Review Article

Substrates of Negative Accommodation

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ABSTRACT: Negative accommodation refers to a unique phenomenon, sparsely documented in rare individuals, whereby evanescent increases in vision (described as "flashes" of clear vision) accompany a decrease in the overall plus power of the eye. Complementing less than a handful of dated studies, a few recent personal communications from eye care clinicians claim to have objectively assessed negative accommodation (e.g., by retinoscopy).

Whereas negative accommodation may explain informal accounts claiming occasional improved visual acuity in uncorrected myopes, its mode of operation is not established. We sketch a potential mechanism by which negative accommodation may operate, if it exists.

Received for consideration January 11, 2004; accepted for publication February 4, 2004.

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INTRODUCTION

In 450 B.C., nearsighted slaves were sold at a discount; with no way to correct it, the vision defect reduced their value. Indeed, trying to enhance vision has a long history dating to the use of crystals as crude magnifying glasses.

Today, several techniques purport to reduce or eliminate the need for corrective lenses in patients with refractive errors. Some of these methods (e.g., refractive surgery, orthokeratology) act by reducing the refractive error; other techniques - often based on behavioral training (e.g., optical exercises) or psychological conditioning (e.g., hypnosis) - target higher cognitive functions, promote ocular relaxation, enhance perceptual learning, blur interpretation, memorization, or create an impression that subjects can see better than they actually do (1,2).

Albeit amply critiqued (3,4) and debunked (5,6), some of these latter schemes (e.g., Bates Method, See Clearly Method) claim to work via both the refractive and cognitive modalities and have become lucrative businesses.

One claim free of commercial overtones, entrenched in folklore, and supported by spotty reports and rather elderly data, recently piqued our curiosity. Negative accommodation (NA) denotes fleeting and occasionally involuntary increases in vision (described as "flashes" of clear vision) which accompany a decrease in the overall plus power of the eye. Reportedly, NA could decrease the refractive error of myopes. One possibility offered by way of explanation involves the base tonus for the accommodative mechanism (7,8). This tonus, combined with the dioptric power of the lens and cornea, and the axial length of the eye, produces the total refractive state. Actively reducing the base accommodative tonus would therefore reduce the plus power of the eye and enable myopes to see better.

We outline a neural mechanism that could potentially account for claims of negative accommodation, if it exists.

A CLOSER LOOK AT NEGATIVE ACCOMMODATION

Accommodation produces a change in the shape of the lens to bring near objects into focus. Under scotopic conditions, and absent visual stimulus, the accommodative mechanism rests in an intermediate position, its "tonic" position, of approximately 1.5 diopters (D) with a standard deviation of 0.75 D (8.9). In the photopic states (i.e., when the visual apparatus is stimulated) there is a reduction of this resting accommodative tonus. Accommodation can be largely and even totally eliminated by cycloplegic drugs.

Some myopes report that on occasion they can briefly clear their vision and see sharply without corrective lenses, episodes referred to as "flashes" of clear vision. It is assumed that these "flashes" are the result of negative accommodation (i.e., active, neurally-mediated flattening of the lens) in combination with reduction of the base photopic accommodation tonus.

Scientific reports concerning NA are dated, sparse, contradictory, and largely anecdotal. Le Grand (10,11) reported data from five subjects who fit the profiles for negative accommodation with the exception that they could briefly clear their distance vision by *making a conscious effort* to see better. He notes that "... this effort seems fatiguing and quickly produces a sensation of discomfort ... and can be maintained only a few minutes." Three of the subjects were made myopic by having plus lenses placed in front of their eyes: the other two subjects were veridical myopes. In all five cases the subjects could reduce the power of their eyes 2-3 diopters. The fifth subject was cyclopleged and in this instance the 2.75 D change in the plus (+) direction was reduced by 50%, suggesting that part of the original change was a reduction of the base accommodative tonus while the other part represented veridical flattening of the lens from the traditional far point.

Attempting to explore NA further, Marg (12) examined individuals who could "flash". Following an exhaustive search, five such subjects were identified, and Marg concluded that only about 1 in 100 people were capable of "flashing". Unlike Le Grand's "flashers", however, none of Marg's five subjects experienced significant changes in refractive error while "flashing".

We have recently inquired among senior practitioners and colleagues seeking further substantiation for accounts of NA. Our multiple queries have yielded only a handful of ostensibly responsible unpublished clinical accounts concurring with NA phenomenology. For example, one of the co-authors (DLG) - a respected expert in ophthalmic optics and clinical refraction - reported (personal communication) having personally examined one individual who had taught himself to voluntarily and significantly reduce his myopic refractive error by at least 0.75 D, verified by concurrent retinoscopy in one eye. With approximately 1.50 D of myopia bilaterally, the subject, using mental concentration, could clear his vision intermittently to 20/15 while reading a distance chart. Curiously, uncrossed diplopia (exodeviation) occurred initially as the individual made this effort, suggesting a relaxation of the near reflex, but the pupil definitely constricted, about 0.5 mm by estimation, confusing the issue.

SYMPATHETIC INNERVATION OF THE CILIARY MUSCLE

The major innervation to the ciliary muscle is parasympathetic with fibers originating in the Edinger-Westphal nucleus of the third cranial nerve travelling with the inferior division of this nerve in the orbit, and synapsing in the ciliary ganglion. Post-synaptic fibers then extend to the ciliary body. Post-synaptic fibers form extensive plexi within the ciliary muscle and subserve accommodation. The parasympathetic receptors in the eye are muscarinic cholinergic. This alters the refractive power of the eye, allowing vision of near objects (i.e., accommodation). The sympathetic supply to the ciliary muscle subserves a reverse accommodation effect and functions as an antagonist to the predominant parasympathetic system. Compared to the parasympathetic system, knowledge of ocular sympathetic innervation was largely sketchier until recently and derived primarily from pathology (e.g., Horner's Syndrome) and drug outcomes (13).

Whereas there is progressive literature providing anatomical, physiological and pharmacological evidence for dual innervation of the ciliary muscle spanning more than a decade (14-17), clinical and psychological evidence is equivocal.

A review of optometric studies of tonic resting position of accommodation indicates that the inhibitory nature of sympathetic innervation suggested by the majority of previous studies can be further defined with respect to specific adrenergic receptors. Some studies have addressed the implication of dual innervation relating to ocular accommodation during sustained near vision tasks (17). This dual innervation model of the ciliary muscle - positive accommodation (i.e., increased lens power) by the parasympathetic branch and "negative" accommodation (i.e., relaxation of accommodation) via relatively weak inhibition of the parasympathetic branch - suggests that while there is sympathetic innervation of the ciliary muscle, it is likely to underlie a less vigorous process (i.e., modulating pre-existing sustained accommodation). In general, the sympathetic effects on the ciliary muscle are believed to act relatively gradually, far too slowly to counter accommodation under normal circumstances. However, it has been hypothesized that a startle response may cause a sudden decrease in the refractive power of the eye (15,18).

Although sudden sensory stimuli (e.g., electric shock or loud noise) may produce a rapid NA-like phenomenon, these effects are on average no larger than a quarter diopter in magnitude, even if longer lasting changes of 0.50-0.75 D can occur in occasional subjects (18). Upon shock, speedy release of hormones into the blood stream instigates a short lived sympathetic response that can "actively relax" the ciliary muscle. These hormones will likely cause a constriction of ocular blood vessels, possibly influencing the refraction and affecting accommodation (15). This impulse may cause NA and elicit a slight transient hyperopic shift in refraction.

It is possible that the sympathetic innervation might wield a larger influence than commonly held (19). However, a "shock" stimulus is unlikely to generate a hyperopic shift greater than 0.25-0.75 D, unless either pseudomyopia or abnormal accommodative tone is present. Toward this end, the NA phenomenon probably manifests differently among individuals as most myopes have only slight accommodative tone for distance fixation.

DISCUSSION

In subjects whose visual acuity increases without change in refraction, one possible explanation is that there is a heightened perception due to higher cognitive functions (e.g., attention), as is occasionally seen under the influence of suggestion and motivation, or following visual schooling and perceptual training (3,4). In contrast, those subjects who either volitionally or involuntarily reduce the power of their refractive error presumably do so through a change in lens power involving the accommodative apparatus.

The normal accommodative process is predominately mediated through action of parasympathetic branches of the oculomotor cranial nerve to the ciliary muscle. Parasympathetic stimulation results in rapid contraction of the outer longitudinal fibers of the ciliary muscle, allowing the inner circular oriented fibers to move forward, relaxing the zonules and resulting in increased anterior-posterior lens thickness and, hence, increased dioptric power. Relaxing the parasympathetic innervation reverses this process and flattens the lens, reducing its power, so that the eye fixes at its far point.

There are data that some individuals have a higher than normal level of accommodation active even during distance vision (i.e., pseudomyopes). For these subjects, blocking parasympathetic input might result in an increase in distance vision. For example, Le Grand's fifth "flashing" patient showed a 50% reduction in the hyperopic shift under cycloplegia, suggesting that at least some of the increased vision was due to over accommodation (10,11). It is possible that some "flashers" might simply be pseudomyopes who have learned to relax their over-accommodated state either entirely or partially. Subjects who lose their ability to flash under cycloplegia can be assumed to fall into this category

(20,21).

Alternatively, the role of sympathetic innervation to the eye, especially the ciliary muscle may have been undervalued. Stimulation of the sympathetic system results in pupil dilation and a brief hyperopic shift (e.g., reduced by timolol and induced by isoprenaline). However, the amount of accommodative response to sympathetic stimulation is slow and usually not significant compared to that of parasympathetic stimulation (22). The consensus among experts in ocular physiology is that the sympathetic system acts to inhibit overaction of the parasympathetic input during tasks requiring sustained accommodation or helps to determine the level and to stabilize the resting scotopic tonic accommodation. It is possible that patients who "flash" and whose flashes are not entirely eliminated by cycloplegic drugs may have learned to stimulate their sympathetic system and pull their lenses into a flatter shape than the normal rest position, thereby reducing the overall power of their eyes. This will appear in line with the phenomenology of negative accommodation. We hope to soon provide empirical data speaking to these issues.

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