Myopia As An Adaptation
By Robert Lichtman

Reading has been a suspect in the etiology of myopia at least since a correlation between education, literacy, and myopia was mentioned by Kepler in 1611, but however attractive this theory is, empirical data to support it has not been forthcoming (Mutti & Zadnick, 2009). The obvious flaw is the fact that we have near universal literacy, but a much lower myopia rate of about 25%. The fact that some people develop varying degrees of myopia while others doing the same work do not implies, in the acknowledged absence of strong data to support a role for genetics, near point work alone is not the culprit.

The earliest modern theories of the etiology of myopia incorrectly and simplistically posited the eye as being analogous to a camera. Although this view is simplistic and outdated, it remains the basic way the eye and visual disturbances such as myopia are still understood. Neurology and the role the brain plays in visual processing, largely unimaginable to 19th century scientists such as Helmholtz and largely ignored by modern "neighborhood" vision professionals, has caused a revolution in our understanding of how visual perception works. However, even contemporary literature tends to overlook a critical element: the role of the conscious "I" in seeing. For unlike the inanimate machines used in photography, the human eye is controlled and influenced by a consciousness that thinks, feels, and does what a machine can never do: subordinate the correct use of the visual system to other needs.

In particular, fear and anxiety play a large role in the production of myopia by introducing factors that affect the ability to form a clear image on the retina as well as regulate the elongation of the eye. I will propose a theory as to how emotions become thoughts, thoughts become motor impulses, motor impulses affect the eye and visual cortex, and how these phenomena ultimately blur vision. It is important to note that since these processes are dynamic and since there is no organic damage to the myopic eye, the condition is reversible.

The development of the eye is governed from birth by a process called emmetropization. Infants are born farsighted and gradually learn how to accommodate to the near point in a process typically completed by age 7, not long after children start learning to read (Wallman, 2004). In addition, an infant's contrast sensitivity function is less sensitive than an adult's up to the first year (Blake and Sekular, 2006). We essentially learn to see in the same way we learn to walk or acquire language: by gradually building skill. The eye corrects for blur and other errors in accommodation by changing the axial length of the eye. For example, if the image falls beyond the retina, the retina sends signals to the choroid to stretch read (Wallman, 2004). This increases the axial length to that necessary for clear vision. This system is better than one in which the eye was acquired "off the shelf", for in this way the normal development of the eye can take into account environmental factors rather than be one size fits all.

With this understanding of emmetropization we see how the axial length of the eye is dynamic and regulated by the retina. In fact, elongation is a misleading word because it implies the eye simply grows longer the way a leg grows longer. Instead, the elongation of the eyeball happens by a stretching of the choroid. This elongation is not the cause of myopia, but rather an adaptation to it. People in my experience tend to view myopia as a "normal deformity", but it is not simply a natural state of blur. Rather, it is a condition where the eye is under stress. Indeed, high rates of myopia correlate to higher risks for eye diseases such as macular degeneration, cataracts, and glaucoma (Cassel & Randall, 1998). This begs the question of what type of context can induce the eye to adapt by diminishing its function. To answer this question, it is helpful to view the eye as subordinate to the individual whose brain it is but an extension of, as a loyal soldier who obeys the commands of the commander whether or not those commands are appropriate.

Blur begins with a fear of not being able to cope. In hundreds of interviews with myopes conducted by the author, those who became myopic before the age of 15 tend to report the onset of myopia as occurring within a year or so of a stressful situation such as moving to a new country, problems at home, learning to read, or adolescence. Late onset myopia most often occurs during college, when perhaps the stress of being away from home mixes with the stress of reading large amounts of unattractive material and being forced to write papers on deadline.

Those who do not report any problems at first will typically remember them later. The rest represent a small enough sample to suggest they either do not remember or do not wish to share the information, although other situations such as dim light or too much sugar in the diet can defocus vision (Cordain, et al 2002).

Nevertheless, it is not these events that induce myopia. Different people will respond to situations differently. Most people will respond to this kind of situation with a sympathetic nervous response that does not last more than a minute or so. In some people, however, this temporary state becomes chronic. Sympathetic excitation is a mobilization of the entire body more or less simultaneously for purposes of fight or flight. Symptoms include increased heart rate, respiration (Geitman, 1981), and two that are of special interest to myopia: Pupillary dilation and hypermetropic accommodation, that is, the eyes adjust to the distance (Morgan, 1939). When we consider sympathetic arousal as being part of a fight or flight response, this makes sense. We might want see into the distance to find an escape route, or open the pupils to see better in dim light.

However, this fight or flight response is more appropriate for hunter-gatherers than it is for children in schools. Pupillary dilation makes it more difficult to see by shortening depth of field, the range in front of and behind the point of fixation. Conceivably, a child used to seeing a greater depth of field can be distressed by a loss of depth of field. In addition, the onset of myopia is preceded by accommodative lag. Accommodative lag means the eye does not accommodate for the exact distance necessary (Nakatuska, 2005). This is normally not a problem, since depth of field is usually large enough for the image to be in focus despite the lag. But accommodative lag accompanied by the loss of depth of field due to a sympathetic response would result in blur, triggering the adaptation mechanism that ultimately fails.

Now we turn to hyperopic defocus. The child struggling with a book at the near point is not at all helped by his eyes autonomically accommodating for the far point, as this hyperopic defocus will blur the text in his hands. In addition, it is not difficult to imagine how this defocus can create a sense of a loss of control, adding to the vicious cycle of anxiety and defocus.

If the child were simply to look into the distance during this time, there is a good chance the diversion will allow the sympathetic response to run its course. In fact, this “cure” was first suggested by Bates (1920). Many children instinctively do this, but others may feel pressured by teachers or parents to “pay attention” and “try harder”. When the child responds to this condition by redoubling efforts to focus on the near point, not only is the sympathetic response reinforced by the fear. In addition, the retina adapts to this state by sending signals to the eye to lengthen in order to see clearly *in spite of*, or in compensation for, the sympathetic response (Wallman, 2004). Alternately, the child may attempt to overcome the hyperopic defocus by trying to clear it by straining the extraocular muscles. My own clinical experience suggests that just as anxiety can trigger muscle tension, muscle tension can trigger anxiety. Once the strain becomes chronic, the individual is producing anxiety even in the absence of external stimuli.

The extraocular muscles (EOMs) are the six muscles that surround the eyeball and whose sole function is to move the eyeballs laterally and up/down (Blake and Sekular, 2006). They are also the only muscles associated with vision the mind can seize conscious control of. In other words, I cannot consciously contract my ciliary muscles, but I can grab my extraocular muscles. Despite this, using the EOMs for anything other than up/down and lateral movements is a strain, as seeing is essentially a passive function. When a person tries to see, he will inevitably grab the eyeball with the EOMs. This “grabbing” happens in one of three contexts I have identified in order to try to control vision, though all of them have the same effect: to suppress eye movements.

The first case being to compensate for the sympathetic response, in the second case the strain is the result of the difficulty the child has in attending to schoolwork because it is simply not attractive to him. While some children will drift off and let their schoolwork suffer, others will yield to social pressure to do well and attempt to force themselves to attend. From here comes the correlation between education and myopia, though it is really a correlation between being willing to submit to strain for the sake of social approbation and myopia. Recall attention is a state, not an activity. You cannot “do” a state. In the attempt to attend, the child will lock up his entire body from the pelvis up, culminating in grabbing the eyes with the EOMs in an attempt to force voluntary attention. This is extremely fatiguing, but it appears to the individual to work

(Ribot, 1890) and is rewarded by society. Still, it is heartbreaking to watch a child do this, sacrificing her spontaneity unnecessarily at the altar of civilization.

Another corollary to forcing oneself to attend is the notion that concentration means the ability to think or do one thing only. This is manifestly impossible, as our attention is always a duality of attending to one thing while being aware of everything else. The attempt to block out all but one stimuli is not the default state of our vision, and so requires effort in its doomed attempt. In reality, concentration is thinking or doing one thing *best*, not one thing only. Educating people to this fact is a first step towards learning how to effortlessly concentrate.

The third case leading to blur and strain of the extraocular muscles tends to follow poor deduction. We normally attend visually to an area no larger than the area of the fovea, about two degrees of the visual field. By constantly shifting attention, we create for ourselves the illusion of attending to everything at the same time (Yarbus, 1967). Again rooted in fear of missing something, or being blindsided by the periphery, some people decide to expand the area of best attention in an attempt to see as much as possible in one fixation. Some people report the motive for doing this is to save time. Others say it makes them feel safer. Both reasons do not stand up to simple experiments of attending both ways and comparing the two.

It is impossible to see as clearly outside the fovea as one does inside the fovea due to the distribution of cones in and outside the fovea, the lower density of ganglion cells in the eccentric field, and the fact that receptor fields get much larger in the periphery (Blake & Sekular, 2006). The attempt to equalize vision both in and out of the fovea is accomplished by lowering acuity to the furthest point that is both outside the fovea and inside the area a person wishes to include in his "big gulp" (Bates, 1920). Recent studies using photorefractors to measure peripheral refractive error demonstrate that in fact foveal myopia is accompanied by peripheral hypermetropia (Siedmann, et al., 2002) due to the pressure the contracting lens exerts on the eye ball. Emmetropes do not exhibit this peripheral ametropia, further giving lie to the notion that attending to a large area at one time somehow heightens awareness of the periphery and thus makes one safer.

While the blur is the result of voluntary motor activity, it was not intention of the individual to blur. Quite the opposite, in fact, so he does not connect the effect with the cause. It is easy to demonstrate this to a myope by asking him to look at a letter on a chart and attend to only a part of it, rather than the entire letter at once. If the letter is small enough, he will not be able to limit his attention to just one part of the letter. Being able to distinguish points one arc degree apart is the definition of normal vision (Blake & Sekular, 2006), and this seems impossible to a myope accustomed to grabbing larger areas in a single fixation. At the same time, asking an emmetrope to expand the area of attention will produce myopia within seconds.

While this spreading out of attention happens in the visual cortex by ignoring higher frequency spatial fields (the reason why, connected to mental imagery, will be explained below), once again the extraocular muscles are needed to hold the eyes in place as the individual somewhat consciously suppresses saccadic motion in exchange for a bigger, albeit blurrier, image.

The common denominator in all these failed efforts to see clearer is straining the extraocular muscles. Under normal use, these muscles have the ability to carry 150-200 times the weight they need to pull and perform 100,000 movements a day (Kavner, 1978). According to Fuchs and Binder, under normal use they never fatigue (Blake & Sekular, 2006). When misused, however, they do fatigue and very quickly fatigue the individual. This is why going for a five mile run can be more relaxing than sitting in front of a computer all day. Correcting myopia starts with relaxing the chronic strain of these muscles in the near point, where the strain is not strong enough to blur vision, and extending that sense of relaxation into the distance by inhibiting

We earlier discussed the process of emmetropization as a feedback between the retina and the choroid. When discussing the feedback that causes myopia, a sticky question comes up: how is it advantageous to blur vision and create strain and fatigue? The first answer is that it is not, but an individual who believes there are no other options will endure the difficulties. In fact, the most common treatment for myopia, corrective lenses, require the individual to maintain the strain in order to see through them. This reverses the feedback, as now strain is necessary to see clearly and grabbing a large area results in clear vision. In addition, minus lenses have been

shown to promote myopia in several other ways. They actually promote elongation of the eyeball, increase accommodative lag, and encourage peripheral defocus (Nakatuska, 2005). Beyond all this, though, there is one intriguing if not sinister advantage to myopia: while anxiety brought on by the fear of not living up to expectations may be an underlying cause of myopia, myopia ironically becomes somewhat of a palliative for anxiety. In her doctoral dissertation, Carolyn Ziegler showed myopes reported more anxiety than emmetropes, but only up to about -3 diopters of myopia. Beyond that, the anxiety levels went *down* as vision got worse. By around -6 diopters, the border of severe myopia, the anxiety levels of the myopes were similar to those of emmetropes (Zeiger, 1976). Her work further suggests that treating myopia would concomitantly treat anxiety, though severe myopes would likely report an increase in anxiety as their vision improved into the -3 diopter zone. This has also been corroborated in my clinical experience, and persons improving their vision need to find new strategies to cope with this anxiety. In some cases, habituation to clearer vision is sufficient.

It is easy to understand why myopia would cause anxiety if the condition is induced by anxiety and characterized by chronic eyestrain; try walking around making a fist and carrying around the fear that if you let go of the fist, you won't be able to pay attention, concentrate, or maintain your GPA. In my interviews with myopes, the fear of letting go is not only a fear of losing control, but also a fear of losing parental love. This is even true for adults, who apparently had the thought as children, took protective measures (held on), and forgot the thought while remembering to hold on. But why would increasing the strain lower the level of anxiety? The answer seems to involve the convergent role of imagery in both vision and anxiety.

Bates (1920) was the first to note the link between imagery and vision, actually developing a protocol to restore acuity through mental imagery. His findings are supported by the work of Steven Kosslyn. Kosslyn and his team found that in both cases of looking at an actual object and visualizing it, the same part of the occipital cortex, Area 17, was stimulated. This is astounding, because it implies that there is a level in the brain where there is no real difference between the experience of seeing something and the experience of visualizing it in the mind's eye (Kosslyn, et al, 1993). The idea that forming imagery can improve vision supports the notion that myopia happens first in the brain. Perhaps the changes initiated by the conscious "I" cause the individual to ignore high frequency receptor fields. Or perhaps the high frequency receptor fields are desensitized by the suppression of movement when the individual locks his attention. Either way, it seems the act of remembering having seen something requiring high resolution spatial fields is enough to restimulate them, or more accurately, stop suppressing information from them. At the same time, it stands to reason that suppression of mental imagery would suppress, or dull, the high frequency spatial fields and thus blur vision.

How does one come to suppress imagery? Borkovic proposed that worry is a verbal activity intended to suppress imagery. It seems as if the experience of imagery is so similar to actual vision that visualizing disturbing things causes anxiety (Behar, 2005). Wilhelm Reich and others have defined anxiety as a holding back of emotion. The basis for not expressing an emotion is fear of a consequence, and this holding back causes discomfort in the form of surplus energy meant for the expression of the emotion but instead bound up in the musculature, including the EOMs (Baker). Borkovic found this anxiety linked to imagery-based worry. His experiments support the notion that a switch to verbal-based worry from imagery-based mentation inhibits cardio-vascular activity. In other words, it appears to calm us down.

Appearances can be deceiving, though. The discomfort of the cardiovascular response to image based worry limits how much worry a person can tolerate. Switching to verbal/thought based worry allows a person to worry much more without feeling the effects of the worry. In other words, switching from imagery-based worry to thought-based worry does nothing to reduce the anxiety, but it is similar to taking a pill that blocks the perception of the anxiety. Just a pain killer allows an athlete to hurt himself even more, thought based worry allows a person to worry and be paralyzed by that worry so much more. At the same time, our school systems are biased toward thought-based mentation so the behavior is rewarded, engaging the child in a vicious cycle that may last his entire life.

The mechanism by which we switch from imagery-based mentation to worry based mentation may involve a suppression of high frequency receptor fields. If so, excessive worry would induce myopia at the same time it palliates anxiety. This resolves the problem of why

people would blur their vision and take on chronic strain: in order to reduce the effects of anxiety. While anxiety's purpose is to provide extra energy for a fight or flight response, in our modern world it actually makes our decision-making processes less effective. In their study of how we process fear, Foa and Kozak found anxious persons exaggerated "subjective personal risk" and made other, similar errors in judgment (1986). Again, the problem is the sympathetic nervous system's all or nothing response; every crisis is dealt with the same way we would want it to deal with a tiger chasing us with no shades of gray (Levine, 1997).

We have previously discussed the role of locking attention as a flawed, mistaken means of actively focusing and how that locking serves to not only suppress high frequency spatial fields via adaptation, but also to desensitize the retina by depriving it of the re-stimulation it requires and typically gets through saccadic motion. As Blake & Sekular explain (2006), the fact that stabilized retinal images are invisible is necessary; otherwise, we would see the blood vessels in front of the retina. This only becomes a liability when saccadic motion is suppressed. While we perform around 70 saccades per second, we are aware of only 1-4 per second (Yarbus, 1967). These 70 saccades per second are necessary to keep the image from stabilizing on the retina, and thus disappearing. However, reducing that number to less than one per second has the effect of suppressing the unconscious ones, leading to retinal desensitization and blurrier vision within five seconds. This can be observed by staring at a sinusoidal grating for more than 6 seconds, after which the eye adapts to the contrasts and it starts to fade away. Holding the eye in place under the wrong notion that it will encourage focus or attention thus actually has the opposite result, the *loss* of the image. This is true of imagery as well; close your eyes and imagine a stationary object, and it too will fade after about a second. Let it move and you can hold it indefinitely. Letting a mental picture move requires one to let go of the EOMs, as bizarre as that sounds. It's not clear why muscles that control eye movements would have an effect on what we see in the visual cortex, but they are nevertheless a key to understanding how we shift from mental imagery to thought based worry.

There is another way in which eye movements may serve to suppress mental imagery. Since the early 1970s, it has been known that people use lateral movements of the eyes as accessing cues. For example, the majority of right-handed people will look up and to the left to remember something seen and look laterally to the left to remember something heard. Looking down and to the left accesses "self talk". This happens to also be the position the eyes are moved to read in English, suggesting the very act of reading may play a role in suppressing imagery (Huang & Byrne, 1978). More research needs to be done concerning the link between rigidly holding eyes in the down position and the suppression of mental imagery.

A condition that now afflicts 25% of the American population and a higher proportion of persons in emerging Asian nations surely counts as an epidemic. Yet despite all the research done on myopia, our understanding has not borne useful fruit towards curing the affliction. It is my contention that research into myopia has treated the eyes as optical equipment purchased at a consumer electronics store rather than as an integral part of our conscious selves. Viewing myopia as an adaptation to chronic misuse opens up many doors to understanding what factors cause one individual to be afflicted and another not. More encouragingly, it suggests a means whereby the condition can be reversed. It is time to lay to rest our nineteenth century notions of the visual system along with our 16th century solution, the concave lens, and direct research towards a cure based on the cause and not the effect.

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