Autonomic Nervous System and Control of Visual Function

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The peripheral nervous system element known as the autonomic nervous system regulates involuntary neurobiological processes such as heart rate, blood pressure, and breathing rate as well as ocular changes such as accommodation, intraocular pressure (IOP) regulation, and pupil size. Autonomic afferents are found in almost all ocular parts and regulate local balance in the body via antagonistic and synergistic interactions. The current review illustrates the critical role played by ANS in regulating visual functions. Any imbalance in the functioning of ANS can lead to pathological changes such as myopia and hyperopic defocus.

The sympathetic, parasympathetic, and enteric nervous systems comprise the autonomic nervous system. Both peripheral nerve fibers and innervation are found in the sympathetic and parasympathetic nervous systems, which receive sensory input from the central nervous system (CNS) and transmit motor output from it. A preganglionic neuron with a cell in the CNS and a postganglionic neuron with a cell body in the periphery are typically found in SNS and PNS pathways.[1]

Numerous studies have found that the ANS accurately controls the physiological functions of the eye, including the regulation of IOP, pupil dimensions, lens accommodation, and circulation within the eyes. Nearly, all ocular components contain autonomic innervation for regulating local homeostasis through antagonistic and synergistic interactions.[2] Numerous ocular functions are influenced by the ocular projections of ANS. There are two of them: (1) pupillary diameter and ocular accommodation, each controlled by the eye's muscles in the iris and ciliary body; these structures are innervated by the ciliary (parasympathetic) and postganglionic fibers of the upper cervical (sympathetic) ganglia and (2) ocular blood flow controlled by the vascular system of the optic nerve, retina, choroid, and iris. The production and excretion of the aqueous humor are primarily regulated to control IOP. Aqueous humor formation is significantly influenced by the autoregulation of the ciliary epithelium and its blood vessels, so even though intracellular outflow is influenced by the control of the episcleral vascular system and trabecular meshwork. Postganglionic fibers from the superior cervical (sympathetic) and pterygoid (parasympathetic) ganglia innervate these tissues (Figures 1 and 2).[3]

The ANS primarily regulates the function of the majority of the intraocular muscles in the eyes. Additionally, the pupillary light reflex (PLR) is frequently utilized to identify ANS dysfunction.[4] Nerve endings are capable of secreting solubilized trophic materials that promote lacrimal gland production, elicit eye movement's reaction times, and maintain the integrity of the corneal surface.[5]

The PNS forms the major innervation of ciliary muscles, and its action is mediated via acetylcholine's interaction with muscarinic (largely m3 subtype) receptors. Positive accommodation is regulated by PNS input to the ciliary muscle, which also generates the demand for quick focus shifts, due to its quick onset effect. Further, it is known that because of the sympathetic innervation to the ciliary muscle, noradrenaline acts on two subclasses of postsynaptic receptors and the inhibitory α1 and β2 adrenoceptors.[6]

Ocular accommodation happens when the lens "bulges" (increases in convexity) as a result of ciliary muscle contraction leading to the reduction of zonular fiber and the increase in the lens' refractive power. The parasympathetic afferents of the ciliary body are predominantly responsible for controlling the dynamic behavior of amicable responses. In contrast to sympathetic afferents, which act for a bit longer period of time (10−40 seconds) to cause hypermetropia of almost 1.5 dioptres, parasympathetic innervation acts rapidly one second to generate a positive accommodation up to 20 dioptres.[6] Prolonged near work can increase accommodation

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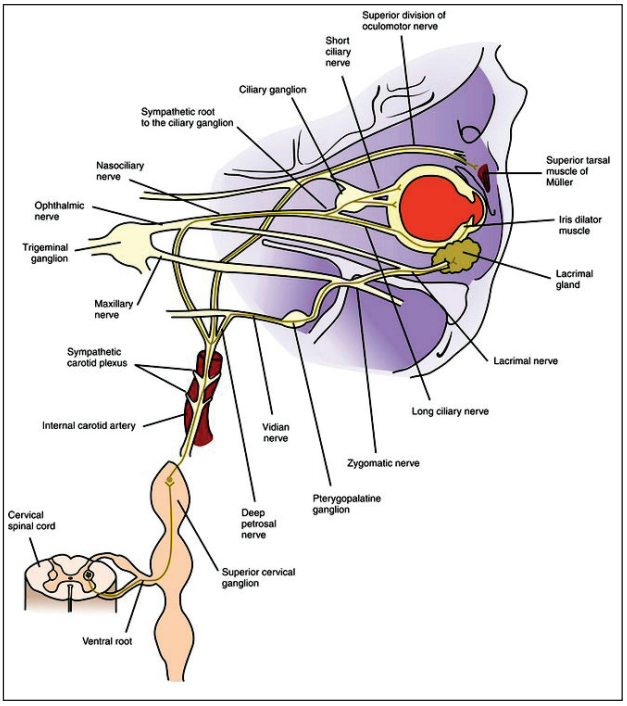

Figure 1. Sympathetic Innervation to Iris Dilator, Müller Muscle, Blood Vessels, and Lacrimal Gland.[7]

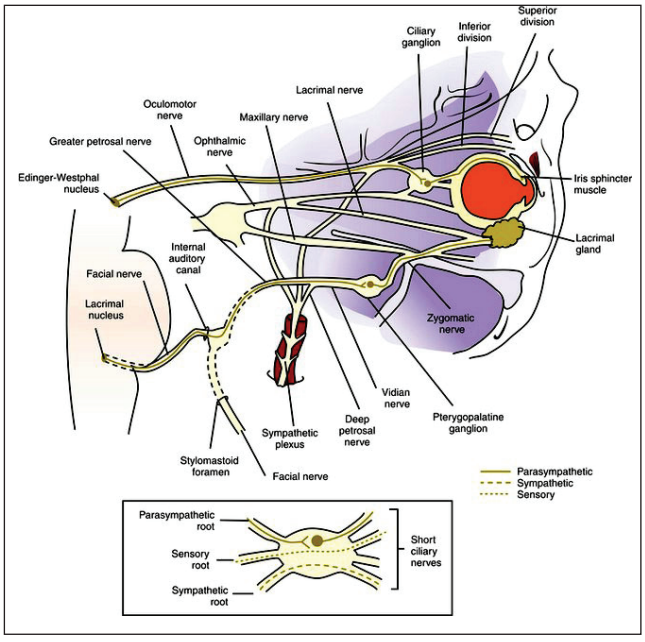

Figure 2. Parasympathetic Innervation to Sphincter and Ciliary Muscles and Lacrimal Gland. Inset Shows Sensory, Sympathetic, and Parasympathetic Fibers into Ciliary Ganglion; only Parasympathetic Fibers Synapse. Each Short Ciliary Nerve Carries all Three Types of Fibers.[7]

lag, aggravate single-vision retinal defocus, and eventually lead to myopia. It is possible that myopia's axial elongation is an effort to lessen the severity of hyperopic retinal defocus.

Nearwork-induced transient myopia (NITM) is the term coined to refer to the transitory myopic change in distance refraction that happens after plenty of near work. Some have hypothesized that innervational and/or neuropharmacological effects are the cause of NITM, while others have argued that the condition is economically sustainable in origin. According to reports, myopes are more likely to develop NITM, whereas hyperopes and emmetropes are less likely.[6] The relationship between myopia and accommodation is probably due to the accommodation system's poor performance when faced with ongoing near tasks. The proper synergy between sympathetic and parasympathetic elements of accommodation could lead to optimal function.[8]

If there is a loss of sympathetic input, the following gradual sequence of events may result in myopia: decreased sympathetic input to the ciliary muscle results in the initial stimulative hysteresis impacts and continued to increase transient pseudo myopic shifts in range reflected in cumulative retinal defocus. Retinal defocus rises up to a certain point along with an increase in the axial length.[9] Retinal defocus, abnormal ciliary muscle tone, and scleral stretching are the main causal factors of myopia, because of an autonomic imbalance leading to accommodation abnormalities.[6] The hyperopic retinal defocus that happens when there is a significant accommodation changes and myopia during close work may degrade the retinal image quality. In some cases, it results in a physiological reaction called myopia, as a rise in length would enhance visibility. Van Alphen in 1961 proposed a concept of emmetropization, suggesting ametropia might be related to the choroid's resistance to IOP.

The authors proposed that the ciliary muscle contraction is the source of axial elongation. Because of ciliary muscles contraction, the choroid is pulled forward and thins, moving the RPE and retina somewhat posteriorly as a result. In 2015, according to Woodman-Pieterse and collaborators, at the 6 D accommodation demand, the temporal and inferotemporal parafoveal choroid had the greatest choroid thinning, which accelerated with increasing quirkiness from the visual field. The arrangement of the nonvascular smooth muscle within the uvea and the geographical variance in parafoveal thinning may imply that these cells are a potential source of the choroid's thinning during the accommodation.[10] Moreover, a considerable increase in the axial length was noted, with the biggest increase occurring at the rate of accommodation. The authors hypothesized that enhanced parasympathetic activation of nonvascular smooth muscle during the accommodation may be associated with regional variations in the choroidal thinning. Myopia formation and advancement are influenced by various mechanical factors, along with ciliary muscle forces, extraocular forces, and convergence.[11]

Although fluctuations in choroid thickness occur with the progression and onset of refractive errors, it is still uncertain, how it influences eye growth.[12] The ANS may play a passive or active role in regulating changes in choroidal thickness that occur during the onset and progression of myopia.[13–16]

The image quality that is predicted on the fovea is strongly affected by accommodation and pupil as a result of PNS and SNS activity. They also attempt to manage ocular blood circulation as well as the tone of choroidal non-vascular soft tissue to monitor and control different variants in the ocular surface area. The ciliary body may act as a mediator of chemical signals that alter scleral dimensions to maintain the axial length required for clear vision because of its distinctive position between both the retina and the conjunctiva[17] or obstruct the diffusion of retinal-derived signaling pathways or signal transduction.[12]

Research trials and histological research show that the choroid has a significant autonomic vasoactive nerve supply. ANS inputs may have a strong influence on changes in choroidal thickness and the regulation of eye development. The pterygopalatine ganglion, which is present deep inside the pterygopalatine fossa, receives its primary parasympathetic input from the facial nerve and sends it to the choroid. The majority of parasympathetic neurons are cholinergic, but they also contain the synthesis of nitric oxide and create thick plexus on the wall surfaces of choroidal vessels, moderating variable vascular permeability, and boosting blood flow.

The abovementioned brief review illustrates the critical role played by ANS in regulating visual functions such as accommodation, IOP, choroidal thickness, axial length changes, and ocular blood flow. Any imbalance in the functioning of ANS may lead to changes in the visual function resulting in myopia and hyperopic defocus. It will be interesting to evaluate the role of Yoga, which is known to restore the balance of ANS function and could also ameliorate myopia progression.

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