Prevalence and expectations of congenital myopia

Kirk T. Reed

Pacific University
Prevalence and expectations of congenital myopia

Abstract
A comprehensive literature search was conducted to provide an updated review of the incidence of myopia among newborns, and to give information on the likelihood of certain endpoints to the progression. Beginning with an etiological review to serve as a reference point, some of the most common theories are covered. The body of the thesis considers reviews of past works designed to determine prevalence of myopia of various age groups. The reviews are divided into 1) prevalence at birth, 2) prevalence up to school age, 3) prevalence through school years and beyond and 4) a review of progression and cessation literature. Tables from various authors are included to aid in a quick overview of the data compiled over the years. It is this author’s conclusion that the final myopic cessation magnitude is dependent upon family history, the child’s initial refractive error trend and a generalization that early high myopes progress rapidly and steadily to higher amounts.

Degree Type
Thesis

Rights
Terms of use for work posted in CommonKnowledge.

This thesis is available at CommonKnowledge: https://commons.pacificu.edu/opt/1143
Copyright and terms of use

If you have downloaded this document directly from the web or from CommonKnowledge, see the “Rights” section on the previous page for the terms of use.

If you have received this document through an interlibrary loan/document delivery service, the following terms of use apply:

Copyright in this work is held by the author(s). You may download or print any portion of this document for personal use only, or for any use that is allowed by fair use (Title 17, §107 U.S.C.). Except for personal or fair use, you or your borrowing library may not reproduce, remix, republish, post, transmit, or distribute this document, or any portion thereof, without the permission of the copyright owner. [Note: If this document is licensed under a Creative Commons license (see “Rights” on the previous page) which allows broader usage rights, your use is governed by the terms of that license.]

Inquiries regarding further use of these materials should be addressed to: CommonKnowledge Rights, Pacific University Library, 2043 College Way, Forest Grove, OR 97116, (503) 352-7209. Email inquiries may be directed to: copyright@pacificu.edu

This thesis is available at CommonKnowledge: https://commons.pacificu.edu/opt/1143
PREVALENCE AND EXPECTATIONS
OF CONGENITAL MYOPIA

By Kirk T. Reed

A Thesis submitted to the faculty of the College of Optometry
Pacific University
Forest Grove, Oregon

for the degree of Doctor of Optometry
May 1994

Advisor:
Richard D. Septon, O.D.
PREVALENCE AND EXPECTATIONS

OF

CONGENITAL MYOPIA

By

Kirk T. Reed

Advised by

Dr. Richard D. Septon
ABOUT THE AUTHOR

Kirk T. Reed was raised in Mesa, Arizona. There he graduated from Mesa High School in 1979. After a couple years of church service, he began studying again at Mesa Community College, earning an associates degree in pre-optometric sciences. From here he went on to received a Bachelors degree in psychology from Arizona State University in 1989. Kirk was accepted into Pacific University's College of Optometry in the fall of 1990, expecting to receive his doctorate of optometry degree on May 22, 1994.
DEDICATION:

This work is dedicated to my son Cody and to my wife. If it were not for their myopic status, I would not have had this interest.
ACKNOWLEDGEMENTS

I would like to thank Dr. Richard D. Septon for his enthusiasm about optometry in general and specifically concerning the topic of myopia progression. His insights into trends of refractive errors gave me my first interest into trying to predict the future for myopic infants.
Prevalence and Expectations of Congenital Myopia

Kirk T. Reed

Pacific University College of Optometry
Forest Grove, Oregon

Abstract:

A comprehensive literature search was conducted to provide an updated review of the incidence of myopia among newborns, and to give information on the likelihood of certain endpoints to the progression. Beginning with an etiological review to serve as a reference point, some of the most common theories are covered. The body of the thesis considers reviews of past works designed to determine prevalence of myopia of various age groups. The reviews are divided into 1) prevalence at birth, 2) prevalence up to school age, 3) prevalence through school years and beyond and 4) a review of progression and cessation literature. Tables from various authors are included to aid in a quick overview of the data compiled over the years. It is this author's conclusion that the final myopic cessation magnitude is dependent upon family history, the child's initial refractive error trend and a generalization that early high myopes progress rapidly and steadily to higher amounts.
INTRODUCTION

Among the volumes that have been written concerning myopia, there are still many unanswered questions. With each study, and paper that is written, to answer a particular question comes a multitude of other questions to be answered. Included in these are questions that parents might ask when told that their baby is nearsighted. They may want to know how nearsighted their baby is, and to what degree it will progress. There have been many prevalence studies citing the percentage of myopes in a particular population at that particular time, and studies following youth-onset myopia through its progress to cessation. There are even studies estimating the amount of myopic increase per year that can be expected. However, none at this point in time, have taken up the specific problem as to estimating an age and amount of myopia at cessation when it is found at birth. It is intended here to draw from previous works and to extrapolate for the particular case of a congenital myope and give some possible answers as to the amount of myopia at cessation.
PART I

ETIOLOGICAL REVIEW
ETIOLOGY

There is a prevailing view that myopia is not a single entity, but a condition embracing many different origins. One of the best examples is pointed out in a study sponsored by the U.S. Air Force School of Aerospace Medicine\(^1\). They showed that severe myopia can be teased out from the rest of the myopias on the basis of the refraction curve. A skewed curve becomes nearly symmetrical when individuals with high degrees of myopia are taken out. Severe myopia is often associated with other problems such as prematurity or infectious diseases and it appears at a much earlier age. Goldschmidt's\(^2\) work on the etiologies of myopia points out that myopic ranges between 6.00-9.00D do not group to any particular occupational categories, but that it is spread evenly throughout occupations. Interestingly, he also states that severe myopia, at least in Denmark, has decreased in prevalence since the 1880's.

In the same paper, Goldschmidt distinguishes between childhood myopia (which develops during the learning years in school and stabilizes in the late teens or 20's) and an adult-onset myopia which doesn't start until one enters into a specific occupational environment. This adult onset myopia does not stabilize, but instead progresses on with continued work in that occupation.

The identification of the etiology of myopia has been a concern for many years. Some of these have been embraced by optometrists and ophthalmologists alike. Although not the primary concern of this paper, a few of the prevailing etiological ideas will be covered to provide a basis for understanding throughout the remainder of this paper.

Genetic

Perhaps the most intensively studied etiology is that of genetics. It is not uncommon for a doctor to ask the parents of a myopic child if either of them were nearsighted. The goal of many papers, this work included, has been to determine whether or not myopia was a function of genes passed down from previous to succeeding generations.

Goss et al.\(^3\) having reviewed the materials written on the genetic component of myopia, stated that there is a lack of consistency between the results and the proposed nature of inheritance. Low myopia most probably is polygenic in nature, where many factors are involved including environmental

KIRK T. REED
influences. The genetic role is also supported by the consanguinity of myopia, racial differences in myopia and incidences such as the incidence of myopia remaining high in Asians who have relocated to Hawaii. Jews, and other genetic subgroups, also have a greater prevalence of myopia. One must be careful when assigning myopic tendencies to a cause such as inheritance, since the same myopia might be derived from the culture or environment. Studies which seem to prove the genetic presence might in reality be supporting an environmental etiology. An example of this is the frequent incidence of myopia found among the Eskimos and American Indians upon compulsory education, where previously they were a relatively non-myopic population. Once near work became a common activity, the incidence of myopia rose.

A key point to remember is that neither genetics alone nor the environment alone may account for the entire amount. The polygenic nature of myopia is combined with the complex intertwining of the environment. Bastien wrote about a person being born with certain genetic predispositions to myopia. Gosset et al. concluded that the genetic input from which myopia development probably arises, most likely comes from more than one gene. It is probable that myopia development results from a complex interplay of genetics and environment.

This broad dichotomy in the study of the etiology of myopia is often referred to as nature vs. nurture. As defined by Angle and Wissmann "nature" is a biological theory that views myopia coming from genetically determined characteristics of the eye, where as the "use-abuse" aspect of the "nuture" theory views myopia as the result of habitual use of the eye at a near focal length. They felt that both theories played a valid role in explaining myopia. In favor of the genetic theory is the fact that spherical refractions of identical twins have a higher correlation than those of fraternal twins. In addition, no one has of yet discovered how muscular tension permanently modifies spherical refraction. Unexplained is how the genetic expression must be pre-programmed to be more common in females, to appear in adolescence in some, to be more frequent in certain races, to express itself more often in the better educated and white collar workers, and to be more prevalent in industrial and urban locations.

Grosvenor cites an interesting screening of 50,000 schoolchildren including various racial groups living in Hawaii. They found a large range in
myopic prevalence between three different races. Polynesian was the lowest with 3% being myopic, Caucasians next at 12%, and Chinese children had the highest prevalence where 17% were myopic. Once again, seemingly supportive of genetics, myopic variations between the groups could easily stem from cultural differences triggered by anything from diet to special pastimes favored by one subgroup.

**Biological Variation**

A subgroup of the genetic etiology would be biological variation. As the name implies, this theory considers the optical components of the eye, the cornea, crystalline lens and axial length, to vary in substantial amounts to elicit a focal point falling short of the retina. The basis of this theory, also known as statistical theory, was first postulated by Steiger\(^7\), in 1913. Just as heights may vary among a group of people, ametropia is present for different reasons. One tall person may have long legs, where as another may have a trunk and neck that is of greater length than others. It may also be a combination of a number of these components. The myopic ametropia can have similar variation, none of them in and of themselves, being significant, but combined leave the person "nearsighted".

**Environmental**

Opposingly, another broad etiological category is that of environmental effects upon myopic shifts. Factors in this area that have been implicated range from near work and psychological stress, to dietary changes.

**Near work**

Extending back as early as Cohn, 1867, authors have been writing to convince readers that near work is a factor in creating myopia. Reading being the most common, any near task demanding attention was suspect. Evidence has been compiled over the years supporting the facts that increased myopia occurs in higher educational levels, groups that show high literacy, groups that are involved with long hours of near work and which show a sudden increase in a population when mandatory schooling has been introduced. Adams et al\(^1\) felt
that assuming future myopes to be the same as future nonmyopes refractively, one could say that myopia came from reading rather than people leaning to near tasks because they were nearsighted. Others felt that directionality could be determined at an early age. For example, myopic children tend to be readers before the myopia expressed itself. Not only is near work suspect, but "closed in" environments have also been implicated in this progression. Young, in 1967, cites studies claiming that animals raised in closed in environments such as laboratories, developed more myopia than thoses raised in the wild. Accommodation was given as the mechanism by which these changes take place. When accommodation was not a factor, due to the use of cycloplegics, myopia did not appear.

Whether myopia is from the stress of near work or is swayed more by biological variations is still not proven. To accurately judge the final outcome of each individuals myopic progression, we must know the extent to which each etiological factor will influence his life.
Recent data shows that myopia exists in up to 25% of the population of industrialized countries and is increasing in children and adults alike. One-fifth of those who are either hyperopic or emmetropic prior to entering college, will become myopes during their college years. Myopes among professional students has been rated as high as 80%. Within our own profession, Septon has found a prevalence of 75% in student populations. He also sought to determine the age of onset in addition to the prevalence during optometry school. It is commonly known that the greatest number of myopes enter nearsightedness during school years ranging from 7 to 15 years old. These increase in myopia through the teen years and level off, so that by adulthood, approximately 25% of all adults are myopic. When does this myopia first present itself? Is it to be a permanent part of the individuals life? To what extent will it progress? A review of previous studies will summarize what has been written to date.

A number of studies between the years of 1919 and 1936 agreed that hyperopia was the overwhelming rule and that a myopic newborn was exceptionally rare. One study that went against the rule was an earlier one in 1861 by Von Jaeger, who when reporting on 100 infants stated that 78% were myopic and only 17% hyperopic. These babies were in the second week of life and were examined without the use of cycloplegics, which may lead to excessively high myopic findings. In 1880, Ely studied 100 eyes during the first week of life. In order to rule out the examiners accommodation, he cyclopled not only the baby, but himself also with 0.5% atropine. Hyperopes were found to be the rule in 72% of the eyes, 17% emmetropic and only 11% myopic. After the original 100 eyes, Ely studied an additional 49 eyes with only a weak atropine solution in the babies eyes and no cycloplegia for himself. This examination gave exaggerated myopic values for 16 eyes, 4 emmetropic and only 29 of the 49 were hypermetropic.

The same year that Ely reported his findings, Horstmann had examined infants himself, 40 in number, between the ages of 8 and 20 days. Ten percent were found to have .50 D to 1.00 D of myopia. Four years later he examined another 100 newborn eyes this time finding only 2% of the eyes to be myopic. In 1881, Konigstein reported 10 cases of emmetropic eyes of 562, the rest being...
hyperopic. Schleich in 1884, also found no myopic eyes when he examined 300 eyes. That same year a Danish ophthalmologist took the 100% hyperopic findings to a new limit. He studied 87 infants that ranged from 7 hrs to 14 days old. Not only were they all hyperopic, but those less than 5 days old were thought to show a pronounced hyperopia which decreased in the older infants examined. Another 100% hyperopic percentage was given in 1884 by Ulrich who examined 102 newborn infants. A year later 110 infants less than 80 days old were all found to be hypermetropic by Germann. A group of 168 eyes within the first month had an average of +5.37D. Forty eyes in the second month had a lower average of +3.30D. The year 1892 brought us the first myopic finding again when Herrnheiser examined 1920 eyes and discovered one case of myopia. These infants, which were in their second week of life were found to have an average refractive error of +2.32D. All but two had between +1.00 and +6.00D hyperopia. In 1893, Biegel found only hyperopes in 39 babies, while de Vries found 5 of 78 infants to have myopia.

Up to this point all examinations had been done with ophthalmoscopes. The rare occurrence of myopic findings can hardly be due entirely to the technical difficulty of this instrument. Most likely the investigators made a systematic, suggestive error on the hyperopic side. Due to the large number of studies in the late 19th century, many of the ophthalmological texts written between the years of 1919 and 1936 took it for granted that myopic refractive errors were scarce and congenital findings were nearly always hyperopic. Any refractive error showing a myopic finding was thought to be a congenital defect. Hyperopia was the norm and any myopia was a state to be developed later.

In 1925 Wibaut undertook a very large study. He looked at the findings of 2398 infants who were examined by several different ophthalmologists. This study suffered from lack of consistency due to the difficulty of the job and the variety of examiners. They found that 99.22% of the babies were hyperopic ranging mostly from +1.00D to +4.00D, while .7% were emmetropic and only .08% (3 cases) were myopic. Such a rare occurrence of myopia can be attributed to the fact that nearly all of Wibaut's data came from Herrnheiser who found only one case of myopia in approximately 2000 eyes. A sampling problem such as this would not take place if the data were evenly distributed among many different examiners.

Cook and Glasscock12, noting that recent refractive error findings on the newborn was scarce, began a study of their own in 1951. Now being one of the
most often cited studies, they examined 1000 eyes which had undergone 4 installations of 1% atropine during the preceding 30 hours. Simple hyperopia was found in 43.9% of the eyes. Hyperopic astigmats were found to be 29.1% prevalent. Simple myopia was found in 16.7% and myopically astigmatic babies in 6.4%. Overall, 74.9% were hyperopic. Over 50% of these were from less than +1.00D to +3.00D as can be seen in Table1.

The greatest number of myopes, 6.6%, required 1-2D of correction, with a range of less than one diopter to twelve diopters. The overall amount was 25.1% myopia. Eighty-eight percent of these ranged from less than one to five diopters.

<table>
<thead>
<tr>
<th>diopters</th>
<th>% hyperopia</th>
<th>% myopia</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>6.9%</td>
<td>5.0%</td>
</tr>
<tr>
<td>1-2</td>
<td>20.5</td>
<td>6.6</td>
</tr>
<tr>
<td>2-3</td>
<td>17.6</td>
<td>2.5</td>
</tr>
<tr>
<td>3-4</td>
<td>12.0</td>
<td>5.4</td>
</tr>
<tr>
<td>4-5</td>
<td>7.0</td>
<td>2.8</td>
</tr>
<tr>
<td>5-6</td>
<td>4.6</td>
<td>0.9</td>
</tr>
<tr>
<td>6-7</td>
<td>3.1</td>
<td>1.2</td>
</tr>
<tr>
<td>7-8</td>
<td>1.9</td>
<td>0.1</td>
</tr>
<tr>
<td>8-9</td>
<td>0.7</td>
<td>0.0</td>
</tr>
<tr>
<td>9-10</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>10-11</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>11-12</td>
<td>0.2</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>74.9%</td>
<td>25.1%</td>
</tr>
</tbody>
</table>

Goldschmidt also set out to substantiate the distribution of refractive errors in the newborn. His study consisted of 356 infants less than 10 days old. All of them were greater than 2500g in weight at birth, which is considered full term. Cycloplegia was achieved by one drop of .5% atropine given 24 hours prior to examination. His results are as shown in Table 2 below.
The mean refraction was found to be +.62D. The frequency of myopia was 24.2%. These results obviously confirm Cook and Glasscock's findings that many more infants are found to have myopic refractive errors than were originally thought. Earlier studies prior to Biegel (1893) all used ophthalmoscopes, which might have given rise to such misfindings. Another reason would be the technical difficulty in examining infants. What Goldschmidt was to definitively prove is that various amounts of ametropia are found in the newborn. Myopia could not be considered so rare among newborns as was once thought. The first hint to an answer of my question of where can we expect the congenital myope to end up refractively was given in Goldschmidt's concluding remarks when he assumed that in the majority of cases, the degree of congenital myopia will decrease and the nearsightedness may even disappear altogether, since more cases of high myopia were among the infants than among schoolchildren. Is it possible to predict a final refraction on a child based on early first refractions? We are at point in our search of myopia that rather than prevalence studies at selected ages, longitudinal studies need to be embarked upon.

A recent work by Martin Banks in 1980 pointed out the tedious, time-consuming, technically difficult task of measuring infant vision. In these more recent studies there appears to be a general agreement upon capabilities. He compiled a table, found below as table 3, of several investigators works. He noted that they commented on the technical difficulties involved. Within refractive errors regardless of the differences in subject populations, the...
refractive error of newborns generally appear to be normally distributed (bell-shaped) with a mean about +2.00D and a standard deviation of 2.00D.

TABLE 3
BANKS14
REFRACTIONS OF NEWBORN INFANTS

<table>
<thead>
<tr>
<th>INVESTIGATOR</th>
<th>AGE (DAYS)</th>
<th>SUBJECTS</th>
<th>TECHNIQUE</th>
<th>MEAN REFRACTION / S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herrnheiser (1892)</td>
<td>-</td>
<td>960</td>
<td>Retinoscope</td>
<td>+2.30D</td>
</tr>
<tr>
<td>Santonastaso (1930)</td>
<td>3-6</td>
<td>100</td>
<td>Retinoscope</td>
<td>+2.00</td>
</tr>
<tr>
<td>Francesco Schetti (1930)</td>
<td>1-2</td>
<td>96</td>
<td>Retinoscope</td>
<td>+2.40 / 2.3</td>
</tr>
<tr>
<td>Cook and Glasscock (1935)</td>
<td>-</td>
<td>500</td>
<td>Retinoscope</td>
<td>+1.80 / 3.1</td>
</tr>
<tr>
<td>Graham and Gray (1963)</td>
<td>-</td>
<td>88</td>
<td>Retinoscope</td>
<td>+2.60 / 1.9</td>
</tr>
<tr>
<td>González (1965)</td>
<td>-</td>
<td>100</td>
<td>Retinoscope</td>
<td>+2.30 / 1.2</td>
</tr>
<tr>
<td>Mehta and Associates (1965)</td>
<td>0-1</td>
<td>280</td>
<td>Retinoscope</td>
<td>+2.20 / 1.6</td>
</tr>
<tr>
<td>González (1965)</td>
<td>2-2</td>
<td>356</td>
<td>Retinoscope</td>
<td>+2.40 / 1.6</td>
</tr>
<tr>
<td>Hosaka (1971)</td>
<td>0-1</td>
<td>250</td>
<td>Retinoscope</td>
<td>-</td>
</tr>
<tr>
<td>Zonis and Associates (1970)</td>
<td>2-3</td>
<td>300</td>
<td>Retinoscope</td>
<td>+1.10 / 1.6</td>
</tr>
</tbody>
</table>

Fletcher and Brandon15 writing "embryologically", point out that myopia is associated with immature eyes and it is metabolic disturbances which result in increased axial length, increased corneal curvatures and increased index of refraction. Mukherji et al16 feels that low amounts of ametropia, myopia included, is just a normal variation, not pathological. Myopia, to date, has long been noted, in newborns, especially among premature infants. They studied the refractive errors of 500 newborn babies. Of these, 140 were premature, meaning that they were born at or before 37 weeks or were below 2500g in weight. Within the group of 500 infants, 44 (8.8%) were emmetropic, 345 (69.0%) were hyperopic, and 111 (22.2%) were myopic. They found that of those myopic, 96.8% were considered to have low myopia (<5.00D). A high myopic refractive error (>5.00D) was found in only 7 (3.2%) of the cases as found in table 4.
They went on to tell us more about those who had the nearsightedness. One-third of the 222 eyes had some degree of astigmatism. The incidence of myopia was much larger among infant females, 60.3%, than in the infant males, 39.6%. And as was previously shown, the lower birthweight babies had a higher incidence of myopia than those with a higher birthweight. Since this study included premature babies, it is possible to look at how the gestational age is an etiological factor in congenital myopia. Those babies who went full term, which is usually considered 38-40 weeks, had a lower prevalence of myopia than those who were born prematurely. In fact, there is a dramatic increase in myopia with each week of prematurity, to the point that three-fourths of those born at 36 weeks can be expected to be myopic. Tables taken from Mukherji’s article are included below.

Another way to look at this is to consider birthweight, since weeks of gestation is related to weight at birth. The infants who had a lower birthweight also had the higher prevalence of myopia.

KIRK T. REED
TABLE 6
MUKHERJI16
INCIDENCE OF MYOPIA ACCORDING TO BIRTH WEIGHTS

<table>
<thead>
<tr>
<th>BIRTH WEIGHT</th>
<th>NUMBER OF EYES</th>
<th>PERCENT MYOPIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2500g</td>
<td>228</td>
<td>48.2%</td>
</tr>
<tr>
<td>2500-3000g</td>
<td>730</td>
<td>13.9%</td>
</tr>
<tr>
<td>&gt;3000g</td>
<td>62</td>
<td>16.1%</td>
</tr>
</tbody>
</table>

And finally, Mukherji compiled a table of infant refractive error studies that took place over the last forty years.

TABLE 7
MUKHERJI16
INCIDENCE OF MYOPIA IN NEWBORN BABIES

<table>
<thead>
<tr>
<th>YEAR</th>
<th>AUTHOR</th>
<th>NUMBER OF EYES</th>
<th>PERCENTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1951</td>
<td>COOK &amp; GLASSCOCK</td>
<td>100</td>
<td>23.1%</td>
</tr>
<tr>
<td>1960</td>
<td>Patel</td>
<td>500</td>
<td>12.0%</td>
</tr>
<tr>
<td>1965</td>
<td>Mehra ET AL</td>
<td>1000</td>
<td>9.0%</td>
</tr>
<tr>
<td>1979</td>
<td>CHATTERJEE &amp; MUKHERJI</td>
<td>500</td>
<td>19.0%</td>
</tr>
<tr>
<td>1983</td>
<td>MUKHERJI</td>
<td>500</td>
<td>22.2%</td>
</tr>
</tbody>
</table>

Summary

There is much variability in the reports about the actual prevalence of myopia at birth. It ranges from a high of 78% by Von Jaeger to a low of 0% in most of the earliest studies. Myopia is generally considered to average around 20-25% at birth as is supported by the tables above. Similarly, the average refractive error at birth, as reported in most studies, seems to be consistently close to $+2.00\text{D}$. Of those myopic, the vast majority fall in the low to moderate range of $-1.00\text{D}$ to $-4.00\text{D}$. To vary from expected norms of weight and length of gestation is to dramatically increase chances of being myopic.

KIRK T. REED
Part IIIB

Review: Myopia Prevalence Up To School Age

The most difficult part of my undertaking is filling in the gap during the ages between infancy and five to six years of age, the age when children enter school. There is an almost limitless supply of subjects in the 0-4 week old category due to easily accessed records and availability of subjects to test. There is no easy accessibility again until they all come together for school. One possible source might be day care centers. Yet even this source might not be truely free of selection biases.

Trying to bridge this gap, Mohindra and Held 17 used their non-cycloplegic procedure of "near retinoscopy" to refract 400 babies between the ages of birth to five years of age. An important note coming from their refractions is that of a narrower bell curve as the baby ages. Under 4 weeks of age a distribution as wide as -14.00D to +12.00D was found. By the age of 2.5 years to 5 years old, the range narrowed considerably down to -3.00D to +4.00D. They found a 50% prevalence of myopia at birth. This decreased to 15% by the ages of 2.5 to 5 years. Notwithstanding the possible falsely high myopic percentage due to a non-cyclopleged retinoscopic technique, their data supports the hypothesis that emmetropization is in effect during the early years of life.

Grosvenor 19 in his review of classification systems has suggested that myopia should be classified based on age of onset. In his review of articles, the presumably more valid recent studies show myopia to be prevalent in about one-fourth to one-half of neonates. This has dropped off to 1%-2% by the time the children enter the first grade, as is clearly shown in his table, reproduced below.
TABLE 8
GROSVENOR19
PREVALENCE OF MYOPIA FOR CHILDREN
FOR BIRTH TO AGE 6-8 YEARS

<table>
<thead>
<tr>
<th>AGE</th>
<th>SOURCE</th>
<th>SUBJECTS</th>
<th>CRITERION</th>
<th>PREVALENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIRTH</td>
<td>COOK &amp; GLASCOCK</td>
<td>CAUCASIAN</td>
<td>ANY MYOPIA 24%</td>
<td></td>
</tr>
<tr>
<td>BIRTH</td>
<td>GOLDSCHMIDT</td>
<td>DANISH</td>
<td>ANY MYOPIA 25%</td>
<td></td>
</tr>
<tr>
<td>BIRTH</td>
<td>MOHINDRA &amp; HELD</td>
<td>AREA</td>
<td>ANY MYOPIA 50%</td>
<td></td>
</tr>
<tr>
<td>5-6</td>
<td>HIRSCH</td>
<td>LOS ANGELES</td>
<td>≥-1.00D 1%</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>BLUM ET AL</td>
<td>ORINDA, CA</td>
<td>≥-0.50D 2%</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>HIRSCH</td>
<td>OJAI</td>
<td>≥-0.50D 2%</td>
<td></td>
</tr>
<tr>
<td>6-8</td>
<td>KEMPH ET AL</td>
<td>CAUSASIAN</td>
<td>≥-0.50D 2%</td>
<td></td>
</tr>
<tr>
<td>7-8</td>
<td>LAATIKAINEN &amp; ERKKILA</td>
<td>FINNISH</td>
<td>≥-0.25D 1%</td>
<td></td>
</tr>
</tbody>
</table>

Thus, as suggested by the table there is an underlying constant of myopes who once born into nearsightedness, will remain so throughout life. The majority of the congenital myopes, being myopic due to some prematurity or underdevelopement of the eyes, will decrease in their negative refractive error throughout their first few years due to those emmetropizing effects. Those 2% who remained generally had a sufficient amount of myopia that it will persist throughout life. These are the ones that Grosvenor calls "congenital" myopes. Further study could show whether or not those who have departed from myopia by first grade will return to it sometime during their school years. A longitudinal tracking from birth is necessary to answer this.

Ingram and Barr20 sampled 148 children in the United Kingdom and found that between the ages of 1 and 3 1/2 years old, those babies having myopia and astigmatia changed in prevalence towards greater emmetropia. Likewise, hyperopic children less than +2.25D shifted towards emmetropia. Those having hyperopia greater than +2.50D were about equally split in increasing and decreasing hyperopia.

Ingram21 published in the same year, 1979, an article wherein atropine cycloplegia was used in determining refractions of 1 year olds. The overwhelming majority of babies in this age group were between plano and +2.50D, with the mode at +0.50D to +0.75D. Once again, myopes were in the minority of about 12% having myopia of any degree and approximately 2% greater than -1.00D.
Grosvenor and Flom, comparing similar studies, has made some comparisons to elicit general statements concerning changes that are occurring during the first 6 years of life.

1) Most children's eyes shift toward less hyperopia by approximately 1.00D.

2) The refractive error frequency curves become much more compacted thus reducing the standard deviation. Many myopes and most of the higher hyperopes disappear altogether. These changes signify the emmetropization process.

3) The leptokurtosis of the distribution curve at birth is highly exaggerated by 6 to 8 years old.

Some generalizations from an overall look at combined studies might be made. It is obvious that refractive error changes are taking place throughout the population prior to school age. From a refractive error of greater than 2.00D of hyperopia, a direction of less plus is traveled until about +1.00D is reached upon entering school. This general trend of emmetropization is accompanied by decreased amounts of myopia also. This tightening of the refractive curve shows that by 1 year of age there is much less variability around the mean. Thus, emmetropization is a major occurrence even in the eyes of infants.

Refractive stability goes hand in hand with this emmetropization. The extreme refractive errors that remained will continue to do so. There will be little change in the high hyperopes if they haven't changed by 7 years old. Only those hyperopes who show greater amounts of hyperopia under cycloplegia than without it, will likely decrease. Similarly, the myope who has milder amounts of nearsightedness under cycloplegia than without, will decrease. As long as it is uncomplicated, congenital myopia, more often than not, remains stable. If there are changes, they will be towards greater myopia. The tables below, adapted from Rosenbloom and Morgan, show yearly averages for the first years of life. Table 9 gives the mean in spherical equivalent refraction that is in place between the first and tenth years of life. Whereas Table 10 shows the prevalence of each dioptric value given in percentages for the ages 3 to 7 years old.
TABLE 9
ROSENBLUM AND MORGAN22
CHANGES IN THE MEAN SPHERICAL EQUIVALENT REFRACTION (SER)
FROM 1 TO 10 YEARS

<table>
<thead>
<tr>
<th>AGE</th>
<th>REFRACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+1.45</td>
</tr>
<tr>
<td>2</td>
<td>+1.33</td>
</tr>
<tr>
<td>3</td>
<td>+1.13</td>
</tr>
<tr>
<td>4</td>
<td>+1.41</td>
</tr>
<tr>
<td>5</td>
<td>+1.41</td>
</tr>
<tr>
<td>6</td>
<td>+1.20</td>
</tr>
<tr>
<td>7</td>
<td>+0.80</td>
</tr>
<tr>
<td>8</td>
<td>+0.54</td>
</tr>
<tr>
<td>9</td>
<td>+0.44</td>
</tr>
<tr>
<td>10</td>
<td>+0.08</td>
</tr>
</tbody>
</table>

TABLE 10
ROSENBLUM AND MORGAN22
FREQUENCY OF 1 DIOPTER SER INTERVALS
FROM AGES 3 TO 7 YEARS

<table>
<thead>
<tr>
<th>AGE (BY %)</th>
<th>DIOPTERS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3</td>
</tr>
<tr>
<td>≥-3.00</td>
<td>0.0</td>
</tr>
<tr>
<td>-2.00</td>
<td>0.0</td>
</tr>
<tr>
<td>-1.00</td>
<td>3.1</td>
</tr>
<tr>
<td>0.00</td>
<td>3.0</td>
</tr>
<tr>
<td>+1.00</td>
<td>37.5</td>
</tr>
<tr>
<td>+2.00</td>
<td>37.8</td>
</tr>
<tr>
<td>+3.00</td>
<td>9.7</td>
</tr>
<tr>
<td>+4.00</td>
<td>5.6</td>
</tr>
<tr>
<td>+5.00</td>
<td>1.4</td>
</tr>
<tr>
<td>+6.00</td>
<td>1.0</td>
</tr>
<tr>
<td>+7.00</td>
<td>0.7</td>
</tr>
<tr>
<td>+8.00</td>
<td>0.0</td>
</tr>
<tr>
<td>+9.00</td>
<td>0.0</td>
</tr>
<tr>
<td>MEAN (D)</td>
<td>+1.6</td>
</tr>
</tbody>
</table>

Summary
By comparing studies, it is shown that the ages between birth and 6 years, is a time of refractive stabilization and a move towards lower refractive errors. This can be seen by comparisons of refractive error curves of two different groups. A distribution of the refractive error at 3 years old is nearly identical to the same distributions at 7 years old, suggesting that most refractive error changes will be made by age 3. Furthering this idea is the fact that the spherical equivalent refraction varies little between birth and 3 years of age. Most of the changes that take place are within certain subgroups. The likelihood that the near emmetropic eyes of a healthy child at birth will develop any large amount of ametropia is very low.

KIRK T. REED
In 1952, Hirsch \(^23\) published a study in which the prevalence of refractive errors of 9552 school children in the Los Angeles area were described. In summarizing his findings he differentiates between boys and girls. From 5 years old to 14 years old, the mean refractive state of the girls decreased from +0.90D to +0.40D. The boys in the same age group do likewise from +0.75D to +0.30D. The medians had a similar decrease, but to a lesser extent. Notwithstanding the differences in the two groups, the smaller decrease of the medians indicates that a few cases may be swaying the mean.

The age of 10 seems to be a turning point. The distribution of refractions is fairly symmetrical then. A skewing to the hyperopic side is evident before 10 years old. After 10, the distribution is myopically skewed.

Less than 1% of 5-6 year old students have myopia in excess of -1.00D. This percent increases in the 15 year olds who show greater than 5% with that amount.

Hyperopia will decrease with age, but not as much as the prevalence of myopia will increase. Hyperopia increases until 6 years old and then begins to decrease.

At the time of Hirsch (1952), there was still the need for longitudinal studies of final refractive error probabilities based on refraction at age five.

### Table 11

<table>
<thead>
<tr>
<th>AGE/SEX</th>
<th>OVER 3D</th>
<th>2-3D</th>
<th>1-2D</th>
<th>0-1D</th>
<th>0-1D</th>
<th>1-2D</th>
<th>2-3D</th>
<th>OVER 3D</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BOYS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6</td>
<td>0.15</td>
<td>0.18</td>
<td>0.34</td>
<td>6.76</td>
<td>77.43</td>
<td>13.14</td>
<td>0.86</td>
<td>1.14</td>
</tr>
<tr>
<td>7-8</td>
<td>0.16</td>
<td>0.33</td>
<td>0.41</td>
<td>10.12</td>
<td>77.14</td>
<td>9.06</td>
<td>1.15</td>
<td>1.63</td>
</tr>
<tr>
<td>9-10</td>
<td>0.31</td>
<td>0.69</td>
<td>0.62</td>
<td>13.86</td>
<td>75.66</td>
<td>5.83</td>
<td>1.55</td>
<td>1.28</td>
</tr>
<tr>
<td>11-12</td>
<td>0.26</td>
<td>0.66</td>
<td>2.16</td>
<td>17.66</td>
<td>75.00</td>
<td>4.72</td>
<td>0.41</td>
<td>1.13</td>
</tr>
<tr>
<td>13-14</td>
<td>1.37</td>
<td>0.63</td>
<td>2.98</td>
<td>17.45</td>
<td>71.01</td>
<td>4.81</td>
<td>0.65</td>
<td>1.00</td>
</tr>
<tr>
<td><strong>GIRLS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6</td>
<td>0.00</td>
<td>0.20</td>
<td>0.25</td>
<td>5.70</td>
<td>72.62</td>
<td>16.61</td>
<td>3.18</td>
<td>1.44</td>
</tr>
<tr>
<td>7-8</td>
<td>0.23</td>
<td>0.22</td>
<td>0.53</td>
<td>8.73</td>
<td>71.22</td>
<td>14.70</td>
<td>2.50</td>
<td>1.87</td>
</tr>
<tr>
<td>9-10</td>
<td>0.25</td>
<td>0.48</td>
<td>1.27</td>
<td>15.17</td>
<td>67.97</td>
<td>11.67</td>
<td>1.66</td>
<td>1.95</td>
</tr>
<tr>
<td>11-12</td>
<td>1.35</td>
<td>1.72</td>
<td>2.70</td>
<td>15.83</td>
<td>66.25</td>
<td>9.84</td>
<td>1.33</td>
<td>0.98</td>
</tr>
<tr>
<td>13-14</td>
<td>1.61</td>
<td>1.92</td>
<td>2.25</td>
<td>19.58</td>
<td>66.13</td>
<td>5.94</td>
<td>1.77</td>
<td>0.80</td>
</tr>
</tbody>
</table>

KIRK T. REED
Much of what we hold to be true today about the progression of myopic refractions was stated by Hirsch in 1964. Realizing that although the refractive state for many groups of children have been previously established, a need to conduct a longitudinal study still existed. The only way one can be sure of the changes that an individual goes through, is to conduct longitudinal studies. These are very difficult for many reasons. The main problem is obtaining a stable enough population that the subjects will be available for the time period requested. Another problem is being able to use the same examiner, thereby ruling out errors that might be inserted due to varying methods and abilities. A final and almost insurmountable obstacle is gaining the cooperation of parents, subjects, school boards and school personnel. Being in private practice, Hirsch was able to organize his time and produce such a study.

Prior data showed that the average refraction of a child moves systematically toward more minus, yet a previous study of a large group of Los Angeles children by Hirsch suggested that each individual child's refractive error changes little between the ages of 5 and 14 years. Hirsch set out to follow a number of school children, a target of 500, in Ojai, CA from kindergarten through 12th grade.

The study was able to follow 383 children, getting semi-annual examinations beginning at age 5 of 6, and continuing for the next 8 years. He reached a number of conclusions.

1) Among 13 and 14 year old children, there is a significant relationship between the spherical refraction and astigmatism. A greater than expected number of myopic eyes had against-the-rule astigmia. Emmetropic eyes had more than expected amounts of spherical refractions. Seemingly, if against-the-rule astigmia is present, you can expect a higher probability of myopia.

2) There is some relationship between the initial astigmia and the ultimate spherical refraction. If against-the-rule astigmia is present at first grade in excess of -0.12D, there is a tendency to develop myopia later in life.

3) A significant relationship exists between spherical refraction at 5 years old and one at 14 years old. While the belief that the initial refraction
will be the rule throughout life, there is a noteworthy exception for those who have a lesser degree of hyperopia, of +0.50D and below. Myopia seemingly is expressed in greatest numbers by 9 of 10 years old, making it appear as a phenomenon of adolescence. Yet, those being emmetropic or less than +0.50D hyperopic often end up myopic. It seems, at least for this group, that their inevitable refractive state is laid out by 5 years of age.

One way to study refractive errors is to look at correlations of one individual's prescription when it has stabilized and compare it to prior years. A correlation of near 1.00 would be expected when comparing the final refractive error to just the previous year. As you progress to correlating each succeeding earlier year, you would expect a lower correlation. Hirsch has shown that the correlation between 5 and 14 years old is actually quite high, indicating that most of the refractive changes have already taken place. Much of the growth that an eye will do, happens by 3 years old. Therefore, any large refractive errors will occur by then also. The direction of refractive error and to some extent the amount that the patient will finish is determined by 5 years old.

Although Hirsch's study has given us valuable information, these simplified facts limit its value, lending more emphasis to the need of a longitudinal study starting at birth.

A table showing the ultimate refraction at the conclusion of Hirsch's study compare to those found as the testing was initiated is enlightening.

| TABLE 12 | 
|---------------|-----------------|-------------------|
| **ULTIMATE REFRACTION (AGE 13-14)** Compared to Initial Refraction | **AGE 13 OR 14** | **HYPEROPIA** |
| **MYOPIA** | **EMMETROPIA** | **HYPEROPIA** |
| >0.26D | 4 | 0 |
| -0.25 TO -0.10D | 6 | 0 |
| 0.00 TO +0.24D | 7 | 0 |
| +0.25 TO +0.49D | 37 | 4 |
| +0.50 TO +0.74D | 21 | 33 |
| +0.75 TO +0.99D | 15 | 41 |
| +1.00 TO +1.24D | 2 | 15 |
| +1.25 TO +1.49D | 0 | 1 |
| >+1.50D | 0 | 1 |
| TOTALS | 92 | 100 |
| **MYOPIA** | **EMMETROPIA** | **HYPEROPIA** |
| >+1.50D | 0 | 69 |

Throughout the school years the same wisdom holds true, that those with higher amounts of hyperopia will remain hyperopic. Emmetropes stay in the moderate range, and myopes stay in the negative refractive range. The rates of
change for myopia, being greater than those of hyperopes, have been noted by various investigators. Rates of change have been found to be linear in 93% of refractive errors, myopes included.

Once through the school years, changing refractive errors seem to slow down. The average refraction is +0.50D at age 20 and +0.12D by age 29. The trouble is that many are continuing school longer than high school. It has often been cited that college students have increased in their myopia while earning their degree. One of every eighteen graduating cadets from West Point left with myopia, while entering with hyperopia or emmetropia. Sixty-three percent who had myopia at the start, increased while there. Other data show that college freshman of 17 or 18 year old develop low grades of myopia by the time they graduated at 22-23 years old. These amounts seldom exceed 1.00D. It can be said that myopia does not increase as rapidly between 18 to 25 as it did during the previous years. These changes generally continue in the same direction, but increases are of less magnitudes.

Septon, reviewing literature, noted that new myopes do not arrive equally throughout all ages, but rather are grouped into clusters. A study of 447 second year optometry students were surveyed to test this hypothesis. The optometry students possessed 74.3% myopia, in which 88% of them were in excess of 1.00D. The ages at which these myopes first presented for care were clustered into 3 groups, 8-9 years, 12-13 years, and 19 years old.

After the age of 25, the refractive state is characteristically stable, as is one’s myopia. An estimation of between 8 and 14% myopia in excess of 1.00D is given for American adults. This climbs to 15-20% myopia greater than -0.50D. Any myopia at all in American populations is estimated to be at 25-30%.

Grosvenor, noting that data for young adults is not easily available, draws from varying studies to gives estimates. Extracting information from the US Department of Health, Education and Welfare (US HEW) he states that approximately 34% of those 18-24 year olds have myopia. This decreases slightly to 33% by 25 to 34 years of age and even more to 31% by 35 to 44 years old. Another estimate is given to be 40% of those between 26 and 35 years old and 32% between 36 and 45 years of age. In summary, he concludes that amounts of myopia equal to or in excess of -0.50D peaks approximately at 30% between the ages 20 and 40 years old.
The next age group, those greater than 45 years old, nearly all need some help with their vision. Since most of those greater than 45 years will need some assistance either up close or at a distance, this data can be considered more representative of the entire population. Data taken from the US HEW shows that myopia beyond 40-45 year old is 32% and decreases to nearly half of that, 18%, by 55-64 years old and again drops to 16% by 65 to 74 years old. Fledelius\textsuperscript{24} shows similar results, where the prevalence of myopia of -0.25D of more is 26% at 46-55 years of age and decreases to 14% past 66 years of age.

\begin{table}
\centering
\caption{Prevalence of Myopia during the Late Adult Years from Grosvendor\textsuperscript{6}}
\begin{tabular}{|l|l|l|l|l|}
\hline
\textbf{Ages} & \textbf{Source} & \textbf{Subjects} & \textbf{Criterion} & \textbf{Prevalence} \\
\hline
45-49 & HIRSCH & OPTOM PNTS & >-1.13D & 7\% \\
40-50 & BORISH & JACKSON AND TASSMAN DATA & >0.50D & 14 \% \\
45-54 & US HEW & NPS & Wearing Correction >0.25D & 32 \%
\hline
46-55 & FLEDELIUS & HOSP PNTS & Wearing Correction >0.25D & 26 \\
55-64 & US HEW & NPS & Wearing Correction >0.25D & 18 \\
56-65 & FLEDELIUS & HOSP PNTS & Wearing Correction >0.25D & 26 \\
65-74 & US HEW & NPS & Wearing Correction >0.25D & 16 \\
>66 & FLEDELIUS & HOSP PNTS & >-0.25D & 14 \\
>70 & BORISH & JACKSON AND TASSMAN DATA & >0.50D & 21 \\
>75 & HIRSCH & CPT PNTS & >-1.13D & 15 \\
\hline
\end{tabular}
\end{table}

KIRK T. REED
As has been done in the past, averaging refractive errors across many ages has suggested that the mean refractive error becomes less hyperopic over time and some eventually will even become myopic. When looking at refractive error distribution curves, it becomes evident that they are skewed myopically, suggesting that a few individuals progress into nearsightedness while the majority remain farsighted. Baldwin's study brought out an interesting point. He compiled data from case records of 78 patients who were between the ages of 6 and 20 years old at their initial examination. He analyzed the trends of serial examinations of each patient and drew some interesting conclusions. Myopes become more myopic through time, but likewise, hyperopes become more hyperopic. In following the records he found that the direction of the refractive error will most often continue along the path it originally started. This goes contrary to the popular belief that the general direction that most travel is in greater minus. This does not contradict the theory that if one is hovering in a very low hyperopic status that they may cross the emmetropic line into myopia and from there continue in a myopic direction. Those who do this seem to be relatively few in numbers, and do so at the early ages of myopic progression such as prior to 14 years old. A possible reason that they are so few is that any low hyperopic patient which would decrease in hyperopia, would not likely present themselves due to any complaints. In fact a diminishing of asthenopic complaints would be more likely. If this in indeed is the fact, further investigation is needed to follow the hyperopic decreases. Baldwin's final conclusions were: 1) 75% of myopes showed change, while only 45% of hyperopes showed change, 2) hyperopia is more stable than myopia, and 3) direction of change is highly predictable and that direction would act to increase the amount of the original refractive error.

In his paper, Changes in Refraction During Life, Bucklers graphs out spherical refractive changes in 110 eyes for 2 to 3 decades. The results are an interesting graphical representation of refractive changes with easy to read generalizations.

1) Myopia increases during the first 20 years and then progression flattens out.
PREVALENCE AND EXPECTATIONS

2) Myopes that start out earlier and with higher amounts have a tendency towards quick and steady increase.
3) There are stationary periods where both low and high amounts of myopia will cease to progress.
4) There seems to be another sudden increase, at least for some, during the 5th and 6th decades of life.
5) Generally myopia will continue to increase during growth years and become stationary as one's height levels off.

The linearity of refractive changes were studied by Goss by considering the records of six optometric practices. Nearsighted patients between the age of 6 and 15 were used. He found that for 90-94% of these youngsters myopia progression was linear. Once the child exhibited a myopic refraction, it continued to increase until the mid to late teens. The age of stabilization (the cessation age) for those who were considered having childhood myopia averaged 15 1/4 for females and 16 2/3 for males. Young adult myopia seems to progress into the late teens or early 20's.

Being linear, the amount the myopia progresses during one time period will be the same that it progresses during any equally succeeding time period. This will continue at the constant rate until the myopia stops. These rates tended to be from 0.25 to 0.50D per year, although some females increase at a rate of .75D per year.

Knowing the rate of progression and the most likely age of cessation, one can extrapolate the final degree of myopia. One must take into account the time one entered into myopia, since myopic changes are at a much higher rate than those for emmetropes and hyperopes and much greater for those starting with early and higher amounts.

Mantyjarvi discovered the mean rates of negative changes while still hyperopic is 0.12 to 0.21D per year, but when the switch to myopia occurs this rate increases to 0.55 to 0.60D per year. The same author, writing on the possibility of predicting myopia progression wrote of the difficulty in trying to predict an individual case of progression, due to tremendous individual variation. Most of this due to hereditary factors. We know that by the age of 14, most myopic changes have occurred, yet small changes may continue until 20 years old. As for the final degree we may say that if myopia starts before puberty, it will continue to between -3.00 and -5.75D. If the age of onset is after puberty, the total amount may be under -3.00D.

KIRK T. REED
The fact that each myopic person has a great deal of variability is agreed with by Goss and Winkler. It has been determined that females stop earlier than males, yet even within the sexes there is variability. The lower cessation ages for females, according to their study, is between 14.44 to 15.28 years, while males cease from 15.01 to 16.66 years. The fact that females stabilize earlier suggests a link between myopic progression and body growth. General body growth stops earlier for females.

They also noted that the rate of increase of myopia and the age at cessation were highly correlated. Higher rates seemed to stop earlier. A slower rate which continues to progress may eventually end up with a similar degree of myopia to that of a faster progressing that stopped earlier. The differences in cessation ages discussed here may be related to the underlying nature of myopic progression and cessation.

A comprehensive review of progression literature has been developed. Before age 7, most studies show that the average refractive status ranges between +1.00 and +2.00D. Before age 7, the mean refractions begin to shift myopically and continue in this direction until the mid to late teen. At this level little changes occur throughout life until the age that presbyopia sets in. Now there is a slight shift back towards hyperopia. Beyond the age of 65, the nuclear sclerotic changes along with other changes, accounts for a slight myopic shift again. More generally stated, once myopia develops, it would be rare for it to decrease prior to the onset of presbyopia. Presbyopia may signal small magnitude changes away from myopia.

A few longitudinal studies confirm the hypothesis that the earlier the myopic onset, the higher the magnitude is when stabilization occurs. Other predictors of myopia might be found in etiological factors such as socioeconomic, cultural, vocational, ethnic or personality differences. Some variability of these factors have been found among those who are myopic, and those who are not. Hereditary versus environmental factors have long been discussed as to which is the overwhelming reason for myopia and its progression. Other factors such as esophoria and against-the-rule astigmatism has been implicated by some, as increasing the chances of progression.

There are many methods that have been attempted to slow the myopic progression once it has begun. Some techniques are used to stop it before it starts. The general consensus is that none of them consistently work with all people. These methods range from not wearing spectacles, to part time
or full time wear, from undercorrection to overcorrection of minus, the use of
contact lenses, vision therapy, and bifocals, from manipulation of diet to the use
of cycloplegics and miscellaneous drug therapies and so forth. Goss31, in
review, concludes none of them in and of themselves, will consistently reduce
myopic increases.

Summary

We have seen that myopia, most likely, has many components
contributing to its etiologies. There are those whose refractive error is obviously
most predominantly derived from one etiological factor rather than another. A
congenital myope who having a substantial amount of myopia that he carries
this ametropia through emmetropization on into adulthood, is most probably
myopic by genetic determinants. Knowing that the trends point to higher myopic
refractive errors if started earlier, we can guess the myopia to be in excess
-5.00D or -6.00D by cessation.

To give one estimate we might postulate on a -0.50D myope who had
this amount from birth and didn't escape through emmetropization. If he made it
past some initial progression increases and didn't start myopic progression until
the age of 8, then progressed at what is thought to be an average yearly
progression of approximately -0.50D per year, by the time he leveled off near
age 16 he would have increased another -4.00D. The variations to these sort of
estimations are limitless. As so often stated by many researchers, individual
variations are great, and the need for large longitudinal studies on the age
group in question remains.

To answer the question of a concerned parent about how nearsighted
their baby will become before myopic increases stop in not a simple task.
Knowing that although nearly one-fourth of all newborns show some degree of
myopia, this in and of itself does not mean their baby will be myopic since most
all of the 25% emmetropize to little or no refractive error. The finding of myopia
at an initial exam would be little more than a prevalence if it were not for family
history. The individuality of this baby may be to decrease in myopia and
become emmetropic.

Until we have more sophisticated methods of determining the exact type
of myopia that an individual embraces, a few basic generalizations may be
helpful. Once the original finding is made and the question of the final amount

KIRK T. REED

33
is asked, the doctor would need to inquire into family history of myopia. A parent with a high degree of myopia could imply a similar magnitude in the baby. Additionally, the baby should be followed for a few years to determine which direction the refractive error is headed. If it is indeed myopic, a few generalizations can be used to estimate magnitude at cessation; 1) direction traveled is in the direction of the original refractive error, 2) generally quick and steady increases are had by those starting at birth with high amounts of myopia, 3) myopia will increase linearly at approximately -0.50D per year until the child's growth stops, and 4) a congenital myope may likely progress to at least -5.00D.

We know the likelihood for high myopic values is probable in the congenital myope, yet there are still some gaps that need to be filled in. The question of how myopic a congenital myope will be is not answerable until we have longitudinal data to support an answer. As of the present, we can just educate ourselves to the facts, postulate on prevalence and progression studies, and make educated guesses.
Bibliography
