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Behavioral optometric science student manual

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Behavioral optometric science student manual

Abstract
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Other Authors of Manual: Angela Darveaux, Katherine Chhor, Kim Schweiger

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BEHAVIORAL OPTOMETRIC SCIENCE
STUDENT MANUAL
OPT 562

By
KRISTEL ROGERS

A thesis submitted to the faculty of the
College of Optometry
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Kristel Rogers obtained a bachelor of arts degree in Biology from Concordia College in Moorhead, MN. She will graduate from Pacific University College of Optometry in May, 2006. She plans to practice in the Midwest specializing in cornea and contact lenses in a primary care setting.
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Pacific University College of Optometry

Kristel Rogers, Bradley Coffey, O.D., FAAO

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Refractive Conditions

The relationship between the overall optical power of the eye and the size/shape of the eyeball determines the refractive condition of a person's eye. It is important to recognize that refractive condition is measured based on the eye focused at optical infinity, so the eye is not accommodating. For an explanation of accommodation, see prior notes in the on-line manual.

Etiology of Refractive Conditions  
(discussed further under section on Myopia)  
1) Genetics  
2) Environment (use/abuse of eyes, trauma)  
3) Pathology/Disease

Types of Refractive Conditions

A person is said to be an *emmetrope* when light from a distant object enters the eye parallel (from infinity) and the optics of the eye cause the light to focus precisely on the retina. The eye can see clearly at all distances without compensatory lenses if the eye has full accommodative ability.

The optical power and length of the eye match in this case to form a clear image on the retina.
A **hyperopic** eye occurs when light from a distant object is focused at a point behind the retina instead of directly on the retina. A blur circle lands on the retina so the person does not see a clear image. This occurs because the relaxed/non-accommodating eye is not strong enough or the eye is too short to focus the light on the retina. The patient may be able to add power to the eye by accommodating, and therefore see an object clearly. Distant objects are easier to make clear on the retina because they require less accommodation; for this reason hyperopia is sometimes called FARsightedness. Closer objects require more accommodative ability and if the patient is presbyopic, they may not have enough accommodation left to form a clear image so they see blur at near.

![Diagram of hyperopic eye](image)

The optical power and length of the hyperopic eye do not match so a blurry image is perceived; point focus is BEHIND the retina.

In order to determine the exact amount of hyperopia in a patient, we often cycloplege the patient. This means that we put a drug in the eye that temporarily shuts off innervation to the ciliary muscle that controls accommodation. The eye can no longer accommodate at any distance with this drug, so a hyperopic eye cannot see clearly at any distance. This is especially helpful for young children that have a high amount of accommodative ability which hides their hyperopia when doing regular refraction without the drug. Cycloplegic drugs may be said to reveal hyperopia and reduce artificial myopia. Hyperopia is revealed because
the eye cannot accommodate through the hyperopia anymore, so the true refractive condition of the eye is revealed or “induced”. Myopia is said to be reduced because the eye is fully relaxed with no accommodation. If the eye is not accommodating, this means less power in the eye so the eye is less myopic.

A myopic eye occurs when light from a distant object is focused at a point in front of the retina instead of directly on the retina. This again results in a blur circle on the retina because the light begins to diverge again after it comes to a point in front of the retina. This condition occurs when the relaxed/non-accommodating eye is too strong or too long. Objects at a distance are blurry for this patient because the eye has too much power, therefore converging the light rays from a distant object more than what is needed to focus the image on the retina. Closer objects, however, may be seen clearly without as much accommodation as an emmetropic eye. The myopic eye is therefore said to be sighted for near, hence NEARsighted.

The optical power and length of the eye do not match so a blurry image is perceived.
The following is a graph that shows how the relationship between the corneal power and length of the eye determine refractive condition. The area considered to be emmetropia overlaps the solid line somewhat, because emmetropia is considered to be 0.25D or less of hyperopia, myopia, or astigmatism.

Presbyopia is a refractive condition that all people acquire as they age. It is the loss of accommodative ability due to the loss of elasticity of the crystalline lens. The lens does not have the ability to form the convex shape needed to focus diverging light rays from near objects so a blur circle is formed on the retina. Progression into presbyopia begins at around age 13, but does not become noticeable for most visually healthy people until after age 40.

How we compensate for Refractive Condition

We prescribe to compensate refractive conditions based on what an emmetropic eye should be able to see at infinity. Optical power is measured in dioptric units, and therefore lens power is also measured in diopters to overcome
optical power that is too strong or too weak. Lenses that compensate the refractive condition are opposite the power of the actual refractive condition.

For a hyperopic eye, we use plus power to compensate the eye that is too weak or too short. The plus power will give the added power the eye needs to bring the distant light's focused point to the retina. If the eye was too short, the additional plus power will also focus the light closer to match the position of the retina.

For a myopic eye, we use minus lenses to decrease power from an eye that is too strong or to move the focused image further back to the retina if the eye is too long. A correctly-prescribed minus lens will diverge incoming parallel light just enough to create a crisp retinal image when the relaxed eye is looking far away. The most distant point an object can be seen clearly is termed the eye's far point. For a myope, the far point (in meters) is equal to the inverse of the refractive condition in diopters. So a 5.00D myope not wearing lenses can see clearly only out to 20 cm (0.2 m) away from his/her eye. We would prescribe the patient -5.00D lenses to move their far point to infinity.

Since hyperopic eyes need additional plus power to overcome their refractive condition, they may be able to compensate for the hyperopia by accommodating when looking in the distance. They can do this up to a point as they look at objects closer and closer to them. Eventually they will run out of accommodative ability and the object at near will be blurry. Uncompensated eyes with low-to-moderate myopia do not need to add as much accommodation at the same distances because their eyes already have too much power.
Therefore, the accommodative demand for the uncompensated hyperope is greater than for an uncompensated myope.

Since the myopic eye does not have to accommodate as much, it also does not converge as much due to the near tried between accommodation, vergence, and pupil constriction. This may cause convergence insufficiency in a myope not wearing appropriate prescription lenses. When the appropriate Rx is worn, the convergence insufficiency may disappear. This convergence insufficiency is not true CI, but is secondary to reduced accommodative demand.

A condition that may result from uncorrected hyperopia with a high AC/A ratio is **accommodative esotropia**. This means when the person looks at a near target, their eyes “cross” in a sense, because they overconverge. The person has such a high accommodative demand at near and if they converge a large amount with every unit they accommodate, they will overconverge. Accommodative esotropia frequently appears in children around age 4 years. To treat accommodative esotropia, we can either compensate the patient’s hyperopia by giving them lenses so they are not accommodating at far; or we can give the patient a bifocal for up close so they do not need to accommodate so much at near. However, when the patient removes the bifocal, s/he will still go cross-eyed. Often times, these two types of lenses work together to resolve accommodative esotropia. Distance plus lens will compensate hyperopia and a bifocal will reduce accommodative demand at near so the patient does not converge as much.
Presbyopic patients require plus power near lenses. If they have a
distance Rx as well, they will often wear bifocals. Bifocals are lenses with two
different powers in one lens; one for distance and one for near. Those that do
not have a distance Rx can wear a plus lens for near to give the needed plus
power to focus near objects on the retina. “Invisible bifocals” or progressive
addition lenses (PAL’s) have become a popular option for management of
presbyopia. These lenses do not have a visible bifocal segment, so are
cosmetically preferred by many patients, but the lens surfacing procedure
produces a lens with more peripheral distortion and lesser optical quality than
conventional bifocals.

Other Refractive Conditions

*Astigmatism* is another common refractive condition where the shape of
the eye is not the normal spherical shape, but more like a football than a perfect
sphere. This results in unequal curvature of the axes of the eye. For instance,
the vertical axis of the eye may have a steeper curvature than the horizontal axis
(called “with-the-rule” astigmatism). To make this concept easier, we think of the
eye as having only three axes; a vertical axis (line drawn from top to bottom of
eye through center), horizontal axis (line drawn from inner to outer eye through
center), and oblique (line drawn from front of eye to back of eye through center).
If the vertical component is steeper than the horizontal component, light rays
entering through this meridian will be bent more than if the light ray goes through
the horizontal component. This creates two different foci for light. If both points
come to a focus in front of the retina, the eye has **Compound Myopic Astigmatism.** If both points come to a focus behind the retina, the eye has **Compound Hyperopic Astigmatism.** If one point is focused in front of the retina and the other behind, then the eye has **Mixed Astigmatism.**

Astigmatics often have poor visual acuity at all distances when compensatory lenses are not worn. They recognize blur in one meridian or direction only; for example, if an astigmatic looked at an L s/he may see the vertical component of the L clearly but not the horizontal component.

The cause of astigmatism is usually the cornea, but it can arise from other components of the eye, such as the shape of the lens or retina. With-the-rule astigmatism, where the vertical meridian (90 degree axis) is steeper than the horizontal, is the most common type of astigmatism, but there are also against-the-rule and oblique astigmatism. Against-the-rule is when the horizontal meridian (180 degree axis) is steeper than the vertical meridian (opposite of with-the-rule). When the steepest meridian lies along the 45 and 135 degree axes, it is oblique astigmatism. You will learn about astigmatism much more second semester! When you look at an ophthalmic lens prescription such as -2.00 -1.75 x 090, the first number refers to any myopic (negative) or hyperopic (positive) compensatory power. The second two numbers refer to the amount of astigmatism (-1.75) and the orientation of the astigmatic axis (090°).

When the optical power of a person's eyes differ by more than 0.50D, s/he is said to have **anisometropia.** One rare type of anisometropia is ** antimetropia,** when one eye is myopic and the other is hyperopic. If anisometropia is
uncompensated, the person does not form a clear image of the object simultaneously in both eyes. This may result in suppression of one eye in order to create a single clear image. The eye that requires more accommodation is usually the suppressed eye. It is important to treat anisometropia early because uncompensated anisometropia is a leading cause of amblyopia.

Anisometropia often creates unequal size retinal images, referred to as aniseikonia. Aniseikonia is usually caused by anisometropia, but it may rarely be caused by two different sized eyes. When a person sees two different sized retinal images, they tend to have problems with sensory fusion. It is normal for the two images to be slightly different sizes (up to around 3% difference), but when the difference is greater the brain cannot merge the two images together into one. The patient may complain of asthenopia.

If the aniseikonia is not large, contact lenses will reduce the magnification difference between the two eyes. If the aniseikonia is greater, iseikonic lenses (or size lenses) can be prescribed to compensate the difference in size between the two retinal images without modifying the needed refractive power. The difference in retinal image size created by the lenses is accomplished principally by changing the thickness of the lenses.
Stress and Visual Adaptation

Our body's homeostatic mechanisms are necessary for survival. When we are in a hot environment, we need the mechanism that switches on vasodilation of the blood vessels and sweating to release body heat. Without this, we might have a heat stroke from our body temperature rising too high. Homeostasis also occurs in response to stress. Without the homeostatic response, the stress would grow to the point where it was intolerable and/or uncontrollable. The sympathetic response to stress includes increased heart rate and therefore increased blood pressure and blood flow to the large muscle groups of the limbs and torso, decreased digestion, increased pupil size and inhibition of accommodation. If we do not rid ourselves of the stress, our body responds more and more by increasing its homeostatic response. According to Hans Selye, the sympathetic system controls the response to stress and the parasympathetic system controls the adaptation to stress. Unfortunately, the adaptations made to stress often lead to stress-related disorders or illnesses.

Many people have formulated theories as to how our body adapts to stress. Selye formulated the General Adaptation Syndrome (G.A.S.). He thought the process of stress occurred in three stages; Stage of Alarm, Stage of Resistance, and Stage of Exhaustion. During the Stage of Alarm, the sympathetic system is increased in response to the stressor. During the Stage of Resistance, the parasympathetic system attempts to cause adaptation to reduce the stress and to bring back the balance between the autonomic systems. The last stage, Stage of Exhaustion occurs if no adaptation to the stressor is found.
The system breaks down because it cannot control the stress. An example of this is a student who is so stressed by finals he/she decides not to study and in turn fails the courses. S/he has not found a beneficial way to balance the stress in life. Day to day stressors do not usually reach this last stage.

Homeostatic mechanisms are not only important for our body’s response to stress, but also for our eyes’ response to stress. Visual stress is often from extended nearpoint work. Homeostatic adaptations made by our eyes due to this stress often have long-term negative effects. Myopia is an adaptation our eyes make due to the esoward shift in phoria caused by stress. The amount of esoward shift the eyes make depends on the person, and it may not be constant. Other adaptations to nearpoint stress include convergence insufficiency, divergence excess, and apparent convergence excess. For further explanation on these binocular conditions, see the section on Vergence.

Homer Hendrickson, O.D. theorized the Optometric form of the G.A.S. to relate to visual adaptation to stress. The first stage, Neurofunctional, is when the patient has objective signs of visual stress (found by the doctor), but no subjective symptoms (noticed by the patient). Signs the doctor may find include an esoward shift in phoria during or after near work and an abnormal decrease in the lag of accommodation (accommodates more than necessary). Both of these changes represent an inward shift of the accommodative and vergence postures. These changes usually are not noticed by the patient so the patient seldom visits the optometrist at this point.
The second stage is when the patient has neuromuscular changes to their eyes. The patient will notice symptoms such as distance blur immediately after prolonged near work due to the slowness in relaxation of accommodation (hysteresis). Eye pain (asthenopia) may also occur. Lastly, the patient may notice they are not as efficient at reading. At this point, the visual adaptation is still reversible. If the patient is not treated for these symptoms, eventually the neuromuscular changes will be irreversible and the eyeball may grow to become myopic.

A.M. Skeffington said the cause of visual stress is "the biologically unacceptable, socially compulsive, visually near-centered task which provokes an avoidance reaction that becomes a drive to center nearer in visual space." What does all this mean? Biologically unacceptable means that accommodation is not for prolonged use. Socially compulsive means we require education involving constant near work. The avoidance reaction refers to the sympathetic response which results in the inhibition of accommodation and pupillary dilation. A drive to center nearer in visual space refers to the esoward shift in phoria secondary to increased accommodative effort that is necessary to overcome the accommodative inhibition. Through the AC/A an increase in convergence will occur in conjunction with the increase in accommodation. The result of nearpoint stress may be myopia or binocular problems due to the adaptations made by our eyes to relieve the stress.

Birnbaum has created the most detailed model for visual adaptation. He combined Selye's physiological explanation of stress with Skeffington's ideas of
visual stress. He also attributed visual stress to more than just nearpoint work. He believed stress in general has an impact on the eyes, as well as excessive use of the brain. He said that our eyes tend to accommodate more when we are being mentally challenged. Likewise, he said that we are visually stressed when we look really hard at an object compared to just gazing at the same object.

Birnbaum theorized three steps in the response to nearpoint stress. Step one is **Pre-Stress**. The eyes are converging and accommodating at an appropriate level for the target. The depth of field, which is the range of object distances where the image on the retina is still clear, is normal. One can over-accommodate or under-accommodate slightly and still see a clear image within the depth of field. Depth of field is also affected by pupil size. As the pupil gets larger, depth of field gets smaller. This is not desirable, we want a large depth of field so we do not have to accommodate as precisely. We can be slightly more relaxed in our accommodation with a larger depth of field. As sympathetic activation is increased, our pupil gets larger and we then have a smaller depth of field. If we are not accommodating within the depth of field, we perceive blur and this blur in turn stimulates the parasympathetic system to turn on more accommodation.

When the sympathetic-mediated stress response is activated, the patient moves into step two of Birnbaum’s model. Activation of the sympathetic system results in two critical events: 1) an inhibition of accommodation that causes a slight (and temporary) decrease in accommodative posture, and 2) slight dilation of the pupil causing a decrease in depth of field. This combination causes blur
and requires the eye to increase the amount and the precision of accommodation so that it is within the narrower depth of field in order to see clearly. The blur is usually not noticeable because our eyes respond so quickly.

Birnbaum's third step is the increase in accommodation to overcome the stress-induced blur. The increase in accommodation is mediated by the parasympathetic system. This is the body’s way of bringing the eyes back to equilibrium in order to see clearly again. However, the eyes must accommodate even more than the pre-stress level of accommodation in order to overcome the sympathetic inhibition of accommodation and line up the target within the narrower depth of field produced by the enlarged pupil. The pupil is somewhat constricted due to the innervation of the parasympathetic system, but not enough to make a difference on the smaller depth of field. Eventually, constant accommodation above the normal level affects the eyes. Our eyes do not prefer to work harder than necessary.

It is important to remember that as accommodation increases, convergence increases also due to AC/A. Convergence is only affected indirectly in this third step controlled by the parasympathetic system, not by the sympathetic system. This increase in convergence is what causes the esoward shift in phoria. Those with a higher AC/A converge more for every unit they increase accommodation, so you are likely to see a greater esoward shift with these individuals. This esoward shift is not tolerable for efficient visual function and serves as the cause of many different forms of acquired visual dysfunction or visual inefficiency.
One could consider a step four to Birnbaum's theory of Nearpoint stress. This would be the shift from the phasic controller to tonic controller (as discussed in adaptations to nearpoint stress). Vergence would make an exoward shift back to normal because once the tonic controller takes over, it does not stimulate the AC/A as the phasic controller does so there is a reduction of convergence that is directly related to the decrease in phasic activity. In this case, vergence and accommodation are now lined up within the depth of field and the person can deal with the nearpoint stress for a longer period of time. This hypothetical fourth step is, in fact, what occurs for visually normal individuals who are not susceptible to visual stress. Birnbaum emphasizes the fact that those individuals who DO NOT shift from phasic to tonic control are at much higher risk for development of stress-induced visual problems, especially development or progression of myopia.

It is obvious stress affects our entire body, including our eyes. It is important to educate patients on the possible effects nearpoint stress can have on the eyes in order to help prevent possible myopia or binocular vision problems. Reminding patients of the need to take breaks from prolonged near work and the preventative effect plus lenses can have on relaxing accommodation are important tips in preventing associated problems with visual stress.
Possible words for Crossword:

Birnbaum
Sympathetic
Parasympathetic
Neurofunctional
Skeffington
Depth of Field
Vergence
Accommodation
Pre-Stress
 Neuromuscular

<table>
<thead>
<tr>
<th>Steps</th>
<th>Pupil</th>
<th>DOF</th>
<th>Accommodation</th>
<th>Vergence</th>
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<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal lag</td>
<td>Normal phoria for certain target distance</td>
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<tr>
<td>2</td>
<td>Large</td>
<td>Small</td>
<td>Abnormal Lag (due to Symp. system)</td>
<td>Same as Step 1, not affected by Symp. system</td>
</tr>
<tr>
<td>3</td>
<td>Smaller than in Step 2, Larger than in Step 1</td>
<td>Same as Step 2</td>
<td>Increased due to Parasymp. system</td>
<td>Increased due to AC/A via Parasymp. system</td>
</tr>
<tr>
<td>4</td>
<td>Same as Step 3</td>
<td>Same as Step 2</td>
<td>Same as Step 3</td>
<td>Relaxes to normal phoria as in Step 1 due to Tonic Controller</td>
</tr>
</tbody>
</table>
Adaptations to Nearpoint Stress

Humans are complex organisms that are constantly at work to keep our bodies in equilibrium. The body uses homeostatic mechanisms to control any change or upset in the balance in our lives. Our eyes are one of the many facets of our body that respond to the environment this way. You may think our eyes are static or that they will only change because we age, but this is untrue. Our eyes are affected by how we use them in day to day life. If we stare at our computer screen for too long and do not blink as often, our eyes respond by producing more tears so our corneas do not dry out. This is a homeostatic response to protect our eyes. Sometimes, when homeostatic responses occur, they resolve one problem in the short term, only to create a new long-term problem. A good example of this is an eye that becomes too powerful so that it can avoid accommodation at near for extended periods of time. This idea of the eye becoming more powerful than needed may be helpful for near work, but when we look in the distance the eye is still too powerful and the distant object does not focus on the retina to form a clear image; therefore myopia has developed.

When we look at close distances for extended periods of time, a process called “Nearpoint Visual Stress” (NVS), can occur. When the eye adapts to NVS by elongating or increasing optical power, this is a “negative adaptation” to stress. A theory, called “Control Systems Theory” is helpful to explain the eye’s response to NVS. It is however just a theory/model to explain an abstract
occurrence of the brain that is not fully understood, and the reader is reminded of this as the theory is discussed.

The phasic controller of accommodation and vergence refers to the fast reflex that occurs due to blur and/or disparity at the retina. This is a fast, strong response that results in accommodation and/or vergence. The parallel response of accommodation and vergence is measured as the AC/A (or CA/C) ratio, which describes the amount of neurological interaction between accommodation and vergence (see notes on AC/A). The phasic controllers are the source for the neurological signals that are the basis of the AC/A (or CA/C). In visually normal individuals, the phasic response lasts only seconds, then the tonic controller is gradually switched on to take over. The tonic controller does not contribute to the AC/A so as the switch to tonic control is made, the AC/A decreases. The tonic system is a slow steady response that can last a long time, unlike the phasic system that has a quick, strong response that does not last for long in normal instances.

These two controllers are like a seesaw. As one goes up, the other goes down; meaning if fixation on a close object continues for a period of time, the phasic controller will gradually decrease its activity and the tonic controller will gradually take over. However, it is important to remember that since the AC/A is not affected by the tonic controller, as we switch to tonic mode, the convergence crosslink signal associated with the AC/A interaction decreases. This reduced convergence innervation is manifest as decreased esophoria or increased exophoria (the phoria drifts in an EXO-ward direction).
This image is an analogy of the Tonic/Phasic Controller model. Think of the turtle as being the slow tonic system and the hare as being the fast phasic system. The turtle is slower, but does not get worn out as fast as the hare, which is fast but does not have the stamina to last as long. The see-saw is a way to remember that, in normals, while one controller is working at its highest level, the other is working at its lowest level.

According to the model, prolonged tonic accommodation is the eventual stimulus for lengthening of the eyeball. It is the body's way (homeostasis) of decreasing the load on the tonic system so it does not have to stimulate accommodation for so long. To prevent this adaptation that leads to myopia, we can add plus power lenses so that we do not have to accommodate as much.

Another way our eyes may adjust is by increasing the tone of the ciliary muscle, essentially "turning up" the baseline level of tonic accommodation. This causes the muscle to return to relaxation much slower and is called **HYSTERESIS**. Accommodative hysteresis is sometimes noticed when people have been reading for a certain length of time (depends on the person) and notice distance blur when they look up. The blur in the distance gradually goes away and clarity returns as the ciliary muscle relaxes. This is often the first sign of the adaptation our eyes are making to the NPS, and if left to progress, may
cause myopia. Accommodative hysteresis is more common in myopes, especially those that became myopic later in life (late-onset myopes).

Our job as optometrists is to prevent hysteresis from developing into manifest myopia. Once the eyeball has grown, it will not “ungrow,” but if the eye still has the capability to relax and make a clear image in the distance, there is hope. We can urge the patient to wear low plus lenses while reading to prevent overstressing the eye. We must also be careful with people that show slight myopia after working a long day in front of the computer. This may be a hysteresis effect and not true myopia. If we prescribe them full-time low minus lenses, the lenses will create an even greater accommodative demand, because minus lenses diverge the light coming from the object, making our eyes work harder to focus the light precisely on the retina. This may make accommodative hysteresis and eventual myopia even worse.

Another way to resolve the esphoric shift that occurs in NVS is to adaptively reduce the tonic innervation level to convergence. Such a change reduces the esophoria at near, but can cause a secondary divergence excess exophoria (or tropia) when the individual looks into the distance. It is also possible that the decreased tonic innervation to vergence can give rise to convergence insufficiency (excessive exophoria/tropia at near) when stress levels are low and the stress-induced eso shift does not occur.

Another adaptation to decrease the need for precise vergence is to suppress the central vision in one eye. Central suppression creates a “dead zone” so that vergence errors are not registered. This can result in binocular
vision problems and reduced stereopsis. Some individuals will adapt to NVS by developing a type of anisometropia where only one eye becomes myopic. In this case, the myopic eye is used for near vision and the fellow eye for distance vision with suppression alternating between the two eyes. When this occurs, binocular vision is compromised for all distances, but the eso shift can be tolerated due to the presence of central suppression.
The Myopic Eye

Myopia is a refractive condition where the size of the eye and the power of the eye do not match so that the image of an object is focused in front of the retina. There are many different theories to explain the causes of myopia and to classify myopia based on its magnitude and components of the eye that contribute to it.

Classification and Causation of Myopia

Curtin wrote about the optical sources of myopia. He classified myopia as physiologic, intermediate, or pathological. Physiologic means that all of the refractive components of the eye are normal, but they do not match up to produce an emmetropic eye. The eye may be a little too long or the cornea too powerful, but both amounts of deviation are within normal limits. This type of myopia is often hereditary, but may be caused by the environment as well (for example, too much nearpoint work).

Intermediate myopia is due to a lengthening of the posterior portion of the eye. It carries characteristics of both physiologic myopia and pathological myopia and the degree of myopia is usually within 3-5D.
Pathological myopia is when the myopia is over 12D. This type of myopia is often related to ocular diseases such as neural tissue pulling away from the optic nerve, tessellation, or retinal detachments in the periphery. Neural tissue may pull away because the sclera is growing and the neural tissue (cone and rod receptors and related cells) is not so it tears. Pathological myopia can be considered either correlation or component myopia. Correlation myopia is when the power of the cornea and the eyeball length do not match up to create a clear image on the retina. Both are within the normal limits, but the two do not correlate. Most correlation myopia is environment related. Component myopia results when all optical components are within the normal range except one. For example, the cornea may be way too powerful, but the length of the eye is normal. Sorsby said that correlative myopes have up to 4.00D of myopia (sometimes up to 6.00D) and component myopes have myopia greater than 4.00D.

Figure 1: Correlation Myopia
The solid line represents the normal distribution of eye length and the dashed line represents the normal distribution of power of the cornea. If both components are within the normal range of the population (below the central part of the curve; usually defined as ±1 standard deviation of the mean), it is considered correlation myopia (Figure 1). If one component of the eye is out of the normal range (power of the cornea in this case), it is component myopia (Figure 2).

Borish's classification of myopia is based only on the magnitude of the refractive condition. He sees all myopia as being similar except in varying amounts:

- <1D = Very low myopia
- 1-3D = Low myopia
- 3-6D = Medium myopia
- 6-10D = High myopia
- >10D = Very high myopia
Birnbaum mostly studied non-pathological myopia, but he recognized that there were three different types of myopia; congenital which is rare, non-pathological, and pathological. He said non-pathological myopia is progressive, and the myopic changes that occur with it can be divided into three phases. The first phase is INCIPIENT (transient). This occurs when the tonic controller of accommodation does not fully relax after performing near tasks for long periods of time. Hysteresis is the term used to describe the slowness in relaxation of the tonic system. Patients may have symptoms of asthenopia and an esoward shift in phoria after doing extensive near work, and therefore may avoid near work because of this. They may also have a low positive relative accommodation due to accommodative inhibition. We often prescribe these patients low plus lenses for near and if needed, train them to increase their accommodative facility.

Birnbaum’s second phase of non-pathological myopia is ACTIVE PROGRESSIVE myopia. The patient no longer has asthenopia but they are noticing constant distance blur. They often remain esophoric at near and at this point, the myopia is manifest so it is harder to reverse. It is often a good idea to cycloplege the patient to see if any myopia remains once accommodation is fully relaxed by the cycloplegic drug.

The third phase of non-pathological myopia is STABLE MYOPIA. The growth in myopia has stopped, but it may become progressive again if more stress is induced on the patient. This type of myopia is almost always irreversible.
Review: Birnbaum’s Phases of Non-pathological Myopia:

1) Incipient
2) Active progressive
3) Stable myopia

Etiology of Myopia

The cause of myopia is still debated and is receiving considerable attention in research labs around the world, but we know that there are both genetic and environmental factors involved. In its simplest form, the Genetic Theory of Myopia proposes that myopia is inherited from one’s parents. Indeed, it has been shown that there is a greater chance for someone with one myopic parent to develop myopia than someone whose parents are not myopic. The risk factor for myopia development further increases if both parents are myopic. However, it is also known that the children of myopic parents do not always develop myopia themselves. Myopia is thought to be caused by more than one gene and may be connected to the way we think and behave. Researchers have attempted to prove the genetic basis of myopia by doing twin studies. However, it is hard to separate what is caused by genes and what is caused by environment in twins living together. Some studies have been done on twins that have been separated, but there are not as many similarities in myopia. This may be evidence that environment also plays a role in the development of myopia. Certain ethnic groups are often more likely to have myopia than others, and this may be evidence of the genetic causation of myopia. However, these groups often share the same environment as well. It is most likely that myopia is caused by a combination of environment and genetics.
Some researchers, such as Sorsby, said we should not worry about the causation of myopia. He thought it was a normal variation in the gene pool, just as red hair is a variation. He called his theory the **Normal Biological Variation Theory**. He thought that myopia only occurred because the optical components of the eye did not correlate to form an emmetropic eye. He rated the different components on their likeliness to cause myopia. Axial length has the greatest effect on myopia; cornea has the next greatest effect, then the lens, and lastly the depth of the anterior chamber of the eye.

A third theory on the cause of myopia is the **Conditions of Use Theory**. This theory is based on the idea that myopia is a result of how we use our eyes. This goes back to the ideas Birnbaum and Skeffington had about nearpoint stress causing an esoward shift in phoria, and the prolonged increase in tonic innervation which eventually could result in an elongated eye as a homeostatic response to stress. Studies have shown an esoward shift after near fixation but not after far fixation. Gilmartin stated that the sympathetic system may turn on in order to stop hysteresis affects and to bring the phoria back to an exoward direction. Owens and Wolf showed that both tonic accommodation and tonic vergence increased after 25 minutes of reading, supporting the idea that prolonged near work can contribute to development of transient myopia. Bell also contributed his theory on scleral stretching from over-accommodation. He thought that accommodation caused the sclera to stretch more because of the pull at the ciliary body, and therefore resulted in a weakened sclera that would be more prone to the axial elongation common in myopia. Coleman supported this
theory by adding that IOP increases in the vitreous chamber when a person accommodates, and this may be the cause of the stretching. The question has been raised whether the sclera is actually stretching the cells it already has or if there are actually more cells being produced to lengthen the sclera. There is no confirmed answer at this point and this theory is not well accepted.

Another theory on the cause of myopia is the Holistic Theory. This theory says that myopia is related to the whole person rather than just the eye alone. People that advocate this theory believe it is not accommodation that causes myopia, but rather the way we use our eyes during near work. If we are under stress or concentrating excessively, the near work is more likely to relate to myopic changes.

Personality may also be related to myopia according to the Holistic Theory. Introverts are more likely to be myopic and extroverts are more likely to be hyperopic. Myopes also tend to lean forward in space due to the concave lenses they wear for correction. This may make them focus on the ground more instead of objects at a distance. They will accommodate when looking at the ground and this may result in even more myopia. To prevent this we can prescribe base down prism in front of the eyes to shift the world upward (toward the apex of the prism) which in turn brings the eyes upward.

Nutrition is also considered in the Holistic Theory. There is evidence that countries with a higher level of malnutrition have a higher level of myopia. It is thought that if a person is Vitamin C deficient, their sclera does not have as much collagen formation and may be more prone to weaken and stretch. There is also
the idea that myopes tend to take in more carbohydrates and flesh proteins
compared to hyperopes, but less fiber.

The last theory we will discuss on the etiology of myopia is the
Deprivation Theory. This is the idea that if we deprive the eyes of normal sight,
they will tend to become more myopic. For example, if we focus at near for too
long, only the central retina is being stimulated so peripheral retina is being
deprived. Lenses in front of the eyes may also deprive the peripheral retina
because only certain rays of light are allowed to enter the eye. Josh Wallman,
Ph.D., did a study on baby chicks to see if extended blocked peripheral vision
would have an effect on normal use of peripheral vision. The results showed that
the chicks developed peripheral myopia; meaning their peripheral vision was
blurry compared to central vision.

Our eyes are deprived of color contrast when we stare at white paper with
black writing all the time and this may also be related to myopia. Research on
the Deprivation Theory has proven that environmental factors can play a
significant role in myopia development. The area continues to receive
considerable research effort and new insights are revealed every year about how
the eyes and vision respond to various conditions in terms of myopia
development and, in some cases, myopia reduction or reversal.

So how do we prevent myopia from occurring? Some think to cycloplege
the eye will prevent accommodation and therefore prevent the scleral
stretching/growth that would occur from over-accommodation. However, a drug
called Perenzipine, which is not a true cycloplegic drug, has been shown to
reduce the progression of myopia, but it is uncertain how. A much easier and less harmful way of possibly controlling myopia is by prescribing plus lenses for near work so the patient does not have to accommodate as much because the lenses provide the power needed for near. These are especially helpful for people with high AC/A that end up over-converging when they have to accommodate so much. The plus lenses reduce the stimulation for convergence so the patient does not make as much of an esoward movement in phoria at near.
REFERENCES
