TOWARD A UNIFIED THEORY OF AMETROPIA: 
REFRACTIVE ERROR AS A LESION

(Original title: Preventing Refractive Error: What’s a Doctor to DO?)

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INTRODUCTION

My premise is that refractive error is disease. Myopia is a lesion. Astigmatia is a lesion. Hyperopia (in the range of adverse hyperopia, which clinically appears to be that greater than 1.00), is a lesion.

Myopia, astigmatia and hyperopia have already been described as adaptive stress diseases. You have a list of my papers in the program that will give you a more in-depth understanding of how I have come to the conclusions you are about to hear. I invite you to listen carefully to see if you find exception with what I am about to say.

[ O/H #1 - Paradigm Shift]

[CHICK, STANDING BESIDE OPEN EGGSHELL: “OH WOW! PARADIGM SHIFT!”]

Optometry has been in a shell for too long: the shell of the medical model, or the classic eye-ball model, or the merchandising model. It is time for optometry to come out of its shell and open its professional eyes to a whole new paradigm: Refractive Error As Adaptive Disease.

Skeffington was only one of the first of our professional fathers to point out that;

[O/H #2 - Refractive Error]

[Refractive error is the last stage of a visual problem.]

People may think that it is a bad thing to be myopic, or hyperopic, or astigmatic, but to the body, these are good things, not bad. They reflect and reinforce the way the body has chosen to cope. Myopia is a wonderful way of pulling a spatial problem in to assimilate it. Hyperopia is a wonderful way of pushing it away, glossing it over, or running away from the problem. Astigmatism is tricky - it is part of the transition to myopia, or the body’s concession when it wants to run away, but is being constrained to perform.

The trigger is stress, and its impact on the individual.

[O/H #3 - Stress Adaptation]

[VISUAL STRESS → ESCAPE (COPE) →
1) RETREAT: FT./FLIGHT; STOP WORKING;DAYDREAM = “LEAVE”
2) TOLERATE: UNDERACHIEVE; SOMATICIZE; SWITCH MODES
3) ADAPT: MYOPIA; ASTIGMIA; ADVERSE HYPEROPIA; AMBLYOPIA; STRABISMUS]
But, wait!

What about genetics? What about animal deprivation studies? What about nutrition? And, What about Naomi? What role do we play as clinicians?

Let’s take the last question first. Since ametropia is almost certainly a disease process, we clinicians need to concern ourselves less with what an eye is, myopic or hyperopic, sick or healthy, but instead look at what the visual system is becoming - whether it is “sicking” or not.

So then, if we are going to be looking for disease, then we have to talk about the disease process.

**DISEASE**

There is a spectrum to health and “normal” and “abnormal” are the polar opposites on that continuum of health. Disease can be defined as the changes within individuals that cause their health parameters to fall outside of the range of normal. When the structures or function of an organ or tissue system deviates so much that normal homeostatic processes can not restore the balance or if they are destroyed, or cannot meet the environmental challenge, then a disease is said to exist. ¹

Diseases are extensions or distortions of the life processes. The stressing agent itself does not constitute the disease - it merely evokes the response, the changes, that are manifested as disease. A disease therefore is actually the sum of the physiological processes that have been distorted. The elements of a disease actually lie within the very mechanism of adaptation.

The symptoms of a disease are subjective, and the individual must be questioned to elicit them. But when they are visible to the observer or examiner, they are called signs of a disease. When there is a demonstrable structural change, we call it a lesion. Ametropias fit the description of lesions.

Our optometric patients live, somewhat comfortably, in the sub-clinical or pre-clinical stages of disease, until the problem manifests itself - frequently on a school Snellen screening test. It takes an advanced stage of a visual problem to show as a visual acuity impairment. The pathophysiology has been in place for some time before we can see the signs.

Learning disabled children have a notorious lack of myopia, the literature shows² -- is this perhaps a reflection of their failure, or inability -- or unwillingness -- to convert symptoms into a disease? It looks suspiciously so.

What are the causes of Ametropia, and as clinicians, what can we do to intervene? As doctors, it is our responsibility to create the environment for healing to take place -- or to prevent disease in the first place.

**ETIOLOGIES**

[O/H #4 Etiologies]

[ 1) GENETICS: INADEQUATE, UNAMENABLE
2) NUTRITION: Frustrating, Somewhat Amenable
3) FUNCTION: Most Recurrent, Most Amenable]
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There are only three possible causes of Ametropia:

1) Genetics are involved, for sure. However, a genetic model allows for little means of amenability - we can’t intervene in meaningful ways if our Genesis is totally responsible for all Ametropia. We have little or nothing to talk about, with the current understanding. Mutti and Zadnik found that a parental history of myopia had a 25% predictive validity. Angi and Clementi (et al) found, by comparing mono- and dizygotic twins that heritablility of refractive error was low – from .08 to .14.

Hauge holds that heretability studies have limited value; and after an initially moderately strong initial coincidence, the identical twin studies show a growing inter-pair difference as the twins grow older. So while genetics is indeed a factor, it certainly isn’t the only factor, and may not be even a particularly strong factor.

Zadnik’s Orinda longitudinal study began with a great idea: to study the genetic background of a cohort and the follow the group’s refractive status for an extended period. Unfortunately, as critics have pointed out, there are a number of flaws: the parents’ refractive error was not determined in a fully scientific way. There was only a questionnaire of what age the parents began wearing any glasses, and classifications made from that information. It dilutes the importance of any conclusions that will be reached. There are at least four other areas of concern about that study. Hopefully these can be corrected.

All in all, genetics is an inadequate argument for ametropia. Young’s Eskimo studies, his point that atropine and other cycloplegics shouldn’t work, and the advent of larger than ever numbers of late onset myopes all point to extra-genetic mechanisms for a significant number of myopes. The question isn’t nature or nurture, its nature and nurture: what is the mix in this disease process?

2) Nutrition is a frustrating possibility of an etiology. We are what we eat, just like our mothers told us, and Lane’s work has implicated a number of processes involving magnesium, chromium, calcium, and vitamin C. He also implicated diets high in refined carbohydrates. British studies have suggested diets low in protein. (That study may have actually been factoring calcium, not protein, but that is a discussion for some other half hour).

In 1995, Dr. Lane reported that food folates (folic acid) were strongly associated with reversal of myopia, with a correlation of .935 and a confidence level of .001 from ages 18-38. From ages 6-17, the relationship was a bit weaker, with a correlation of .82 and confidence of less than .005. Pharmacological additives did not get equivalent results. We must eat foods in their nearest to natural states, he is still saying.

Why do I say that nutrition is frustrating? Well, we can change our diets, but how do we change a child’s tastes? And our American soils are depleted in minerals since 1936, according to a Government
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We need to eat well, but what? I will give you a handout later that helps work through this dilemma. So we have to consider that nutritional distortions are at least somewhat amenable.

3) Functional - this etiology is the most recurrent theme in the literature of the last 15 years or so.\textsuperscript{18,22} The impact of the environment is right now, in fact, the most discussed of all the factors. Animal deprivation studies and pharmacological studies are testimony that we can alter the eye’s environment and function as well as observe changes in the eye. A functional approach to control of myopia is the clinician’s only really productive tool in ametropia prevention.

Unfortunately there is no coordinated effort in exploring the domain or scope of refractive error. Hyperopia is treated as a non-problem, astigmatism is a nuisance and myopia is regrettable.

Any discounting of refractive error is in error. Scientists and clinicians call for proofs yet many of them are just defending their own inertia. They do almost nothing for their patients’ welfare while they take the money to the bank and wait for “George to do it” – ametropia research, that is.

The literature supports no fewer than 19 different possibilities for the etiology of myopia. That means any of millions of possible combinations may be at play in the patient before me. What can any doctor do, really? In my paper, Stress and Eye, I showed how the 19 combinations can be reduced to just seven, with Genetics at the top of the list.\textsuperscript{23}

\begin{verbatim}
[O/H #5: 7 Classes]
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\begin{verbatim}
[ 1) GENETICS; 2) MALNUTRITURE; 3) STRESS AND APPERCEPTION; 4) ENVIRONMENTAL FACTORS; 5) INFORMATION PROCESSING; 6) PATHOLOGICAL SEQUELLAE; 7) EXPERIMENTAL ESCAPADES]
\end{verbatim}

All seven, the paper goes on, reduce to one: Stress. Stress is a component of all disease.

However, to paraphrase Herbert Weiner’s observation on disease, may I say that, “Refractive Errors are mere abstractions. They cannot be understood without appreciation of the person who is ill.”

Robert Sapolsky, a notable physiologist from Stanford,\textsuperscript{24} observes that the psychology impinging upon a system is as decisive a factor as the physiology of that system. If we can change the way even a rat perceives his world, he says, you will dramatically alter the likelihood of its getting a disease.

Because of observations like these, we must investigate Who the ametrope is, What his environment is like, and How much time he spends in stressing environments, both visual and emotional.

ANIMAL RESEARCH

But what about all the animal research? Well what about it? Let’s consider the deprivation studies.

The study of chicks may be a poor choice of species to generalize from, since bird eyes are quite different than mammalian eyes. The scleras are comprised of Type 1 collagen, not Type 2. The ciliary
muscle is striated, not smooth and thus under different control. And studies with species that do not
have a frontal, binocular visual system, so that all parameters can be studied, is a liability, as well.

Zadnik pointed out that the ages of these animals is nowhere near comparable to human ages for
the onset of myopia.25

The pharmacological studies with pirenzipine seem promising, but Devadas and Morgan found
last year that its action is dose-dependent, working only at very high concentrations, rendering it and
others that they studied as physiologically insignificant, they said.26

McBrien, Leech and Cottrial found about the same thing, with intravitreally-injected pirenzepine
working well, but with subconjunctivally placed pirezepine not working nearly as well.27

McBrien also did an interesting study with chicks and atropine which may have far-reaching
implications. They found that form deprivation myopia was prevented by intravitreally placed
atropine.28 The study is long and complex, but I found it very interesting that not only the posterior, but
the anterior chamber was smaller in the atropinized experimental and control animals. It suggest that in
addition to any retinal/scleral effect, that the whole eye may be shrunk by the chemical. If that is so –
that the atropine directly affects the collagen protein of the sclera – then it would lend indirect support
to the physiological difference between myopes and hyperopes, since atropine only reveals latent
hyperopia in hyperopes and causes very mixed results in myopes, actually increasing myopia in some.29

Returning for a moment to consider the animal studies, the results are quite exciting (one hardly
knows what to expect, actually). The results vary on whether you use white or black occluders, minus
or plus lenses (which has been hit or miss within studies), form deprivation only, total deprivation or
partial deprivation, what animal and what species of animal (Rhesus and Stump-tailed Macaques have
different responses). It is all a bit mind-boggling in complexity.25

Then, once again, as Zadnik questions, is it relevant to humans? These animals correspond to
humans under six months of age and with some rare exceptions that will produce form deprivation
myopia in humans, no visual deprivation of similar magnitude occurs in children.25

These experiments are distracting to the average clinician who wants to help prevent visual
problems and enhance a person’s visual and cognitive performance. It is as if we have left sound and
productive methods and research behind because of various investigators who are infatuated with
novelty. We need to have reasonable approaches: we would like perfect, proven approaches, but we
are not going to have these for another 10-20 years – and only if we can get out of the experimental rut
we are in. We have lost valuable time, I believe, because we as neuro-developmental optometrists have
not been assertive about what we know and do every day. It is time to stop being wimpy and get to the
research labs with the old models that worked so well. Not perfectly, mind you, but well.

We can prevent about 2/3rds of refractive error, it seems, from the literature and clinical
experience.29 Our alternative is to do nothing and watch them get worse. This should be found
unethical and morally intolerable by the caring clinician. We do know what to do – it’s almost too late,
but we need to begin to do it.

**TONIC STUDIES**

Studies of dark focus and dark vengeance (tonic focus and tonic vergence) show that both are
intermediate values – anywhere from about 1.0D. to 2.5 D in front of the individual, with myopes having
the higher values.30,31 While studies of dark vergence show loose association with dark focus, one study
revealed a good correlation of the two under the condition of mental processing.32 That should make
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the ears of those of you who do perceptual therapy perk up, because it supports the old wisdom that when we look at the functioning of the visual system, we are looking at how the brain is functioning in very real, direct and indirect ways.

Since seeing takes place in the CNS, we must probe the CNS to determine as best we can what interference is occurring and what direction it wants to move. We can then devise a treatment plan to intervene or eliminate, if not just reduce, the movement of the organism to cope with the interference (or, stress).

TWO WORLDS

Philosophically speaking, we may say that humans exist in two space worlds: distance and near. If one has trouble operating in – accommodating (both optically and cognitively) – the nearspace world, you must either adapt – become myopic – or guard against encroachment into that space. To study an object, one must be able to bring it in and attend to it in nearspace, not farspace.

Thus, hyperopes would derive much motivation from the farpoint world, guarding the estate, and myopes derive much of their motivation from analysis and processing of nearspace.

One writer believes that it appears as if the visual system always tends to myopia, if so, we are fighting how the environment impacts us by emmetropizing. (I called it “hyperopization” in my paper, Stress and Eye. Because the animal research appears to have validated the presence of a regulatory system, Van Alphen’s comment in his paper on Emmetropia and Ametropia becomes valid. That is:

[O/H #6 Van Alphen]

“Only when the brain is invested with a regulatory capacity that a unified concept for the origin of ametropia becomes feasible”. 34(p. 84)

Because animal research has demonstrated that there is an emmetropizing mechanism with regulatory capacity, we can look for a unified concept of ametropia. Perhaps then as clinicians, we can be “real doctors” working to prevent and, to some extent, cure, ametropia.

[O/H #7 - PERSONALITY: MYOPIA AND HYPEROPIA]

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PERSONALITY

As Aristotle said, “Nothing is in the mind that did not pass through the senses.” Ward Halstead, a Chicago psychiatrist, called vision “One of the major contacts with reality in the early experience of the child. The outcome of the future development of the personality is to a great degree a function of the extent and quality of that visual contact.” Seeing takes place in the CNS. (Importantly, we also seem to be observing that what passes through the visual sense also affects the sensory structure.)

Studies of the relationships of personality style and ametropia have suffered from some basic problems and disagreement. The different researchers have all used many different personality probes that are not necessarily equal. Additionally, the tests may not have been looking at the attributes that clinicians associate with the various ametropias.

What are the attributes associated with myopia? Myopes seem to have a profile that makes them: worry, depress, fear and they are lent to withdrawn-ness, a sedentary lifestyle, a detailed analytical nature, and a higher degree of desire for inter-personal relations. Angi and Rupelo described their myopic population as “sufferers”.

What are the attributes associated with hyperopia? The candidates for adverse hyperopia are more likely to be: assertive, alarmable and hot-reacting; action-oriented; global analytical approach. These and a higher degree of goal, problem and task-orientedness would seem to predispose one for progressive hyperopia.

There are individuals who mix these: they will do the opposite of one or the other, but statistics should prove an association of the above factors, generally speaking. The problem is to get personality inventories that are accurate in measuring the qualities being looked for.

Researchers before Van Alphen’s seminal paper – and since – have asserted a relationship between anxiety and myopia. Just how to sort out what kind of, and how much, anxiety is required to trip the pre-myope into the active distortion is the problem before researchers now.

ETIOLOGIES REVISITED

So what causes ametropia?

Well, myopia is just what we were told in grade school – the eye is too long. Almost all of that extra length is in the posterior chamber.

And astigmas are toroidal surfaces of the cornea and posterior pole of the eye.

Hyperopia is still a puzzle, in the main. It is not an optically weak crystalline lens because Gawron, some Russians, and others have noted that myopes actually have the weakest lenses. Axial length doesn’t correlate well with hyperopia, but I have not seen any statistics for any relationship between the amount of hyperopia and axial length. I suspect that there probably is. The index of the lens is the most underappreciated component in the possible production of farsightedness – even sudden farsightedness. The culprits are probably hypercalcemia and sugar alcohols. Stop and think: at least once or twice a year, we all get patients whose myopia has suddenly gone from -6.00 or -3.00 to a -3.75 or a -1.00 in a matter of a week or two.
You have seen them, too. What is the problem with them? Hyperglycemia. Sudden onset of diabetes, which is documented to result in hyperopia, not myopia. (Slow onset diabetes results in myopia.)

So, while I am not saying that hyperopes are diabetics – I am saying that there are physiological processes in the body which are quite capable of producing hyperopia above 1.00 D, which Rosner has shown is associated with reduced academic skills.

ACCOMMOVERGENCE

In my paper on Stress and Eye I outline a mechanism by which hypercalcemia may produce geometric changes and probably indicial changes in the crystalline lens structure. In that paper, I also describe how against-the-rule astigmatia is probably best described as the horizontal torus formed by the pull of the superior oblique and inferior oblique muscles across the posterior pole. In a paper I presented at the 1997 Kraskin-Skeffington, I began the detailing of a model of accommodation and convergence as unified, rather than merely synkinetic, processes. I called it “Accommovergence”. That work is still progressing, and it is gaining new weight with just about all the literature I have found.

Some understanding of that model is necessary for the rest of this discussion. As this theory is worked out, it’s more and more apparent that the driver and power behind ametropic changes is the ongoing battle among the sympathetic and parasympathetic branches of the autonomic nervous system, and their fight with the voluntary nervous system. The interference patterns that are set up can be studied and interrupted by the clinician – that means that prevention is and always has been possible.

OEP THEORY & TRANSIENT/SUSTAINED ACCOMMODATION

The preliminary indications are that Skeffington and Alexander and all the other functional fathers that we owe allegiance to were right. For as far as they went, they were right – and decades ahead of their peers. The “check, chaining and typing” appear to be solidly based in the neurodevelopmental principals of vision, cognition and personality.

The evidence suggests, theoretically, that the ciliary muscle and lens system functions primarily as an oscillating analyser with a dual-phase mechanism. This phenomenon of oscillation is known, consisting of a fast and slow phase. The fast phase is controlled by pulse rate. The oscillation of the image may help to find the retina and the direction of blur. It performs the function of a transient accommodative system. Peering postures – including the “myopic slouch” – appear then to help the EOM’s to compress the globe, squeezing it like an egg in a fist, nestled inside the constricting muscles, with the recti pulling back and the obliques pulling forward. The globe can be either lengthened or shortened, depending upon which muscles have been primarily stimulated. Lengthening of the eye under accommodation has been reported by at least one set of researchers.

With-the-rule astigmatism appears to be formed when the superior and inferior recti are stimulated, stretching the cornea vertically as the individual peers at far. The cornea has been measured to horizontally flatten with convergence by Lopping and Weale. If the ciliary mechanism is being functionally blocked from higher centers, the EOM’s will be called on for longer, more sustained accommodation. Anatomically and functionally, the EOM’s appear to be functioning as a long-term accommodative system. They are hereby postulated to perform the function of the sustained accommodative system.
Do the EOM’s really control accommodation? We will try to demonstrate this now. This won’t work well enough for everyone, but those of you who are myopes and low hyperopes should see the phenomenon I want to demonstrate. Astigmats will get an extra run for their money. In your program is printed a Reduced Snellen chart and an astigmatic dial. If you will remove your glasses – you hyperopes can attempt this with the distance portion of your Rx., if you are presbyopic, otherwise, leave your glasses on. Hold the chart at arm’s length and bring it in slowly, until you find your punctum remotum, or the best focus you can attain – WITHOUT STRAIN, please. Consciously avoid accommodating, if possible. You hyperopes will probably be at full arms’ extension. This should still work. Keep the chart straight ahead of your eyes, in primary gaze position. Now, tip your head down while still looking at the chart, so that you are peering up under your eyebrows. Don’t move your arms yet. Some of you will notice that the target blurs, some will see it get clearer. Those of you who see it blur, slowly move the chart in and you should find it refocuses – perhaps clearer than ever – at about 1” closer. Those of you with AGAINST-THE-RULE cylinder will note that the smear is reduced or gone. Now, if you repeat it, but drop your eyes, to look down your cheeks, you will find it gets even closer -- about 1-1/12”.

Your EOM’s are helping your eye to accommodate. Blumenthal theorized this and Takeda and his colleagues tried to demonstrate this experimentally but found it only worked for them at far. They suspected that the equipment – a modified Badal stimulator – limited their results at near. Well, you don’t need an instrument. A chart – or want ad section – will suffice. Those of you with cylinder of over 1.00 D will note on the astigmatic dial that the axis rotates about 15 degrees.

So what is the point? The point is that striated muscle – the EOM’s are probably an important part of the accommodative mechanism. They have collateral and simultaneous function with the smooth muscle of the ciliary focusing system. Vergence during all of this is kept in fine tune by the Trochlear and Abducent nerves so that diplopia will not occur.

DUCTIONS

We probe these complex relationships with duction and blur findings. We study what the patient wants (that’s the blurs) and his range of freedom (that’s the breaks and recoveries) as he or she inhibits the pattern of blur. As Skeff told us 50 years ago, we are not looking at muscle strengths, we are looking at and should be studying the relationship existing between (at least) two patterns – neural patterns – learned neural patterns.67

At the same time, Skeff told us that any rational program of lens application must be based upon a study of ductions. This dual effector model of accommodation, with transient and sustained components that intimately integrate with the vergence system, furthers our understanding of why ametropia develops by helping us to put structural legs under a theoretical platform.

Stop and think: we can’t – shouldn’t – go fitting glasses and contacts onto living, thinking persons like their eyeballs were mounted on an optical bench, yet that is what is done in 90% of the offices in the world.

If we do not intervene in the individual’s visual problem, then the ametropia and suppression patterns and deviations that can occur, which we consider clinically “wrong”, are actually the visual system’s best solution to its problems.

The stressors that provoke the psycho-physiological changes are complex. Much of what stresses humans varies considerably from one person to another, based on three unique factors.
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[O/H #8 -- 3 variables]

1) Heredity: we can’t choose our parents. (Nature)
2) Experiences: unique to each individual. (Nurture)
3) Apperception: how we respond emotionally to an experience (Beliefs)

One, our heredity, because we can’t choose our parents (Nature). Two, our experiences, unique to each individual – that is the nurture; and Three, our Apperception – that’s how we respond emotionally to an experience (or stimuli) – and that is based in our beliefs, built on prior experiences.

SCHOOL WORK

Let’s consider what a child is subjected to in a classroom:

School rooms present a contained environment to a child comparable to a caged animal’s. He or she is constrained to a seat and nearwork environment for four to six hours a day – and the mental processing adds further stress.

Containment of an animal is one of eight factors that raises ACTH levels, which then increases cortisol levels (hydrocortisone) and cortisol does not-so-nice things to collagen, the main protein of the eye. Only one report that I found has attempted to study cortisol in myopia, and the model was fatally flawed. Angi and Rupola, et al., had sampled Cortisol once a day, yet since its production varies widely throughout the day, it needs to be studied on a diurnal basis – 24 hour urine samples need to be brought to each of our offices for study – can’t you just see it?

Retinal defocus while reading probably occurs due to centralizing of attention. Josh Wallman has proposed that the large areas of peripheral image degradation is a form of form deprivation. Research seems to point to a retinal growth factor, probably involving dopamine and a collagen synthesis factor. One researcher (Angrist), in particular, noted that scleral tissue is normally a wondrously complex structure and questioned why it should give way and stretch as it does in myopia – this is an enigma, he said. Part of the answer may be that there are time-dependent ocular rigidity actions in the sclera that allows a visco-elastic property to do a slow stretch after an initial resistance.

Time alone may be a significant factor when the myopic candidate’s eye has been subjected to EOM tension, increased intraocular pressure, and the retinal factors released by form deprivation like Wallman talks about in myopes, plus the possibility of some unknown chemical release over time with ciliary muscle tension on the retina, as Van Alphen demonstrated. Van Alphen was just one of the researchers who suspected that since the stretching of the sclera occurs at relatively low IOP’s, it must be affected by some other mechanism.

The local and general adaptation syndrome, as put forth by Hans Selye, would seem to offer a productive area for research. You will have to read Stress and Eye for a better discussion than this right now.

Weisel & Raviola concluded that myopia was caused neurally by alteration of the visual perception, not by the local effect on the eye. Van Alphen believed that the competitive environment of the classroom was an element that should not be underestimated, because learning has a much more complex psycho-visual mechanism than mere closework does. If we can take any lead from these researchers’ conclusions, it appears that the brain must be “in gear” for myopia to gain a foothold.
In 1987, Bullimore & Gilmartin found that tasks with high cognitive demand cause the dark focus point to pull in, independent of the optical stimulus of focus. So we could conclude that the classroom is an ergonomic and psychogenic breeding ground for ametropias.

**THE EXTRAOCULAR MUSCLES [EOM’s]**

The EOM’s have been noted in the literature since 1794 as suspects in changing the size of the eye. Prentice in 1895 appears to have been on a solid track when he noted that the muscles acted upon a disturbance in the sclerotic coat. He felt the disturbance was nutritional. Bates, back in 1912, felt that the EOM’s were responsible for accommodation — unfortunately, he discounted the ciliary mechanism. He observed myopic astigmatism produced by children straining to see. Scientists have tended to discount all that he did because of some significant errors in his premise.

However, we have difficulties explaining some phenomenon if we discount the EOM’s. Against-the-rule astigmatism has had any number of proposed etiologies and the EOM’s fit in well with some, make others unnecessary. With-the-rule astigmatism is associated with accommodative deficiency, but no mechanism has been widely promoted. The role of the EOM’s in accommodation would answer this nicely – the horizontal torus generated by the recti seems to fit the puzzle pretty well, for now.

And there’s another major problem that the role of the EOM’s in accommodation and hyperopization might readily explain, in part or in whole: the trouble with sympathetic inhibition. Oh, you didn’t know it was in trouble? Well, it appears that it is. Here is why:

1) If dark focus (DF) and dark vergence (DV) are both intermediate values — and they are; and,

2) We also know how DF and DV pull in – it’s the EOM’s and ciliary mechanisms.

We have a problem with:

3) How do DF and DV get restored (or reach out) to optical infinity?

You see, we have been told all along that sympathetic stimulation focused the eye to 20 feet. But *HOW?* What’s the mechanism?

I asked several colleagues if they were taught anything other than the radial fibers of the ciliary muscles. None were. Anatomists tell us, though, that the radial fibers are few in number, weak, and may not even exist, that they are just transitional fibers from the sphincter to the longitudinal fibers.

However, if the EOM’s were involved, then sympathetic stimulation would excite contraction, and the correct combination of EOM’s would result in a fore-and-aft compression of the globe, resulting in a “hyperopization” of the entire optical system via axial foreshortening.

We know that it is sympathetic stimulation that puts focus out to optical infinity, but I have not found anyone who has suggested a practical way from known anatomical features for that to happen. This model of the EOM’s as primary activators in the accommodative and emmetropization processes starts to open new concepts for examination, literally and figuratively.
Refractive Error as a Lesion: Toward a Unified Theory

The somewhat mysterious intricacies of the empirical OEP formulas are perhaps scientifically unraveled a bit if we can accept the theory that accommodation is biphasic – transient and sustained components requiring voluntary and involuntary kinesis in not a synkinetic manner, but unikinetic. Secondly, distance acuity is maintained by the action of the EOM’s in global compression.

Prisms in our prescription formulas have been meted out judiciously in the past. However, if low power (mini-) prisms help to center the patient’s visual range or help him to learn a new neural pattern of operation, we may be gifting that patient with instant changes, like I will play for you in a reading demonstration.

CONCLUSION:

A little girl had just finished reciting her times tables to her grandfather. He then asked her what 2x13 was. “Oh, Grandpa,” she said. “Everyone knows there’s no such thing as a 13’s tables.” – The moral is that just because you don’t know the answer to a question doesn’t mean that there isn’t one – that includes ametropia.

To conclude, refractive error is a psychophysiological adaptive disease that originates in the neural substrates of the brain, thoroughly mixed in with nature and nurture, nutrition and personality. Learning and anxiety – including performance anxiety, especially – seem to be direct triggers for expression of ametropias, primarily in a nearpoint environment.

We probe these relationships through our case history, and, with blur and duction findings. When we compare distance and near values, the “low” break and recovery shows the direction of interference, Skeff always said.44

We can expect the individual to operate freely within his ranges of freedom. Glenn Fry said that it is only when the direction of interference gets to the point where accommodation changes will there be a perceptual blurring.60 When the blur occurs, then accommodation must be changed. I believe that my very liberal use of base in prism – just like the German ophthalmologists of 150 years ago61 – is operating successfully in changing performance and preventing refractive changes just because of this.

Remember how Skeff always said,

[O/H – Skeff quote]"The value of a lens is neural, not optical."44

To demonstrate this, let me play two short recordings for you. They are short, but loaded. These recordings are both 10-year old girls. The first is in academic trouble of long-standing, the second recently dropped in math from her normal A’s and B’s and complained of distance blur – not near blur – she is a 1.00 D hyperope. The narration should be self-evident as to what is going on.

[TAPES]

The differences you have heard were neural, not optical. The first girl’s mother asked a good question – she said, “Yes, but is she comprehending?” I replayed her the tape and I pointed out where she enumerates the items of the cat in the mirror. The brain changes, with the lenses and prisms. Comprehension changes.
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I had a narcoleptic, who fell asleep every time he read – he went out three times on me during the duction tests. He had attended two years of college: “How,” I said, suspecting that he was malingering, “can you read for college without falling asleep?” He explained he only fell asleep if he paid attention, so he was able to read all his books into a tape recorder, and then listen to the tape. Reading, it would seem from this illustration, probably occurs at two different levels, neurally.

[O/H - qEEG’s]

Here is a rare case from my files. This 30-year old college student had been referred by a psychologist. His electrical activity in his brain has apparently changed after just eight weeks of visual therapy. In the first set of maps, we have a brain that is low power and not unusual specifically but generalized in dysfunction consistent with the man’s history of mild cerebral palsy, was the interpretation of the neurologist who read these.

[O/H qEEG #2]

Here, twelve weeks after the 1st qEEG, his beta waves – associated with cognition and alertness – have increased by more than 2 S.D.’s. The overall power had increased as well. The neurologist was at a loss and said it gave the appearance of someone on benzodiazepines. (He wasn’t.) His whole visual and cognitive performances had changed due to proper lenses, prisms, and eight weeks of visual and perceptual therapy.

Is there any great doubt that the brain is changed by the environment impinging upon it? Optometrists change the brain when we impose lenses and prisms and thus change the visual environment. [But we must be judicious – many of the failures in bifocal control of myopia experiments either used reading adds substantially higher than most clinicians would use, or did not specify the height of the segment (it needs to be mid-pupil or a bit below).] Prism powers in the <4P.D. range have the greatest effect.

Can we control the ametropias? YES.

Are we helpless victims of our genetics? Not necessarily.

As a final point of illustration, let me show an example of myopia control in one family – my family. This is their refractive family tree. One advantage of this illustration, as small a sample as it is, is that the refractions are known for the entire group.

[O/H #11 – FAMILY TREE (See attachment)]

Note that the grandparents’ average refractive error is a +2.00 D. The parents’ of the cousins and my wife and myself is -4.75 D. the refractive error among the eight cousins is -4.12 D., pretty consistent with the parents, aunts and uncles. The refractive error among our own children is significantly different – and that is probably statistically significant, as well. The average is -0.40 D. What made the difference? It’s that I have intervened with lenses, prisms, nutrition, and visual therapy variably on all five of our children. Our daughter who is -4.00 D. had a debilitating illness, but was significantly myopic even before her sickness.
Refractive Error as a Lesion: Toward a Unified Theory

[HANDOUT – The Myopia About Nearsightedness]

Can we control refractive error? Yes. Is it perfect? No.

HANDOUT 1

This handout is what I use to advise parents of children at risk. The questionnaire that is published in your book is probably quite valid, but truthfully, I have found no way to use it clinically. It lends itself more to being a research screening device. The Mandelbaum test performs irregularly for me. I have not given up on it, but I have not discovered a mesh that works for everyone. It would be neat to find a way to make it work.

So, optometry doesn’t know what two times 13 is. That does not mean there isn’t an answer.

We can control the majority of ametropia. Not all of it.

We owe it to our patients to use techniques to analyse their potential for changes toward these diseases and to care for them in the most proper way. We need to be diagnostic doctors – REAL doctors – not merchandising doctors. Diseases beg for healers – will we be the healers we are called to be? Thank you.

[O/H #12 – Questions for research]

<table>
<thead>
<tr>
<th>Animal studies:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Would myopia develop in de-adrenalized animals?</td>
</tr>
<tr>
<td>2) What are the Cortisol levels in the animal studies?</td>
</tr>
<tr>
<td>3) Will lens-induced hyperopia be induced in animals whose EOM’s are detached?</td>
</tr>
<tr>
<td>4) Scleral strip tests: subject them to drugs and normal stress products to determine their properties, plus effects on uveo-scleral outflow.</td>
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</tbody>
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<thead>
<tr>
<th>Human studies:</th>
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<tbody>
<tr>
<td>1) Dark focus to far point focus: what is the nature of the mechanism? Is it axial change? Corneal? Crystalline lens?</td>
</tr>
<tr>
<td>2) Prospective study of IQ/reading/myopia.</td>
</tr>
<tr>
<td>3) Vocabulary and myopia.</td>
</tr>
<tr>
<td>4) Exophoria and myopia.</td>
</tr>
<tr>
<td>5) Determinations of the crystalline lens thickness under sustained accommodation.</td>
</tr>
</tbody>
</table>
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16. Lane, B; Food Folate vs. Supplemental in Myopia Development and Tear Film Integrity, Optom Vis Res, 71(12)=Dec Suppl, 1994.


43. Bowan MD; Accommoovergence: A New (?) Concept for Preventing Visual Problems; Transcript of the 40th Annual Kraskin-Skeffington Invitational Symposium, Ophthalmic Extension Prog Found, Jan 1996.


55. Hosack D; Observations on Vision, Philosoph Trans Royal Soc Lond, 84(196ff.) 1794.


The Bacon/Bowan Refractive Family Tree

LEGEND: Boxes = Males; Circles = Females
Precise values less than or equal to the amounts noted.
White fields = Hyperopia, Gray fields = Myopia.
(Spherical equivalents used.)

Myopia control instituted at ages from 3½ to 5.
Used vision therapy and plus lenses -- in single vision Rx or segs.
I. The Cure for Myopia. III. What Can be Done?
II. What is Myopia? IV. The Future.

I. THE CURE FOR MYOPIA (Nearsightedness).

Q. Can myopia be reversed?
A. Unfortunately, no.

Q. Can myopia be prevented?
A. Fortunately, yes, most of the time. That's the cure for myopia: PREVENTION.

Q. But what about surgery? I see many ads.
A. Radial corneal surgery and lasers only address symptoms of the problem. Studies have never shown close associations of myopia with the corneal shape. Just as you learned in school, in myopia the eye is optically too long. The great majority of refractive surgery patients will need glasses or contact lenses over the weeks, months or years following the surgery.

Q. But what are the best things that can be done for myopia?
A. Work to prevent progression of nearsightedness, once it's started. The only real cure for myopia is to prevent it. You have to understand what myopia is.

II. WHAT IS MYOPIA?

Myopia is one of three adaptive optical errors that occur as responses of the visual system to sustained, repeated stress -- they are stress diseases. The others are astigmatism and progressive hyperopia -- farsightedness. In nearsightedness, the eye is optically too long. Astigmatism distorts the image on the back of the eye and farsightedness causes the individual to pay too much attention to focusing on near work, causing eyestrain and reduced performance.

The idea that myopia is genetic is not borne out by the available data. Family histories, at best, have only a modest predictive value for eye problems. Genetics may predispose certain families toward specific eye problems, but the trigger to the distortion of the optics of the eye is stress and reading, in the great majority of cases. Since the human eye is almost full size by age four and certainly adult size by age eight, the concept of an eye that "grows" too long is not a valid concept. Most myopia begins after age eight. Most beginning myopes get their first glasses at ages 9 to 10, but there is a new group of beginning myopes who have begun to emerge over the past 20 years: adults who use computers.

A British ophthalmologist has described myopia as "juvenile expansile glaucoma", caused by reading. Technically, he's probably correct, although it's a benign condition, and not the adult, damaging form of glaucoma. Myopia has affected mankind since the beginning of time whenever the visual system is stressed and the individual can't or won't escape the stressing environment. The combination of
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events that causes the nearsightedness include extended nearwork, depletion of Vitamin C, stress-induced calcium losses, muscular weakening of the rear chamber of the eye, and increased pressure in the eye relative to the softened sclera, the white superstructure of the eye. The combination results in a gradual ballooning of the eye -- incredibly, adding only 1/2 mm. of extra length to the eye reduces vision to 20/200 or poorer! The increased length of eye is all in the posterior chamber.

III. WHAT CAN BE DONE?

The main factors in preventing nearsightedness are proper nutrition, preventive lenses to protect normal farsightedness, proper visual hygiene, and stress reduction techniques. To elaborate:

1) Nutrition - supplementing the diet with several hundred milligrams of vitamin C each day is reasonable. (Experts have estimated that cavemen received 1100mg. or more per day in their diets.) Adequate dietary levels of calcium, magnesium, and zinc are in order, as well. Children given calcium in one British study showed stabilization and reversal of their myopia. A diet adequate in fresh fruits and vegetables should be preventative, but not many children have the taste or opportunity to do so. B-complex vitamins help to reduce the effects of stress of any sort.

2) Proper visual hygiene:

   A) Posture: sitting upright for reading or writing is very important. The visual system works best in a slightly downcast position.

   B) Lighting: lighting engineers recommend a 200 W. glare-free study lamp. The benefit is to increase figure-ground contrast and also to constrict the pupil, which increases the depth of field and thus reduces the need for critical focusing, which is the primary distress on the visual system.

   C) Time: the eyes appear to be designed for up to 20 or 30 minutes of sustained work without sustaining ill adaptive effects. Therefore, eye breaks every half hour - looking away and stretching hard or walking away for a moment, plus a walk-away for 10 minutes every 90 minutes when reading, writing, or computing, will minimize the stress effects on most people's eyes.

3) Preventive lenses - "Plus 50" or "Plus 75" reading lenses are powerful tools against myopia for most students, beginning as early as second grade for most, though some students are showing signs of this developmental nearsightedness by the middle of first grade. Personality style analysis can help to detect earlier need for these "brain glasses", as they're sometimes called: children prone to myopia seem to be the sort who are intelligent, analytical, withdrawn, stress-absorptive, underactive children, who are driven to please their superiors. Bifocals with clear top portions are sometimes a more practical way to provide the reading lenses without the bother of taking them off to see distance objects.

Rigid, gas permeable contact lenses have been shown to slow or stop progression, but just why isn't exactly clear. Soft contact lenses are well-known to permit progression: it's called "myopia creep".

Base-in prisms ground into the lenses have had preventive value since the mid-1800's when ophthalmologists in Germany used them on students becoming progressively more myopic. They are still sometimes used for this purpose. [There are no guarantees that preventive lenses will work for any particular individual, but research shows that they do work 60-70% of the time. Our only other option is to do nothing and watch the 60-70% of at-risk people get worse and worse vision.]

4) Stress reduction techniques - action is a useful antidote to stress. The body under stress gives all the appearance of being readied for fight or flight, even when either of those responses would not be appropriate. So a walking program, or low-impact aerobics, a sports hobby or other energy-burning activity will tend to help.

Relaxation, meditation, and/or prayer are scientifically proven ways of reducing stress. Progressive relaxation techniques were used for fighter pilots in WWII and Korea to not only reduce
stress, but also improved flying combat skills. They reduce stress effects and can improve thinking skills in students, as well - it would be interesting to study formally. Teaching children how to take a "5-minute vacation" as Dr. Herbert Benson talks about in his book "Beyond the Relaxation Response", would be beneficial at many levels.

"Children need more baseball, less T.V.", said Dr. Francis Young, a primate researcher from Oregon. He was responding to questions about his conclusions of what made animals nearsighted in his experiments. Children need more outdoor activity and preferably interacting with other children.

Performance anxiety needs to be dealt with constructively, as well as control issues, so that a pattern of prolonged distress response(s) doesn't become a habit.

IV. THE FUTURE

Will there ever be a remedy for nearsightedness? It's hard to see how that can be, especially since our society is moving toward MORE nearwork with the advent of the microcomputer and the Internet Age. Hopefully, more and more vision care providers will finally acknowledge that functional nearsightedness (the nearwork theory) is responsible for most garden-variety nearsightedness - up to 75% it would appear. Then we can get on with the real cure for myopia, PREVENTION.

For Additional Reading:
Bowen, MD: The Control of Myopia; Journal of Optometric Vision Development; Volume 12, Number 2, June, 1981.

Bowen, MD: Stress and Eye: New Speculations on Refractive Error; Journal of Behavioral Optometry; Volume 7, Number 5, 1996.

Preventing Refractive Error: What's a Doctor to DO?

Merrill D. Bowan, O.D.

For a more in-depth understanding of the concepts being dealt with in this workshop, please refer to the following publications by this presenter:


Refractive Error and Personality Style; Transcript of the 30th Skeffington Invitational Symposium, Jan 19-21, 1991, Optometric Extension Program, Santa Ana, CA.

Stress and Eye: New Speculations on Refractive Error; J. Behavioral Optom. 7(5)115-22, 1996.

Differential Diagnostic Questions to be Answered to Determine the Roles of Vision and Perception in the Efficacy of Learning; Transcript of the 40th Kraskin-Skeffington Invitational Symposium, OEP Foundation, Santa Ana, CA.

Accommovergence: A New (?) Unifying Theory of Accommodation and Convergence; Transcript of the 42nd Kraskin-Skeffington Symposium on Vision, in press, OEP Foundation, Santa Ana, CA.

Bibliography:

Handout 3

Toe Curls - A Method to Relax Focus Spasm

Merrill D. Bowan, O.D.

Visual fatigue and focusing spasm quite frequently go hand in hand with sustained close work. School study, video display terminals (used for both business and pleasure), and tasks that require extended eye-hand work on jobs and hobbies with much detail are some examples.

Surely you have experienced looking up from the TV Guide®, only to find the T.V. screen is blurring. You have to blink and blink to clear it up. Why? -- the focusing gizmos in your eyes are stuck, and have to be unstuck -- blinking often does just that.

For years, it has been possible to do that quickly and easily by doing a "toe curl". With children at risk for progressive nearsightedness, the method has been clinically seen to reduce or eliminate the mad rush into more and more blur. (Proofs for this last statement will be a long time in coming, but the method is quick and easy, and you can demonstrate its harmlessness and effectiveness very easily.)

Students and workers need to be encouraged to do this hygiene technique every 20 or 30 minutes, as well as at the end of any intense or prolonged close work with the eyes. It is simply a dynamic isometric tensioning (remember Charles Atlas?) of all the muscles for a few seconds.

We call it a "Toe Curl"

How -- After 20 or 30 minutes of close work, look away from your work to something that has printing on it, like a clock, a poster, a sign outside the nearby window -- whatever target you have that has notable detail on it.

While staring at the details, numbers or lettering, tighten your toes downward inside your shoes, then progress up your legs, through your torso, fists, arms and neck, tensing all your muscles intently for about five seconds and then quickly release them all at one time.

If the target clears, that means there was a spasm of focus that just relaxed. If it does not clear, then there was not! (Or, you may have done a wimpy, wimpy job -- you may want to do the exercise one more time to make sure.)

That's it!

[The technical explanation for why this works is a basic fact of the brain that when you stimulate the voluntary nervous system as you just did, the involuntary system is forcibly relaxed. It is called "reciprocal inhibition" -- so much for big words.]

Will it work? Yes. Will it prevent all nearsightedness from progressing? No. Is there anything better? Nothing cheaper. Is it relaxing and effective? Yes, yes, a thousand times yes. (Start counting: "once yes, twice yes, thrice yes...") Go do it!

Ibco2.doc

Handout 4

Draft #3
REFRACTIVE PERSONALITY SURVEY

This instrument is helpful in determining if a child may be at significantly greater risk for developing any of the refractive errors: nearsightedness, farsightedness, and astigmatism. Each question is answered separately, but questions #2 & #3 are asked together before answering them, so that the child can understand the contrast that will occur.

1. I need to be liked by people a whole lot.
   A. Oh, YEAH!
   B.               C. Doesn't matter
   D.               E. No way!

2. Toys and things are better than anything.
   A B C D E

3. Friends and family are better than anything.
   A B C D E

4. I would rather have a new friend than a new toy.
   A B C D E

5. I love to be with friends.
   A B C D E

6. I like being the leader when I’m in a group of friends.
   A B C D E

7. I don’t like being the leader when I’m in a group of friends.
   A B C D E

8. I find something else to do if I can’t be the leader.
   A B C D E

9. I often have trouble listening to what grownups want from me.
   A B C D E

10. When I see someone hurting, I try to make them feel better.
    A B C D E

11. Often, I will do almost anything to get ahead of people.
    A B C D E

12. I like to think my way out of problems or trouble.
    A B C D E

13. I would rather run away from most problems or trouble.
    A B C D E

14. I try to stop problems before they begin.
    A B C D E

15. I will let other people win so that they will stay and play with me.
    A B C D E

16. I will do almost anything to win.
    A B C D E

17. I try to get people to do things my way.
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A B C D E 18. I like to read almost more than playing outside.
E D C B A 19. Not getting my way makes me more angry than sad.
E D C B A 20. People's problems are their own. I don't try to help.

<TOTAL NUMBER OF ANSWERS IN EACH COLUMN

(Multiply the total number count of each column by the values below.)

1st___ x 1 =___; 2nd___ x 2 =___; 3rd___ x 3 =___; 4th___ x 4=___; 5th____

x 5 =___

_____  FINAL QUESTIONNAIRE TOTAL

Cover test at near:
1 3 5 EXO ORTHO ESO

Mandelbaum test (Dark focus):
1 2 3 4 5 >/= 2.0D. >/= 1.5D. >/= 1.0D. >/= .75D. >/= .5D.

Final questionnaire total + Cover Test + Mandelbaum Test = _______
PREDICTIVE INDEX

(The last two tests increase the predictive value of the questionnaire, but may be omitted and the questions alone indicate refractive error potential.)

Myopic potentiality index is represented by the lowest 30% of scores; Progressive hyperemic potentiality is represented by the top 10% of scores. [Data is currently being collected.] Preventive care by a developmental, behavioral, or neuro-developmental optometrist is strongly recommended.

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