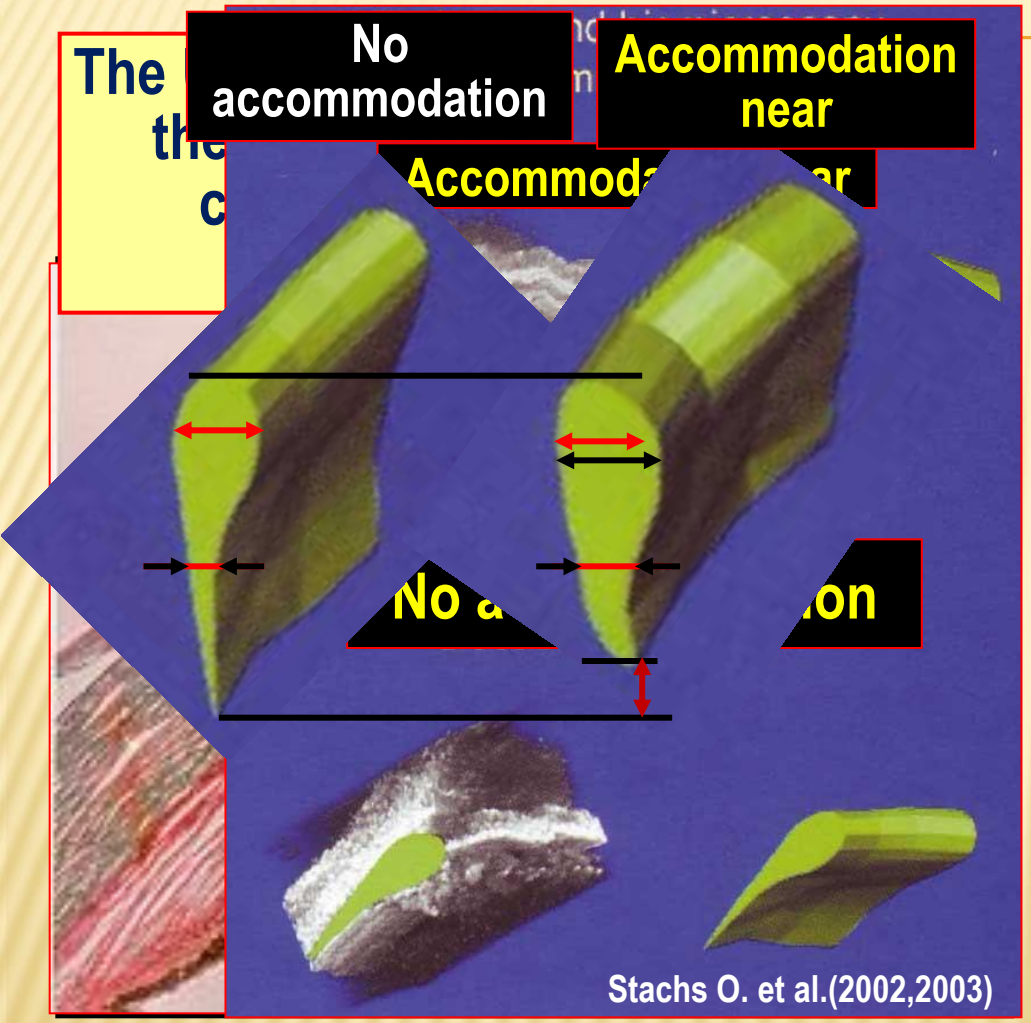
And this path is open only when the ciliary muscle have the average tone.

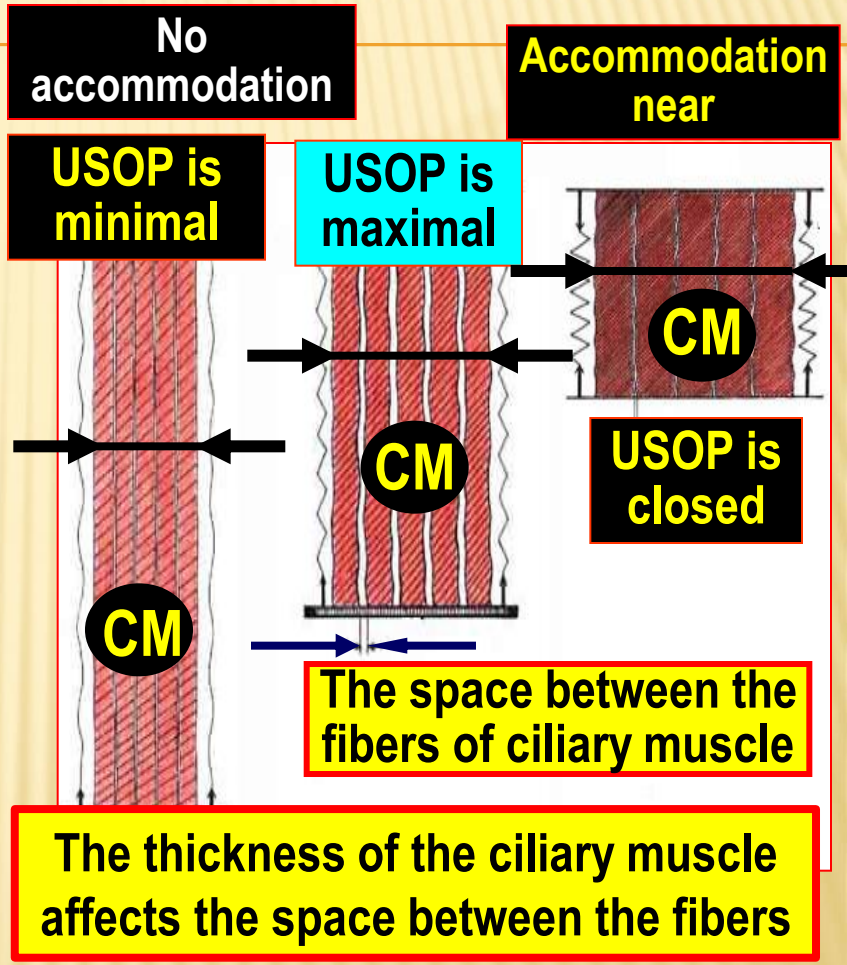
The second way of delivery of ingredients to the middle and posterior parts of the sclera in mammals is provided by the work of the mechanism of ultrafiltration of aqueous humor from vessels choroid.



In the absence of accommodation, this mechanism is ineffective.
However, even with the intense visual work at near this mechanism does not provide full maintenance metabolism in the sclera.

The USOP regulation mechanism





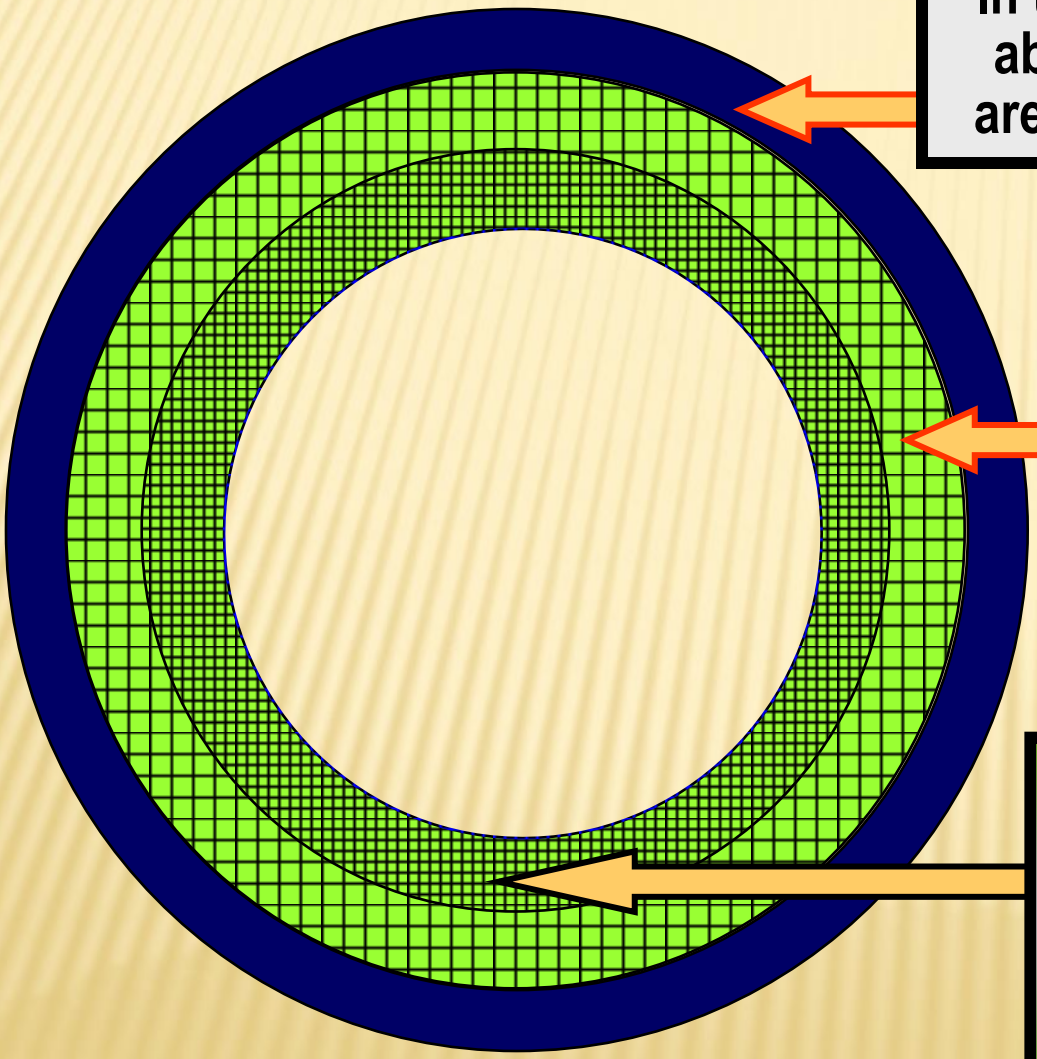
Extreme phase the accommodation mechanism is unfavorable for USPO

THE WORK OF THE MUSCLE CILIARY IN VIVO

A. Glasser, 2004

It is cleary visible that basically works - the Muller muscle

Location of bundles of collagen fibers on the thickness of the sclera

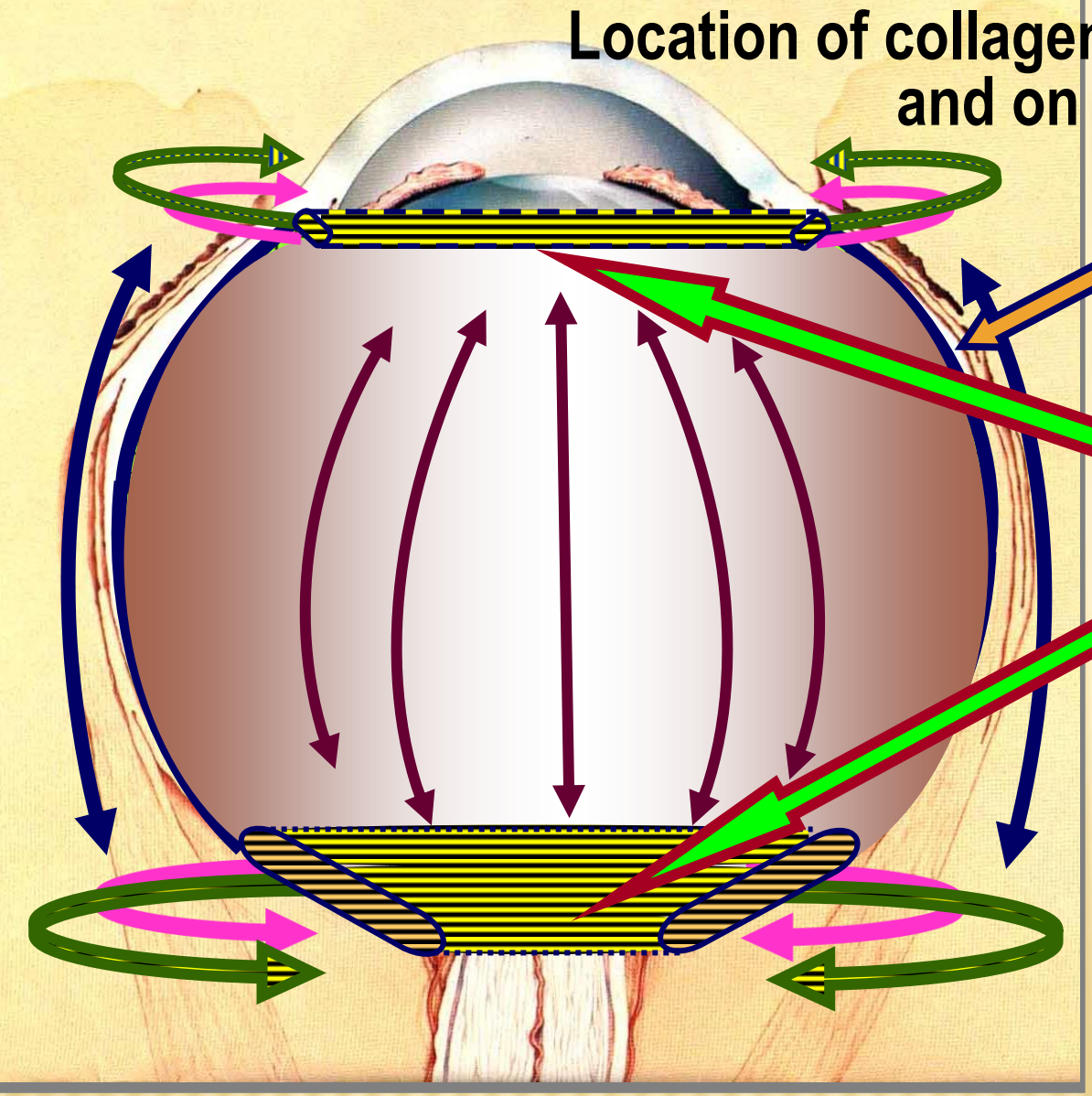


In the surface layer elastic fibers are absent . Bundles of collagen fibers are located in a meridional direction.

In the middle layer there is ~ 30% of all elastic fibers. The bundles of the collagen fibers are located in a meridional and oblique directions.

In the inner layer there is ~70% of all elastic fibers. The bundles of the collagen fibers are located in ta layer structure in a meridional and oblique directions.

Location of collagen fibers on the segments and on the surface of the sclera



Surface meridional bundles of collagen fibers

Equatorial located beams of collagen fibers

=

Anatomy of the pole of sclera in the form of a spring allows to easily adjust the length of the optical axis of the eye.

Part 3.

The executive mechanisms of adaptive lengthening of the eye:

our *metabolic theory of the adaptive myopia*

Hypothesis:

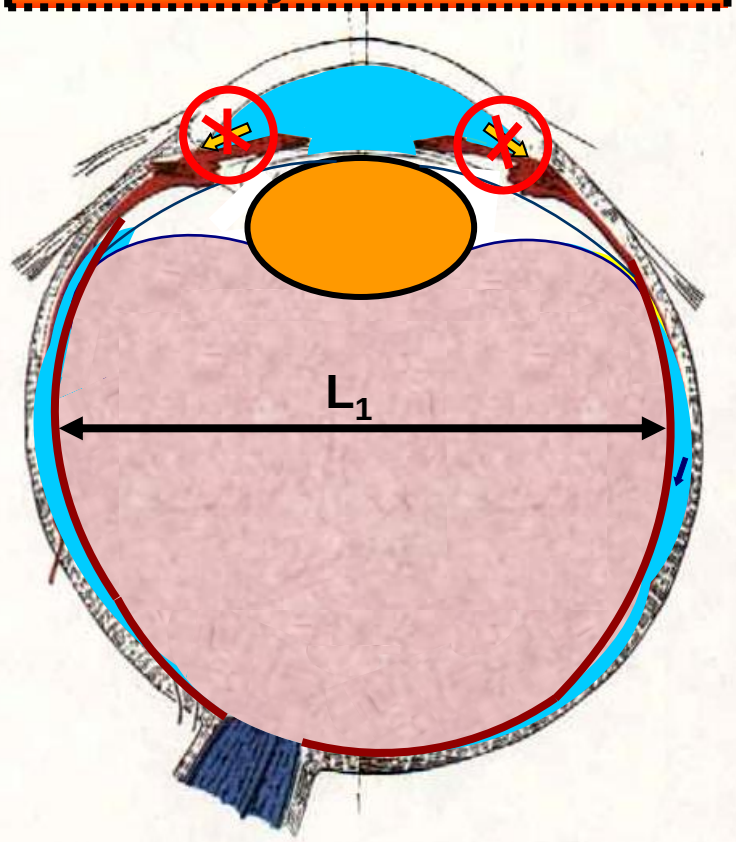
- In the eyes of mammals the mechanism for selecting length of the optical axis of the eye under continuous visual load can be associated with a temporary interruption of the uveoscleral outflow pathways (USOP).

- Physiologically well-founded selection of length of the optical axis enables lower power consumption of the eye.

The root cause of myopia on the load type. Look completely near

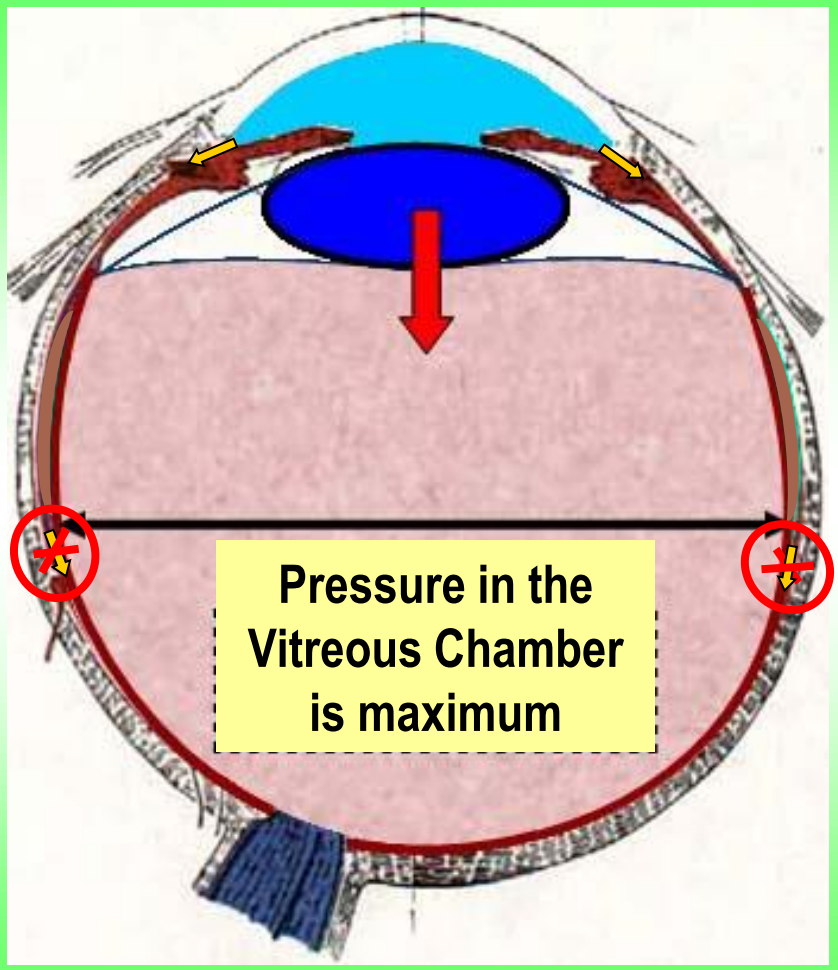
The root cause of the progression of myopia of the load type – is physiological: while working at the close distance the USOP is overlapping and disrupting nutrition of the posterior part of the sclera.

The Eye work near



The root causes of myopia by the unloading type.

Look fully away.



1.The space between the fibers of the ciliary muscle is minimum.

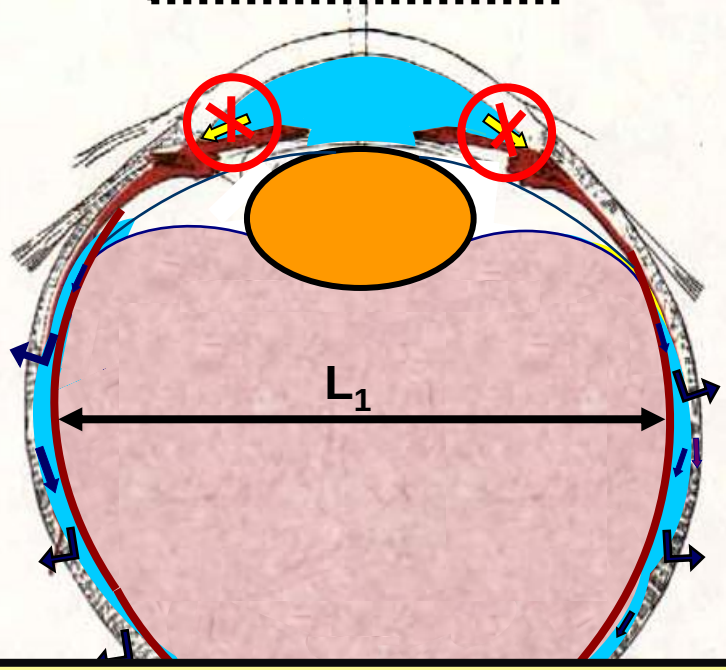
2. Blood flow to the eye and production of the aqueous humor decrease in three times.

3. Equator vitreous Chamber blocks the access of the aqueous humor to the posterior part of the sclera.

1.The load type myopia.

Adaptive myopia at maximum tone of the ciliary muscle.

Near vision

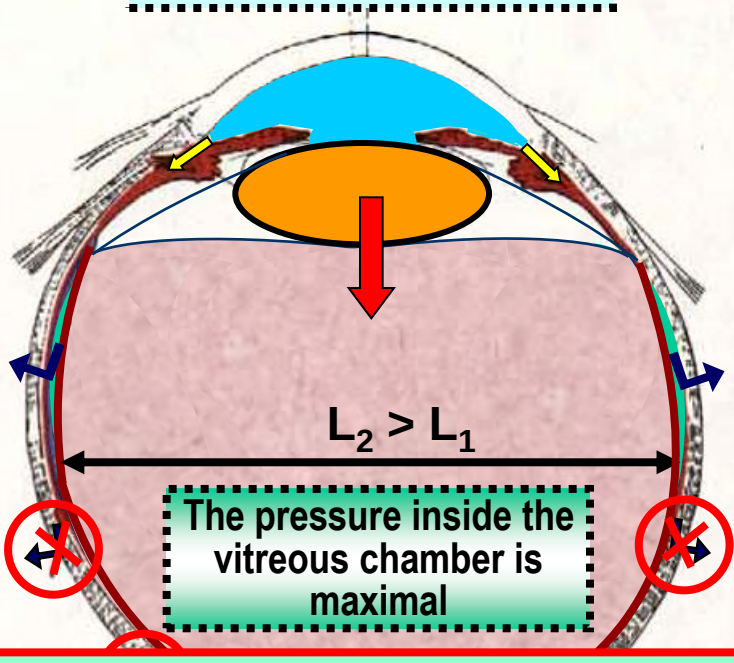


Maximum tonus of the ciliary muscle. USPO iclosed

2. The unload type myopia.

Adaptive myopia at minimum tone of the ciliary muscle.

Distance vision



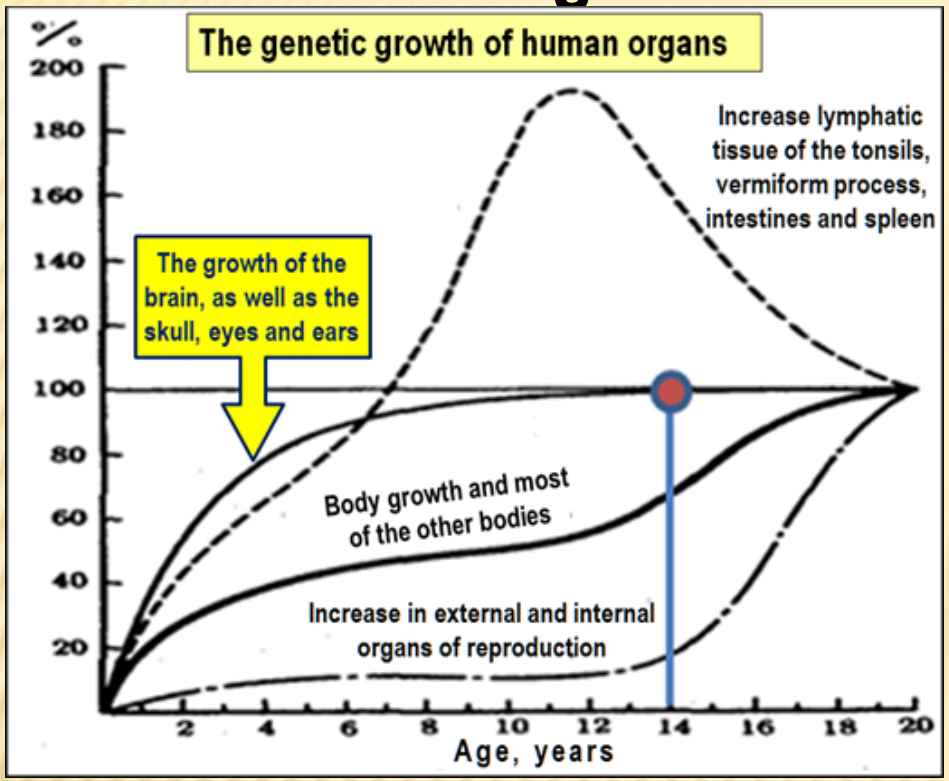
Minimal tonus of the ciliary muscle.USPO limited

And these two types of development of the adaptive myopia will be included in the New Classifiation of myopia.

Part 4.

Key moments of early refractogenesis

Curves of growth of separate human organs



It is clearly seen that by the age of 14 the "genetic" growth of the human eye stops (red dot).
In a norm, early refractogenesis in childhood ends with this condition: the eyes grow synchronously with the body and internal organs.

The genetically "slowed" youthful growth of the eye at the age of 10-14 years (unlike the early childhood from 2 to 5 years) can not ensure quick adjustment of the eye`s optic axis directly, without attracting any other executors – physiological, for example.

Curves of growth of separate human organs

- Indeed, adaptation requires that the axial growth of the eye is accelerated many times compared to the relatively slow growth of the body and brain in an early adolescence..
- So we know that every child has a genetic program for the gradual growth of the eye in all directions, according to the corresponding parallel growth of the skull and orbit.
- And this program will be executed, even if we`ll cut the optic nerve.
- And normally the eyes will certainly reach the state of weak hyperopia.
- In a norm, early refractogenesis in childhood ends with this condition: the eyes grow synchronously with the body and internal organs.

Key moments of late refractogenesis

1. This period refers to the age of 14–23 years and has been relatively little studied.
2. There are few clinical data that confirm that a significantly faster growth of eye`s optical axis is possible during this period, compared with the period of early refractogenesis.
3. And this requires the possibility of additional rapid adaptation of the visual mechanism.
4. And this is a key issue in the philosophy of the adaptive myopia development!
5. If the mechanism of accelerated ocular "axial" genetics does not work in adolescence and adulthood, then what executive mechanisms lead to such a massive and rapid emergence and development of progressive myopia?

Key moments of late refractogenesis

- **The adaptive myopia rapidly develops through non-genetic, but physiological executors.**
- **And it is possible even in the period of early adolescence with normal genetic growth of the eye.**
- **The emergence and adaptive progression of emmetropy and initial myopia presumably are linked to the manifestation of a regular physiological mechanism, which is the same for humans and animals.**
- **The length of the eye, corresponding to visual loads, is formed in such way to ensure the lowest possible level of energy consumption during intense and prolonged near visual work.**

Part 5.

Key points of the retinal-defocus theory

The genetic theory of retinal defocus changes (IRDT) by G.K. Hung and K.J. Ciuffreda (2004).

It is based on the idea of a possible acceleration of the processes of genetic eye growth even in adulthood.

The most widespread theory of peripheral defocus is known for its shortcomings and is based on a number of hypotheses that are not fully supported by scientific research.

The genetic theory of retinal defocus changes (IRDT) by G.K. Hung and K.J. Ciuffreda (2004).

- According to the IRDT, the retina is the "brain center" of the eyes growth even if the optic nerve is cut.
- It turns out that the eyes of animals can grow rapidly even without connection with the brain, if the focus shifts in a certain direction.
- Thus, the processes of early and late refract genesis in IRDT are "unified and inseparable": the genetic mechanisms of morphosis in childhood are simply extrapolated to adolescence and adulthood.
- It is assumed that the speed of the work of genetic mechanisms can increase several-fold.

The genetic theory of retinal defocus changes (IRDT) by G.K. Hung and K.J. Ciuffreda (2004).

Advantages of IRDT:

- This is another historical attempt to explain the refractogenesis of progressive myopia by disorganization in the work of mechanisms of collagen formation in the sclera.
- G.K. Hung and K.J.A. Ciuffreda tried to explain the mechanism of eye focusing through the work of neuromodulators, which are sensitive to changes in the contrast of the retinal image. This attempt should certainly be welcomed. This is a promising path and an important attempt, although not the first one.
- Also worthy of respect is the attempt of G.K. Hung and K.J.A. Ciuffreda to find direct actuating mechanisms of the influence of visual load on the processes of collagen formation in the posterior part of the sclera.

The genetic theory of retinal defocus changes (IRDT) by G.K. Hung and K.J. Ciuffreda (2004).

Disadvantages of IRDT:

- The assumption that the brain does not participate in the process of late refract genesis contradicts a huge number of clinical facts.
- The hypothesis that there is a growth center of the eye in the retina, separated from the brain, is the most incorrect one.
- The very idea that the retina develops, and then delivers along its own ways to the sclera some chemical substances, that ensure the regulation of the rate of refract genesis at a genetic level, is certainly taken from the science fiction. But nowhere will we find the message that the retina is a biochemical "machine" that directly controls the growth of the sclera.
- A serious disadvantage of IRDT is the low response rate to the change in the surrounding visual environment (years).

Conclusion.

1. We found that the main reason for the adaptive growth optical axis of the eye associated with the implementation of the law on energy conservation in the development of biological systems.
2. The adaptation mechanism of growth of the optical axis of the eye is common in humans and animals.
3. The executive mechanism of adaptive growth is a mechanism for creating temporary functional insufficiency the uveoscleral outflow pathway of aqueous humor.
4. In fact, the adaptive myopia is a classic case of predominance of accommodation over outflows.

Conclusion.

5. The theory of changes of retinal defocus has significant flaws and yet contrary to accumulated clinical facts.
6. The metabolic theory of the adaptive myopia can explain known clinical facts and give practical advice on promising ways of slowing down the acquired myopia.
7. The obvious consequence of metabolic theory is the appointment of an early correction to slow down the myopisation process.
8. Acquired myopia without complications, in fact, is not a disease, but a natural process of adaptation to visual habitat.

Thank You for Your attention!

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