NeuroAdaptation

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Neuroadaptation is the process by which the brain modifies its sensory input, in response to touch, heat, cold, pain, sight, sounds, or smell. Nervous system adaptation enables us to cope with a constantly changing environment. Because we are adaptive, our fitness to survive is enhanced. Modern humans have been successful inhabitants of this planet for approximately 160,000 years mainly because we are capable of adapting to change.

The adult nervous system is remarkably plastic. Numerous studies have shed light on the molecular basis for this fascinating process from the regulation of stem cell function to its influence on behaviors. In the adult, neurogenesis, including
adaptive roles in learning and memory, changing environments, depression and moods, and responses to injury including neuropathic pain, are all linked to the hippocampal region of the brain. The hippocampus is a horseshoe shaped segment of neural tissue that sits between the temporal lobes and next to the amygdala. It is responsible for consolidation of memory, emotion, navigation, and of importance to ophthalmologists, spatial orientation. Research has revealed an intimate relationship in this area between neuroadaptive mechanisms and addictive behavior.

Our need to understand the underlying mechanisms of neuroadaptation, as it relates to visual function, has once again taken center stage. It has become a poignant topic of conversation between physicians who must treat patients, that as a result of an alteration in the visual system, are struggling to adapt. Patients who undergo a modification in the visual system, whether induced by the wearing of a new pair of eyeglasses or bifocals, or by having undergone a corneal ablative procedure or lens implantation with the novel optics of multifocal intraocular lenses, will undoubtedly be challenged by newly created perceptive change. How quickly and how well they adapt to this will ultimately determine whether they become evangelistic believers in this new technology, or whether they will disparage it and their physicians for the rest of their lives. While many patients adapt quickly and successfully to a surgical procedure, some adapt slowly, if at all.
As ophthalmologists, we can be stymied by the dissatisfaction of an unhappy patient, especially in the face of a successful visual outcome from a surgical procedure. What factors are at play that can allow one individual to embrace refractive revision and another to reject it? The answer to these questions remains within the realm of this rich and elusive process called neuroadaptation.

Neuroadaptation can occur within the visual system in response to either a monocular or binocular visual disturbance. Visual adaptation depends to a great extent on visual awareness. In the case of a monocular visual disturbance, the brain learns to compensate by altering its perception. It has been shown that even in cases where a clear image is focused onto the retina, that neuroadaptation may still be required if there are inherent optical aberrations within the visual system that the brain cannot accept. Given time, the mind applies its negating effect to the undesirable pattern. Should the aberrations be eliminated (as in wave-front enhanced, custom, excimer-laser ablation), then the brain will, at least for a period of time, apply the negating effect that it has previously learned to this new clearer image, thus degrading it. Ultimately, if age and time work in the patient’s favor, than the final image quality becomes acceptable. Both patient and physician must be cognizant of the nuances of this process. It is not acceptable to simply tell a patient to “give it some time.”
To better understand this phenomenon of neuroadaptation, let’s use as an example, meridional anisokonia. Meridional anisokonia occurs when there is a difference in the astigmatic refractive error between the two eyes. It is a binocular phenomenon. It can occur in either eye and can result in amblyopia if it appears early in life. Strabismic amblyopes who do not have significant astigmatism often exhibit a decrease in contrast sensitivity measurements when tested with vertical gratings rather than with horizontal. This is believed to be the result of horizontal image displacement in the deviating eye. The more ametropic meridian in highly astigmatic individuals can be associated with a marked reduction in acuity despite optical correction. What is going on? A defocused image in one meridian can actually prevent the establishment of normal neural pathways from eye to occipital cortex. What happens if a previously nonexistent astigmatic error is inadvertently introduced? The post-operative patient with an increase in magnitude or change in direction of an astigmatic refractive error must adapt to the visual perceptive change. First, the brain becomes confused with the new imagery. The conflict must be resolved if the patient is to accept the refractive change. A lot of the ability of an individual to resolve such conflict may rest more with their chronological age, than with the degree or magnitude of the refractive error. What we do know is that the “plasticity” of this neuroadaptive process is a function of age, the younger the patient is, the more likely they are to accept this newfound perceptive alteration.
Time in this case, is not on our side. If the patient is fortunate, then the neuroadaptive process will take over and the final image quality will be perceived as satisfactory. This is precisely how, when confronted with the first pair of bifocal glasses that cause an intolerable blurring at first, the new presbyope develops a level of acceptable visual function within a matter of days. Remember the patient’s postoperative complaint of *erythropsia* prior to the advent of ultraviolet-blocking IOLs? By the second postoperative visit, the complaint was gone, when obviously the altered visual perception was still present. The brain had adapted.

Stereopsis, or 3D vision, is the ability of the binocular optical system to merge two images, one from each of the slightly disparate parallax points of view from each eye that provides an enhanced perception of depth. When we as surgeons, intentionally disrupt the “one-eye, one-image” perception that is required for successful merging of the images from two eyes, we can create a perceptive paradox that the brain simply cannot undo. An example of this chicanery is the newly embraced portfolio of the multifocal IOLs. By requiring the simultaneous perception of multiple images, in focus only at the differing focal lengths created by these optical marvels, we undermine the ability of the optical system to adapt. Some can ignore the perceptive annoyances, others cannot, and the success of these IOLs depends entirely on the brain’s ability to act on the disturbance.
The study of neurodaptation is based primarily in psychophysics. Two extensively studied phenomena are known as binocular rivalry and visual crowding. These visual phenomena are capable of erasing visual stimuli from conscious awareness. Unlike factors that lead to visual processing early in the system, processing of these phenomena occur within the primary visual cortex (V1) and the middle-temporal visual areas. Brain imaging and EEG studies have demonstrated that suppression of unwanted images during retinal rivalry reduce the visual stimuli perceived in the monocular regions of V1 and keep them from conscious awareness. Randolph Blake and colleagues at the Department of Psychology of the Vanderbilt Vision Research Center at Vanderbilt University have extensively researched this area and suggest that suppression of vision rivalry and crowding involves a reduction of neural activity not an increase or elimination.

Just how the brain recruits the neurons to make this happen is a scientific fascination in itself. Just as every processing point along the visual pathway contributes to the final, clearly perceived optical image, an interruption in the smooth flow of information can become problematic. Until the image signal hits the sixth-order neurons, both images are monocular. It is here where ocular dominance and retinal rivalry exists. From the lateral geniculate bodies, the images begin to fuse. Flood these centers with retinal signals from multiple images and the deep centers of the brain that need to make sense of the chaos begin to fail. Neural
adaptation associated with both retinal rivalry and image crowding occur at the earliest stages of visual processing. The fact that neuroadaptation to this visual disparity can happen at all is testimony to the amazing plasticity of the system. For it to occur over a period of weeks to months and even years, signifies that complex neurogenesis is at work. It takes time to make new neural connections or suppress old ones.

Neuroadaptation begins at the beginning of life and remains an encompassing, ongoing phenomenon, unless something intervenes to disrupt it. When light first hits a baby’s retina at birth, the startled look in the baby’s opened eyes reflects a dramatic flood of information to the occipital cortex. The hardware is there, but the software has not yet been developed. The earliest images that reach the occipital cortex will be inverted. Neuroadaptation flips them cortically so that up is down and down is up. The brain continues to process visual stimuli throughout life and make sense of the images the retina receives. When reading, our eyes move in spurts across the page. To meld the saccadic movement of our eyes into a smooth perception of letters and words requires higher cortical processing. The brain adapts to the information from these images and combines them across glances. If, during the course of our lives, we lose the ability to modulate visual information, than retraining the brain to perceive visual stimuli differently is required. That is precisely what is done for vision rehabilitation in
cases of injury or disease, such as in stroke patients and those with age-related macular degeneration. Permanent damage to the processing areas in the primary visual cortex may unfortunately render neuroadaptation useless.

What should our patients expect when we, as surgeons, modify a lifetime of visual perception with one quick stroke of a diamond blade or a laser beam? It is certainly intriguing to postulate a pivotal role for the neuroadaptive mechanisms, which can act as friend or foe, in predicting which patients will accept visual perceptive change and which will not. The psychological implications of neuroadaptation, which have been well studied in addictive behaviors such as drug dependence and excessive gambling tendencies, may provide some insight. If the same regions of the brain are involved, and they are, can there be a potential link between addictive personalities and failure to accept and adapt to refractive change? Should we be avoiding risk-taking personalities as poor candidates for cataract and refractive surgery? Certainly more work is needed in this area. What is certain however, is that our assessment of our patients behavioral, social and psychological needs are at least as important as the analysis of their A-scans and wavefront maps.
References


