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Abstract

A current review of the proposed causes and controls concerning the management of functional myopia is discussed. Nutritional-Disease, Mechanical-Anatomical, Environmental, and Genetic theories are reviewed. Topics concerning the controls of myopia include orthokeratology, vision training, surgery, pharmaceuticals and bifocals. A macroscopic theory of myopia development is presented and the merits of the various methods of control are evaluated.

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A CURRENT REVIEW OF CAUSATION AND MANAGEMENT OF FUNCTIONAL MYOPIA

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ABSTRACT: A current review of the proposed causes and controls concerning the management of functional myopia is discussed. Nutritional-Disease, Mechanical-Anatomical, Environmental, and Genetic theories are reviewed. Topics concerning the controls of myopia include orthokeratology, vision training, surgery, pharmaceuticals and bifocals. A macroscopic theory of myopia development is presented and the merits of the various methods of control are evaluated.

History of Myopia Causes

The word myopia comes from the Greek "myo" meaning to wink or half close the eyes and "ops" meaning eye. The term was introduced as a result of the habit which myopes frequently have of half closing the lids or squinting at distant objects. Since the time of Aristotle, three hundred years before Christ, men have speculated as to the cause of this condition of the eye. In a summary of the historical progression of myopia causes Goldschmidt (1968) states that Galen as early as the first century A.D. would influence opinion throughout the whole of the Middle Ages and the Renaissance with his theory that the composition and consistency of the fluids of the eye caused near sightedness. Hundreds of years later, in 1604, Kepler was the first to draw attention to the fact that those who used their eyes for extended periods of reading and writing became myopic. It was not until two hundred years later that a great interest in this subject would be rekindled by Donders, who believed that myopia was acquired as a result of close work, but that the acquired characteristic was then transmitted to descendents. He described three factors as being of particular importance. The first of these was the pressure of the muscles on the eye during convergence. Secondly, an increased pressure in the ocular fluids caused by blood stasis when a person adopts a stooping position and thirdly, fundus changes leading to a softening and extension of the coats of the eye. Not long after Donders theories in the mid 1800's Cohn, after examining several thousand schoolchildren concluded that the number of myopes as well as the degree increased with age. His investigation indicated that myopia first began after several years in school and was more frequent in upper grades. Several years later Tscherning (1900) examined 7,000 Danish conscipts and found 32%, who had been students

previous to induction, were myopic while only 5% of those who had been employed as laborers and farmers were. This led him to conclude that near work was responsible for the condition.

It was not until 1913 that any serious objections were made to the basic assumption that close work in some way causes myopia. In that year Steiger found that corneal power varied considerably in persons with the same refraction therefore he believed a compensating variation in the remaining components, and primarily the axial length, must exist. He concluded that the components of refraction associate freely and vary in accordance with a normal distribution. This being the case he theorized that myopia is genetically determined and a second school of thought was born. Studies by Tron in 1929 later showed that all the optical components of the eye did follow a normal distrobution except for the axial length which is skewed on the high end.

Goldschmidt (1968) cites other theories which have been proposed in addition to the previous. He states that Weiss in 1885 asserted that myopia was caused because the optic nerves were too short and pulled at the rear of the eyeball. Graffe in 1854 claimed that gazing at an object caused hyperemia and inflammation in the macular region and thereby caused an abnormal growth and elongation of the eye. Levinsohn who in 1914 was the first in this field to experiment with monkeys, found they became myopic when hung upside down for "a long time" and concluded the ocular axis was elongated because the eyeball was pulled up by the optic nerves. These are just a few of the thoughts and theories that have evolved and been dispelled. Those that have been substantiated and built on are primarily the Genetic-Biological theory and the Environmental-Use/Abuse theory. These will be reviewed in detail as will others.

Classification of Myopia

Before we begin our discussion on the various causes of myopia we must set some limiting factors on classification. It would be much too large a project to attempt if this were not done therefore, the boundaries at which we wish to stop must be as defined as possible.

Firstly, Nocturnal myopia will not be considered in this paper. We feel it may be omitted since it is sufficiently different to warrant it's own study. Secondly, Transitory myopia will be omitted. Borish (1975) defines this as a myopia induced in an eye which is otherwise fixed in it's refractive status by a number of conditions such as changes in structure, position or index of one of the media, disease, drugs, or trauma. Therefore, myopia considered to be caused by such things as diabetes, keratoconus, senile nuclear cataracts, morphine, or lens subluxation, etc. will be left for further reviews. The third limitation is the most difficult because there is no clearly drawn borderline which separates Pathological or Congenital myopia from the more innocuous form. One particular type may be ruled out at the outset and that is myopia of premature birth. Originally thought to be found only in infants with retrolental fibroplasia Fletcher and Brandon (1955) have shown it occurs regularily in all premature infants and will therefore be deleted. The other forms of Pathological myopia with causes due to unknown factors will at times be mentioned inadvertently.

Kuhn (1962) outlines six separate classification systems which may be used. Such a complex design prevents everyday use since the differences between systems are subtle and often overlap. However, a few are worth mention. He believes that a system based on degree of the refractive error would contain four groups: Low (0-3.00D), Medium (3.00-6.00),

High (6.00-10.00) and Very High (over 10,00). With this system the age of the individual would also have to be considered, as "low" myopia in an adult would be "high" in the case of an infant. A second system classifies myopia as being stationary or progressive. Since all myopes are at one time or another progressing the age at which this stops as well as the degree of myopia achieved must be considered. We will review this progression in greater detail further on. Classification based on accommodative activity is possibly of the greatest importance. Pseudo, Functional, Accommodative, or Schoolroom myopia all apply to a temporary or permanent convexing of the crystalline lens due to a spastic condition of the ciliary muscle. It is this type which Young (1977) believes is the initial stage of True myopia. In this first event accommodation can no longer be relaxed completely and a level of .50-1.50 D myopia is maintained. If this is maintained long enough he contends, there is an increase in the size of the vitreous chamber which results in the True, Organic, or Structural classification.

Tscherning (1883) believed that two forms of axial myopia existed. The first he called myopia from near work and its characteristics were as follows: Appears first at age 6 to 15, does not progress past age 25, attains medium degrees rarely exceeding 9.00 D, and has few complications. The second type he called dangerous myopia. The attributes of this classification are: Developes early in infancy, continues to increase throughout life, generally exceeds 9.00 D by age 20 and is more prevalent among women. He considered this type to be a malignant choroiditis with dangerous complications such as retinal detachment. If these two forms of myopia, which Tscherning describes, are considered to be the two major classifications, it is the first of the two we wish to consider in this paper.

Developmental Progression

Before we begin reviewing the causes of myopia we should first look at the pattern in which it developes. This is the foundation on which the theories are built. Cook and Glasscock (1951) found that myopia does occur at birth but this is not the usual case. The average refraction was +2.00 D ±2.75. 75% of newborns are hyperopic with half of these being between +.25-+3.00 D. The extreme end of this range was +12.00 D however, low amounts were much more common as 88% of the total population fell between -.25to +5.00 D. By age 6 Hirsch(1963) has shown that the mean refraction is approximately +1.00 D less hyperopic and that the variability seen at birth has decreased greatly. Sorsby (1974) suggests a process of emmetropization stating that at birth the sagital diameter of the eye is about 18mm. This has elongated to 23mm by age 3 and at this point is very nearly its adult size. This growth would produce a myopic drift of 15.00 D if compensatory changes did not occur. Between the ages of 3 and 14 years axial growth is slight, averaging about .1mm per year. This represents a change in refraction of 3.00 D toward myopia during these eleven years. The growth of the eye is evenly distributed during these years of childhood and there is nothing to suggest a spurt at puberty. During this time also the cornea and lens become flatter to compensate for this elongation. The lens power alone declines 2.00 D over the period between 3 and 14 years of age. Hirsch(1964) summarized the trend of developement as follows: If a child at age 6 has a refractive error between +.50 and +1.25, he has a greater chance of becoming emmetropic. If he has a spherical refraction from 0 to +.50 there is a high probability he will be myopic. A child who is myopic at age 5 to 6 will remain myopic and will probably increase.

The younger the myopia appears, the greater the amount will be before stabilization occurs. The curves generally level off at around age 13 and by age 16 most children have their adult refraction. After the age of 20 Morgan(1958) has found little change in refraction. Females ages 20to 40 changed .22 D toward myopia over the 20 year period and males changed as little as .04 D in the same direction during this time.

In summarizing this section on the developemental progression of myopia we feel it necessary to relate several additional points Young(1975) outlines. These are that girls tend to develope a higher amount of myopia than boys, develope it earlier, and more girls are myopic than boys at an early age. Secondly, the earlier a child developes myopia, the greater the total amount will be and thirdly that the age at which myopia is developing has been decreasing steadily over the years. These points as well as others presented later in this review will help us to evaluate the different theories on causes of myopia.

Frequency and Degree

The final consideration is the frequency and degree of myopia. We have already noted that myopia occurs in about 25% of all newborns but by the age of 6 years this percentage shows a marked decrease. From this age on many studies have been done. We will take a sampling of these in order to provide a general background.

There are several different dependent variables which frequency and degree of myopia have been paired with. The first of these is occupation. Tscherning(1883) studying Danish conscripts, as previously cited, noted that students, office workers, artists, and tailors had a much higher percentage of myopia than did hard laborers and farmers. He also mentions that the degree of myopia is lower in the first group, stating that the

cases of myopia over 9.00 D are found more frequently among peasants. Goldschmidt(1968) compares Tschernings results with a similar study he conducted in 1964 and found that the frequency of high myopia (over 6.50) has fallen from 1.7% (Tscherning's data) to 0.6%. He also notes there has been an increase in the number of cases of lower myopia during this time.

Next we can compare amount of myopia and education. If we look at the differences between grade levels we notice an increase in myopia accompanying higher levels. Sato(1957) states that 70% of university students in Japan are myopic as compared to 45% of middle school pupils. We can also compare children of the same age but enrolled in different types of schools. Goldschmidt(1968) found that 9.2% of Danish municipal school students were myopic compared to 15.4% of those attending private schools. He also compared "A" grade students with students recieving lower marks and found a greater percentage of these students were myopic. 73% of those myopic in this study had under 3.00 D of refractive error.

We must also consider racial differences. Grosvenor(1977) summarizes these racial variations in refraction showing considerable differences do exist. In comparing Blacks to Caucasians he finds that 13% of the whites are myopic as compared to only 8% of the Blacks. In another study comparison, 52% of the Chinese tested were myopic while 20% of the Caucasians were. Borish(1975) summarizes that approximately 20% of all Americans in the U.S., 27% of the British, and 13% of the French are myopic. Beaulaurier and Hillier(1981) report myopia to be a rare condition in the Honduras with hyperopia being much more prevalent.

Rasmussen(1936) looked at this question in a different way. He found that of 120,000 pairs of glasses prescribed in England, 70% of them were myopic corrections the average degree being approximately 3.00 D.

As we see, myopia has been looked at in many different ways. The frequency and degree depend on many different variables and the interaction between these. There are no clearly defined boundaries but rather patterns and trends with which we must deal.

Nutritional Theory of Myopia

The nutritional theory of the cause of myopia has as its major tenet the supposition that nutrients lacking in the diet cause structural changes in the coats of the eyeball. These changes cause weakening and stretching of the coats and thus the axial length increases causing myopia. If we define the term "coats" here we can mean it to refer to either the sclera or the choroid or perhaps both tissues.

Bell(1978) states that the sclera is composed of two layers of connective tissue. The first of these is collagen which makes up approximately 70% of the dry weight and the second is hyaluronic acid. With increasing age the collagen fibers form cross linkages and become more stable and also cause an increased tensile strength of the tissue. As a result the sclera looses elasticity with age. This can be used to explain why juvenile glaucoma causes a distension of the globe while adult glaucoma does not. It can also be used to explain why progressive myopia occurs before adulthood. He continues by saying that vitamin C plays an important role in the formation and maintenance of the collagen molecule. If the diet is lacking in this vitamin a collagen precursor forms having no fibrillar character. As support for this theory he summarizes a study done by Garzino(1956) who found that collagen fibers of highly myopic people usually had a smaller diameter than that of an emmetropic eye and also that they were surrounded by more fluid than is normally present.

Avitaminosis A and hypocalcemia have also been implicated as causes of myopia. Feldman(1950) conducted a study in which the vitamin A and calcium levels of myopic patients were monitored. Blood protein levels were also recorded. Included in his study were 20 hyperopes who acted as controls. His results showed that the protein levels in both groups were not significantly different. No conclusions could be drawn from vitamin A levels however, a significant difference in calcium levels existed between the two groups. 77% of the myopes had a subnormal differential calcium level as compared with 55% of the hyperopic control group. The differential calcium level referring to the percentage difference between ionic and bound blood calcium. In reviewing this study the number of subjects should be critically considered. There were 50 myopes and 20 hyperopes involved which means a difference of two or three subjects in the hyperopic group would have caused quite a large change in the percentage values. If only three more subjects in the hyperopic group had had low differential calcium levels the percentages between the two groups would have been equal. Feldman also reports of several clinical cases of myopia which he treated with large doses of vitamins and calcium over a period of one year. His results here are inconclusive with some cases showing a decrease in myopia while others progressed further.

Sato(1957) reviews an Acidosis theory which was popular in Japan during the 1940's. According to this theory a diet high in sugar and glucose causes acidosis of the body. If there is acidosis of the whole body, then local acidosis occurs much more readily and if this event occurs in the macular region, the sclera will be deprived of calcium and there will be a weakening of the scleral tissue thus causing the axial length of the eye to increase. This theory fell into disfavor after

further studies in Japan showed that serum calcium levels and the carbonic acid gas cohesive forces were the same for myopes as they were for normal subjects. Further studies found that the quantity of calcium in the human sclera was not lower in myopic subjects.

Lane(1979) summarizes his studies on nutrition and myopia by making several conclusions. The first of these is that when hair analysis was done, myopes showed dramatically lower chromium comcentrations than did hyperopes. He also found that the ratio of chromium depleting refined carbohydrates to total carbohydrates ingested was three times greater in the myopic group. Calcium levels were significantly elevated in 7 to 17 year old myopes but showed much less elevation for ages 18 to 35. His final observation was that progressing myopes showed higher ratios of protein intake compared to U.S. Recommended Daily Allowance than did those myopes who were not progressing. This second group of non-progressing myopes had ratios of 1.0 or lower. Lane concludes by saying that he believes this data shows that nutrition influences the bodies ability to maintain normal ranges of interocular pressure and also influences the distensibility and contractibility of the schlera.

Reviewing the literature on the nutritional causes of myopia will lead to several conclusions. The first of these is that there are conflicting reports from different researchers. Recent research, with the aid of sophisticated instrumentation, is now enabling researchers to do much more detailed studies. Perhaps with modern techniques and further research, firmer conclusions may be drawn and a nutritional link to myopia found but at present, this relatioship remains controversial.

Systemic Disease Theory of Myopia

When considering systemic disease as a causative agent of myopia there are actually two factors we must discuss. The first is maternal disease during pregnancy and the second, childhood disease. Both of these have been studied as possible causes of myopia.

As previously cited congenital myopia has been shown to be associated with premature birth. In dealing with maternal disease as a possible causative agent, this type of myopia must be factored out, since it is due to developemental events. Gardiner and Griffith(1960) have researched this area and offer the following as evidence in support of this theory. They found that in a comparison study 13% of the mothers which had toxemia during their pregnancies had children without myopia as compared to a 50% myopia rate in those mothers who had a toxemia. In this context the term toxemia includes hypertension, pre-eclampsia, and renal disease. They also found that 25% of the women, that delivered non-myopic children, had had an illness which seriously interfered with health during pregnancy compared to 75% of the women delivering myopic babies. Most of the myopia was greater than 4.00 D. They contend that since the myopia was, in almost every case, the only physical defect found in the newborns the myopia must develope late in the pregnancy. Furthermore, since the eyeball lengthens during the last three months of pregnancy, they believe that maternal disease during this time will retard the growth of the eye. These children are in fact born with "premature" myopic eyes although the rest of the body is fully developed. The myopic infants are of normal birth weight even though a loss of protein is common in toxemias.

We will now consider childhood disease as a causative agent. Hirsch(1957) has conducted a study on the relationship between measles and myopia. He found that children who have measles during their sixth,

seventh, or eighth year seem to be much more likely to develop myopia in excess of 1.00 D than children who have measles at other ages. 14% of the first group developed myopia greater than 1.00 D as compared to 2.8% of those children who developed measles at ages other than six, seven or eight years. Hirsch offers no explanation as to the mechanism by which this myopia developes.

A possible mechanism may however be drown from work done by Greene and Mahon(1979). They performed experiments with rabbit eyes and found an irreversible "scleral creep" or stretching which occurred when the temperature and intra-ocular pressure were increased. If we relate their findings to the fever accompanying the measles and perhaps prolonged bedrest, squinting, coughing, or eye rubbing which would cause an increase in the IOP., we might conclude that a plausible mechanism does exist. Accommodation and convergence would also act to increase pressures during the time of such illness. Perhaps this is the science behind the "old wives" practice of keeping children suffering from diptheria, measles, or chicken pox in a dark room to prevent near-sightedness. Wirts(1976).

Evidence does exist showing a relationship between systemic disease and myopia. Myopia as a result of maternal disease is generally of a higher degree than that caused by childhood disease. During periods of acute illness the sclera may show reduced tensile strength, and be vulnerable to stretching from intra-ocular pressure. These theories are not considered to be of major importance at this time because of further developements and research into other causes of myopia. Little recent work seems to have been done due to the difficulty which exists in experiments dealing with these variables.

Mechanical and Anatomical Theories of Myopia

Mechanical and anatomical aspects of myopia developement are often considered in association with other theories of myopia, such as the Use-Abuse theory. In order to take a closer look at the work which has been done in these areas we will look at them separately.

The refractive state is determined by more than a dozen different parameters. Total refractive state depends on the two corneal curvatures, several different indices of refraction, lens curvatures, anterior chamber depth, and axial length. All of these parameters differ more or less from individual to individual. Hirsch(1972) summarizes the work of several men who researched these variables and threrby laid the foundation for the Mechanical and Anatomical theories of myopia. In 1946 Stenstrom found that all elements making up the total refractive state were normally distributed except for axial length. He showed that there is some degree of correlation among these elements which acts to counter-balance each other or to "emmetropize" the resultant refraction. An example would be an eye with a longer axial length having a flatter cornea. He also determined that the axial length had twice the effect on refraction as the cornea or lens and that the anterior chamber depth had only a tenth as much effect as axial length. Axial lengths vary from approximately 20 to 30mm. Since each millimeter difference in range of axial lengths can produce a change in refraction of 3.00 D, we see a 30.00 D total refractive difference, which axial length influences. Front corneal surfaces vary from approximately 38.00 to 48.00 D. This element can exert a total of 10.00 D leverage on refraction. However, even though emmetropization occurs, myopia may be present if one of the elements varies

markedly from the mean value. For example, if the axial length exceeds 26 or 27mm myopia is the usual result. Sorsby(1974) refers to this same process by which the refractive components are correlated, as coordinated growth. He states that the mechanism producing this coordination is largely automatic and continues by defining two types of ametropia. The first of these is called the "correlation ametropia" in which all of the refractive compoments fall within the normal emmetropic distribution range. These range values he has determined from examining eyes with refractions from plano to +.50 D and are as follows. The axial length varied from 22.3 to 26.0mm, the power of the cornea from 39.0 to 47.6 D, and the lens from 15.5 D to 23.9 D. The respective means with their standard deviations were 24.4mm ±0.85, 43.1 D ±1.62, and 19.7 D ±1.62. In "correlation ametropia" the coordinated growth process has somehow been disrupted and even though the individual components fall within normal ranges the collective result is ametropia. This form of ametropia however, is of a low degree ranging from 6.00 D of hyperopia to 4.00 D of myopia. In refractive errors outside this +6.00 to -4.00 D range Sorsby found that with few exceptions the axial length was outside the emmetropic range. This form of refractive error he calls the "component ametropia" and in general the degree of ametropia is proportional to the anomaly in axial length.

We will now turn our attention to the mechanical forces which act on the sclera. There are three major considerations to be reviewed each of which has been implicated in causing an increase in myopia. They are extra-ocular muscle contraction, ciliary muscle contraction in accommodation, and an increase in intra-ocular pressure. These are all associated with an increase in axial length however, the mechanical aspects

as well as anatomical variation, found in all biological systems, contribute in differing ways to each of these three theories and in fact, it may be impossible to separate any one of these from the other two. These three mechanisms occur together during any near point activity. We will consider them separately here however in order to determine how each might contribute to the increase in axial length and thereby cause myopia.

We will first look at extra-ocular muscle contraction and its enfluence on the sclera. Bell(1978) has reviewed some of the early work which has been done in this area. He states that sustained contraction of the exta-ocular muscles exerts a mechanical squeezing of the globe, raising intra-ocular pressure and weakening the sclera. Bach-Y-Rita(1968) has shown that succinylcholine induced extra-ocular muscle co-contracture produces a shortening of the globe in both experimental cats and human subjects. This deformation of the globe would be expected to also cause a rise in intra-ocular pressure and scleral stress. However, the effects of this experiment are not typical of normal ocular movements. Extra-ocular muscle co-contracture can sometimes be elicited during tonometry but there is no evidence of it during normal human activities. Kuhn(1962) describes another way in which muscle contraction might influence axial length. During the act of convergence the medial recti are mainly activated while the lateral recti are inhibited. As the eyes revolve about the centers of rotation the medial recti are lifted away from the globe, while more of the body of the lateral recti makes contact with it. At the some time, the two oblique muscles increase their traction in order to prevent retraction of the globe back into the orbit. The point of insertion of the obliques is located in the region of the posterior pole, which is the area in

which the axial elongation generally takes place. Kuhn believes that a continuation of this process results in a pulling of the sclera at that point thereby causing a weakening and stretching of the scleral tissue. Greene(1980) has further researched this theory and also believes the oblique muscles are involved. He states that the attachment lines of both the superior and inferior obliques are at the back of the globe with the inferior being closer to the macular region. His studies have shown that these muscles exert local stresses which depend on several variables. These are the tension of the obliques, the width of the attachment line, and the thickness of the posterior sclera. The worst case would result with narrow muscle attachment lines 5mm in width or less, the superior and inferior oblique muscles attached very close together, an oblique tension of 40 grams per muscle, and a thin sclera 0.1mm thick. Under these circumstances, the tensile stress in the region of the macula and between the two obliques would be 80 grams/mm^2 and would result in scleral creep. We see here that a person might be anatomically predisposed to this increased axial length if the above conditions were approached.

Concerning the mechanical action of accommodation on the sclera, Bell(1978) believes that since the ora serrata moves forward about .05mm with each diopter of accommodation, stress is exerted on the choroid which in turn exerts stress on the sclera since they are attached. As support for this theory he cites the work of Gimbel who has shown that cycloplegic agents that completely eliminate the accommodative response have been shown to be effective in arresting myopia. Greene(1980) has shown that even though accommodation raises the intra-ocular pressure 2mm Hg or less it may still be a contributing factor in causing scleral stress.

The third mechanical force acting on the sclera is intra-ocular pressure. Collins et. al.(1967) have shown that I.O.P. can be increased by 14mm Hg in cats when the extra-ocular muscles are chemically stimulated. As previously noted, there is a slight increase inI.O.P. from accommodation. Coughing, squinting, rubbing of the eyes can also cause increased I.O.P. and possibly act as a mechanism which would cause myopia. Bell(1978) has summarized some of the work done in this area and concludes that I.O.P. increases of as little as 10mm Hg in rabbits will increase axial length by .05mm and also the fact that juvenile glaucoma sufferers frequently show an increased axial length. As further evidence, Deodati and has associates in 1975 found the average I.O.P. of those with myopia of -10.00 D or more to be 2.3mm Hg higher than emmetropic controls. Greene and Mahon(1979) have shown that permanent plastic deformation of the axial length of eyes studied in vitro can be caused by increased I.O.P. and further that this processed is enhanced by greater temperatures. They call this deformation scleral creep.

Not all investigators have concentrated their work on axial length and scleral creep as causes of myopia. Sato(1957) has done much work on the mechanical aspects of the lens and how it effects the refractive state. He believes that there is an adaptation of the crystalline lens due to prolonged accommodation. This begins with increased tonus of the ciliary muscle and with continued accommodation, hypertrophy of the muscle. With time an organic adaptation takes place and the lens is permanently altered. As proof of this theory Sato points out that hyperopes have lenses of greater refractive power than emmetropes and myopes due to the fact that they must accommodate to a greater degree.

In the initial stages of myopia greater accommodation causes organic steepening of the lens which causes a decreased accommodative demand. This decrease in accommodative demand does not occur in the hyperope and therefore the lens increases its refractive power to a greater degree than the myope. This theory does not explain the progression of myopia of smaller degrees, however and also the fact that a loss of hyperopia does not occur with continued accommodation into the middle years of life.

In concluding the mechanical and anatomical theories of myopia we see that correlation ametropia is responsible for myopia under 4.00 D. Above this value one or more components of refraction are atypical and we believe the result of pathology or genetic factors. Theories for increased axial lengths and organic lens adaptation can be used to explain myopia of lower degrees. Changes in axial length may be caused by increased intra-ocular pressure brought on by convergence or accommodation, to a lesser degree. Scleral stress results in scleral creep and may be caused by co-contracture of the obliques with convergence. Lenticular changes may result from prolonged ciliary tonus, although evidence for this is not as strong as that for axial changes, it may also be a contributor in causing myopia.

Environmental or Use-Abuse Theory of Myopia

The environmental theory of myopia is perhaps the best known and most widely researched of the theories we will discuss. Many Optometrists have regarded it as the most credible explanation for myopia development.

Kuhn(1962) describes myopia as the end result of an individual's adaptation to visual stress. The structure, or eye, adapts by an increase in axial length in order to reduce the near point stress placed upon it. Kuhn believes there are three recognizable stages. The first of these stages occurs before any real signs of myopia are present. The patient may show subtle signs on examination such as low plus acceptance, retinoscopy showing a slight minus correction, or esophoric tendancies at near even though distance vision remains unimpaired. In the second stage retinoscopy and subjective tests will manifest some low degree of myopia, usually less than -1.50 D. This usually occurs at nine to eleven years of age and distance vision is compromised. The third stage occurs after myopia has been present for some time and uncorrected distance vision permanently blurred. If sustained near point concentration is maintained by children and young adults two possible outcomes may occur. First, the person may develop myopia or second, an avoidance to the task may occur. Both of these will lower the students level of achievement. Let us now discuss some of the studies which have been done to show that near point stress is involved in the development of myopia.

Possibly the most important study which supports the environmental viewpoint was done by Francis Young(1969) on Eskimos in Alaska. This study came about after two Optometrists noticed that younger Eskimos who had been given formal education, tended to show a relatively high incidence of myopia while older family members did not. Young found a greater difference between the proportion of myopes vs. non-myopes occurring from age fourty-one and older compared to the proportion for ages under fourty years. Only two subjects out of 131 in the fourty-one and above age group showed myopia. This represents 1.5% of the sample group. The fourty and below group had 152 of 377 possible subjects

showing myopia or 44.7%. This far exceeds the amount of myopia usually seen in an American or European population of the same age group. The older group on the other hand, falls far short of the amount usually demonstrated by American or European populations and is more comparable to that found among African natives, Borish(1975). Young continues by explaining a possible cause for this as follows. During the winter the Eskimos live for long periods of time under relatively low levels of illumination. Most of their rooms are small and illuminated by a single 40 watt bulb in a ceiling fixture. This, he believes, provides a level of lighting which is low enough to induce a maximum level of accommodation in an individual attempting to read. He concludes however, by stating that the major difference between parents and their children is the greater amount of near work and the reading continuum they are presently subject to.

In another study linking education to myopia Angle and Wissmann(1978) analyzed data collected by the U.S. Public Health Service and U.S. Bureau of the Census. Three independent variables were analyzed using myopia as the dependent variable. These were 1. Age from birth, 2. Age from birth and age from puberty 3. Age from birth and highest level of education. Results showed a tendancy for myopia to progress with each month of age from age 12 to 17. This progression was on the average .008 D per month or .096 D per year. However, neither age from birth or age from puberty was statistically significant at the .05 level. Highest level of education was strongly related to myopia with myopia increasing .22 D per academic year.

Francis Young has also contributed much experimental evidence to this theory in his work with chimpanzees, the nearest sub-human primate.

Young(1971) summarizes his work on the effects of restricted visual space as follows. Experimental animals were placed in chairs with their heads in an enclosed hood for two week periods of time and then refracted, exercised for one day, and returned to the hoods. This was possible because monkeys sleep in a sitting position rather than lying down. A control group of chimps, without hoods, showed no significant refractive changes over a one year period. The adult animals kept in the near visual situation began to show myopic changes within the first month after being placed in the chairs and continued to show myopic changes up until the end of the sixth month in the chairs, at which time they leveled off and showed little or no change for the remainder of the year. The animals that had a confined visual space were able to see at a maximum distance of twenty inches and an average distance of fourteen inches. This environment created an average amount of myopia of .75 D with eight of twelve adult subjects showing myopia shifts. Riffenburgh(1965) has shown that adult humans over the age of twenty can develop up to 1.50 D of myopia per year also if engaged in intensive near work. Greene(1970) has shown that submarine crew members, when subjected to abnormal amounts of near point stress of up to twelve hours per day, develop an increase of myopia of up to 1.75 D over a four year period.

Young(1971) continued his experiments by varying the age of the monkeys which were placed in the hoods. He next used adolescent animals equivalent to 12 to 15 year old humans. This experimental group began to show myopic changes after two to three months and developed as much as 2.00 D over a one year period as compared to the adult group which developed .75 D over the same period of time.

Since Young believed that lowered light levels while performing near tasks increased accommodation, he next varied illumination to deter-

mine its effects, if any, on the chimpanzees. Animals subject to very high (25 f.c.) and very low (.25 f.c.) of illumination did not develop as much myopia as those under intermediate light levels of approximately four foot candles. Accommodation he concludes, is directly related to inadequate near point lighting and also to the development of myopia. Young believes that myopia development is a two staged process. The first stage appears to be the development of ciliary spasm where the animal does not relax accommodation for long periods of time. Once this spasm develops, it appears to be followed within one to two months by a change in axial length. He has measured an increase in axial length of up to 5mm and myopic progression of up to 8.00 D associated with it. As further evidence that accommodation is a key element in causing myopia Young(1981) summarizes his studies in which chimpanzees, which had been placed in hoods and developed myopia were given 1% aqueous atropine. The chimps had been in the hoods approximately four months and developed 1.00 D of myopia on the average. Three drops of atropine were administered three times daily and the animals returned to the nearpoint visual space situation afterward. Results showed an average regression of approximately .50 D, a leveling off, and then no further increase of change of refractive error over the remaining two months under the hoods.

Sato(1957) has shown that atropine is effective in humans also as a cure for myopia. He states that younger children benifit most from its use with 90% of the subjects in fifth grade or lower showing a reduction in myopic progression. By comparison, only 53% of high school students benifited from atropine use. Sato continues by stating that this therapy is only effective with weak or refractive myopia and not strong myopia which we have already assumed to be over 9.00 D and more pathologic in nature.

Since it appears that accommodation plays a major role in the developement of myopia let us examine the possible mechanism by which this takes place. As the eye accommodates several events take place. First, the ciliary body contracts, moving forward and causing the lens to buldge primarily on its posterior surface as is seen when observing the fourth Purkinje image, Borish(1975). This buldging causes the pressure in the anterior chamber to decrease while conversely, the pressure within the posterior chamber increases. Young(1981) states that preliminary studies have shown an increase of 6mm Hg in the posterior chamber if the eye is fixating at twelve inches. This increase in pressure is maintained as long as accommodation is stable but decreases as accommodation drops due to a receding fixation point. He therefore concludes that if an animal or human is placed in a nearpoint visual situation for extended periods the pressure within the vitreous chamber will increase thus causing an enlargement of the chamber and the developement of myopia. We have already discussed the concept of scleral stress which would be involved in this mechanism. Young(1981) finds that scleral creep will no longer occur in monkeys after the age of eight years and in humans around 25 to 30 years of age. This correlates well with his studies of adult monkeys which developed smaller degrees of myopia than did the younger subjects. The adults show what he calls "pseudo" myopia which is due to increased accommodation or lens equivalent power while the younger chimps show greater amounts of myopia or true myopia which is accompanied by an increased axial length.

If this theory on myopic development is to hold, it must further explain what happens to the eye when a certain degree of myopia has become established and the eye no longer is under the full accommodative demand placed on it by the near point object of regard. It would seem to

be evident that at some low degree of myopia the progression should logically stop. This would depend also to some extent on the habitual working distance of the myope. Young(1981) believes that this would be the case were it not for the intervention of vision care and the implementation of minus lens for near work. This causes the individual to become emmetropic for distance vision and to again exert full accommodation at the near point. The increased accommodation causes further progression into myopia and another trip to the vision care specialist. Via this process myopia of greater than 2.50 D may develop and still not be the result of a pathological process. Sato(1957) reports that between the years 1914 to 1937 the percentage of myopic students in Japanese middle schools increased from 15 to 45%. It seems possible that this increase could also coincide with the advent of vision care on a larger scale in that country.

In conclusion of this discussion, it appears that much evidence has been compiled on the environmental cause of myopia. Most of this has come out of the work of Francis Young and his co-workers who have shown that artificial myopia can be caused in chimpanzees by placing them in artificial situations where accommodation is stimulated. Atropine use, which inhibits accommodation, has been shown to decrease existing amounts and halt the progression of myopia. This adds further support to the major role which accommodation plays in myopia developement. Poor lighting and visual hygiene has also been associated with greater accommodative demand and thereby linked to myopia. Number of years of education has been linked to myopia as has amount and duration of near point visual stress. Although this review is by no means exhaustive as to the numbers of different studies which have been done in this area, it seems that enough has been shown to strongly tie near point visual demands with myopia.

Biological or Genetic Theory of Myopia

Our final consideration will be with the Biological or Genetic theory of myopia developement. The theory had its beginnings in 1913 when Steiger theorized that near work had no direct relation to myopia and that the condition was entirely hereditary. He explained the seeming relation between near work and myopia as the result of natural selection. Since that time this theory has been widely investigated and enjoys its greatest popularity among the medically oriented vision care professionals, Duane(1979). The genetic aspects of myopia may be studied in two ways. First by examining uniovular and biovular twins and secondly by using the family tree approach. We will examine the literature in these two areas beginning with the twin studies.

There are two types of twins. Identical or uniovular twins develop embryonically from the same ovum while fraternal or biovular twins develop from different ova and sperm cells. Genetically speaking, the uniovular twins are isogenic meaning every gene present in one is also present in the other. Therefore if a trait is genetically determined it should be present in both of their phenotypes. Biovular twins are anisogenic meaning some genes are identical and others are not. Goldschmidt(1968) has done considerable work in the study of twins and makes several conclusions. He finds that concordance or agreement of refractive errors is higher in uniovular twins than in binovular and that this concordance is most pronounced in the emmetropic range and in lower degrees of ametropia. However, the difference in concordance between the two twin types is greatest in higher ametropias. Sorsby(1974) agrees with these findings that identical twins tend to have similar refractions while binovular twins show no such similarity. He has also

found the six different refractive components to be similar in uniovular twins. He found that in 78 pairs of identical twins 70.5% had close agreement in refraction and differed by under .50 D while 30% of the fraternal twins and 29% of the control group had similarly close agreement. Karlsson(1967) collected data on 99 monozygotic pairs of twins and 39 dizygotic pairs. He found an overall concordance rate for myopia in the first group to by 94% whereas the second group showed a 29% concordance. Unlike the two previous studies cited his data showed a high concordance rate in both mild and severe cases of myopia.

Another type of study which has been attempted is that in which identical twins are reared apart and the genetic tendancies toward myopia noted. Unfortunately, as Young(1981) points out, studies of this kind are inconclusive due to the fact that the twins are so similar in behavioral characteristics that it would be difficult to produce totally different behaviors. Another problem is that even though the twins may be raised far apart, their environment may be essentially the same due to the fact that an attempt is made to place both children in environments characteristic of their natural parents.

We will now examine the way myopia can be studied via the family tree design. Grosvenor(1977) reviews the work of Hirsch and Ditmars and states that the higher the degree of myopia found in offspring, the greater the percentage of parents who were also myopic. Subjects with refractions from 1.00 to 2.00 D had a 20% rate of myopic parentage while myopes over 7.00 D had a 55% rate. From this they concluded that patients with higher degrees of myopia show hereditary influences, while those with lower degrees of myopia show less or no hereditary influence. In Duane's text of Clinical Ophthalmology(1979) Sorsby has written a chapter on the genetics of myopia and concludes that family

studies have shown a parent/child correlation coeficient for axial length and corneal power on the order of.50 and that this would be expected for polygenic determination by a number of genes without dominance. Kuhn(1962) cites a study done in 1939 in which parents and offspring refractions were compared. It was found that 12.7% of the children of non-myopic parents developed myopia, 37.1% of the children with one myopic parent developed myopia, and 72.2% became myopic if both parents were myopes. From this he concludes that refractive error has the characteristics of a recessive trait. Kuhn also states that the highest positive correlations were found between mother-daughter and mother- son respectively. Goldschmidt(1968) has also found a positive correlation between the number of myopic offspring and the degree of myopia of the mother.

Young(1975) on the other hand, in studies of both humans and subhuman primates, finds no relationship between the refractive characteristics of parents and their offspring or between the refractive characteristics of the siblings themselves when these are equaled for age. As further proof he refers to the Eskimo study, previously reviewed in this paper, which showed a sudden and great degree of change in a population. This rapid a change in the appearance of myopia cannot readily be accounted for on the basis of heredity. Also the statistical correlation found between parents and children was significantly lower than that found between siblings. To explain the findings of other researchers who have previously found a correlation between myopia in parents and their children Young uses an analogy: "If an English speaking male marries an English speaking female, they have children who speak English. But no one will argue that speaking English is due to heredity". From this

it can be seen that myopia could be due to the social behavior of reading and sustained near point work passed on to the children by their parents. Without the control of the environment it seems impossible to make any concrete statements about the role of heredity on myopia.

Let us now consider the modes of inheretance which have been postulated. Here we can see that there are as many proposed modes as there are authors on the subject. For example, Sorsby(1979) believes any refractive error which is between +6.00 and -4.00 D, or a correlation ametropia, to be of a polygenic inheritance pattern. Beyond this range, or a component ametropia, he believes it to be of the monofactorial pattern. Goldschmidt(1968) reviews several different studies: Wold concludes that if neither parent is myopic the trait is recessive but if both parents are myopic it is dominant, Paul believed it to be dominant, Beresinskaja judged it to be recessive. Goldschmidt concludes that it is impossible to make a statement on the mode of inheritance purely by studying the pedigree.

In concluding I would like to review an article by Garber(1978) on the subject of myopia and heredity. Garber states that there may be a tendancy for myopic parents to raise myopic children but this is due to the encouragement well educated parents give their children in education and therefore reading and near work. This he concludes, is why myopia is most common in advanced, literate societies and rare in primitive and illiterate societies, Borish(1975). This view is similar to that previously mentioned by Young who also believes that conclusions drawn from genetic studies may be environmentally influenced. From the material presented here it seems that Tscherning's(1900) thoughts on myopia are still the most probable explanation. Weak myopia seems to

be due to the effects of environment and less to heredity while strong myopia appears to be the result of genetics. Studies on the genetics of myopia seem to lead to varied conclusions and are flawed in that it is not possible to control environmental factors. The evidence supporting the biological or genetic theory of myopia development is not conclusive on its own at the present time.

PART TWO: The Control of Myopia

I. INTRODUCTION

Theories on the control of myopia have come from a number of directions through a number of different scientific schools. Current literature on myopia control may be found under such headings as pharmacology, surgery, psychology, and nutrition. Orthokeratology, vision training, radial keratotomy, and plus lens approaches, to name just a few, have been presented as methods for controlling one of the most frequent conditions encountered by the eye care practitioner. Although the incidence and degree of myopia varies greatly according to demographic factors, literature cited by Borish (1970) estimates that in the United States between 11% and 39% of all children and young adults may be classified as myopes.

In addition to the high prevalence of myopia in the general population, many other factors make myopia a very real cause for concern to the eye care practitioner. As stated earlier, myopia tends to be progressive. Psychological factors must also be considered since the myope is confronted with the fact of having poor eyesight, an idea reinforced by the need for contact lenses or cosmetically unattractive spectacles. Birnbaum (1979) points out that the major concern however lies in the fact that the uncorrected myope is in effect visually handicapped. For these reasons, the practitioner must be aware of the various means available in the control of myopia.

Before any conclusions may be drawn concerning the effectiveness of a particular technique in controlling myopia, several points must be qualified. Kerns (1979) emphasized the importance of initially classifying the type, magnitude, and progression rate of the myopia in question. For instance, a study which claims that bifocals caused myopia to progress would not be a valid one if in fact the experimental group consisted of purely pathological myopes.

Second, consideration must be given to the dioptric amount of myopia reduction needed to make the results significant. A study which claims absolute control in subjects with 0.50D of myopia initially, clearly has different implications than a study which claims partial reduction of 1.00D in subjects who initially had 3.00D of myopia.

And third, factors other than the technique focused on may be in part responsible for changes in refractive state. Therefore, demographic factors such as age, sex, nutrition, and geographical locations are important considerations in a complete study on myopia control.

Myopia control is by no means a new field. Tscherning (1900) disclosed a surprising amount of knowledge and understanding of the underlying causes and treatments concerning myopia. He discussed the use of atropine to relieve myopia caused by "spasm of accommodation". Spectacles designed to suppress the influences of accommodation at various distances, which included bifocals, were also described by Tscherning at the turn of the century. Extraction of the crystalline lens in cases of myopia of high degree and other surgical techniques involving tenotomy of the recti were also discussed.

It is unfortunate that over eighty years after Tscherning's book was published, we are still without a universally accepted approach for arresting myopia progression. Furthermore, although the techniques known to Tscherning and his contemporaries have been constantly updated with advances in technology, the concepts and theories concerning myopia and myopia progression have remained relatively unchanged.

Areas of emphasis in this section will include multifocals and plus lens approaches, pharmaceuticals, contact lenses and orthokeratology, vision training, and surgery.

II. HISTORICAL VIEWS OF MYOPIA CONTROL

An early account of preventive vision care for myopia was described by Bates (1920). His theory on myopia control, which became known as the Bates Method, has received considerable attention from the general public. Bates believed that accommodation was controlled by the oblique muscles which adjusted the eyeball for vision at different distances. Exercises were designed to induce maximum relaxation of the accommodative system and therefore prevent the occurrence of myopia. Recommended exercises included: 1) shifting and swinging of direction of gaze between two separate targets; 2) a method known as palming, in which the palms of the hands are placed over the eyes in order that blackness be seen; 3) viewing a familiar object such as a Snellen Chart on a daily basis; and 4) a self-taught method for achieving maximum visual acuity through central fixation.

Although the background upon which Bates bases his theories is most questionable, Grosvenor (1980) points out that, "... the Bates system of Myopia control parallels in many respects the procedures of rotations, fixations, and accommodative rock ... advocated by many functional optometrists."

Traditional Chinese vision care is based on ancient holistic principles which focus on prevention through eye exercises. Pavlichko (1980) described the exercises designed to prevent myopia as, "... a form of acupressure; digital pressure is applied to acupuncture points around the eyes." Students are also advised to take frequent breaks from near visual activities, maintain a proper reading distance, and hold reading time to about 90 minutes per day.

Nolan (1974) constructed a myopia prevention booklet designed to inform parents of ways to help prevent the development of myopia in children with a hereditary tendency. Points emphasized in the booklet include: an explanation of excessive near work as a causative factor; the importance of proper desk placement, working distance, and illumination; encouragement to frequently look up from the reading material; and a recommendation that plus lenses be prescribed for all near work, including watching television.

Many methods for preventing myopia have been advocated by many clinicians. Various holistic approaches have been designed which utilize the ideas presented by early Chinese medicine, Bates, and others. However, many similarities become apparent in each, such as the role of the visual environment, the importance of controlling the visual environment, and the need for maintaining an efficient and flexible visual system. The significance of these factors will become evident in subsequent discussion of other major approaches in the control of myopia.

III. VISION TRAINING

The utilization of visual training techniques alone in myopia control has produced dubious success at best. Although isolated case studies reported by Rowe (1947) and Preble (1948) showed improved visual acuities in the presence of visual training programs, reports by Woods (1945) and Kennedy (1951) indicate otherwise. Critics argue that subjects simply learn to better interpret the retinal blur circle.

In one of the earliest reported studies concerning visual training in myopia control, the famous Baltimore Myopia Study in 1944 received widespread attention and thorough examination by a number of individuals. Woods (1945) described one of the first accounts concerning the study. The project was funded by the Curtis Publishing Company while the visual training was conducted by A. M. Skeffington. One hundred and eleven subjects were selected and examined at the Wilmer Institute, however, results of the preliminary examination were concealed from Skeffington during the training. The Wilmer Institute on the other hand, had no knowledge of the type or extent of training. Refractive errors ranged from -0.50 to -9.00D and the ages of the subjects ranged from nine to 32 years. An average of 25 training sessions were performed during the 13 week study. Periodic examinations were made through the completion of the training program, at which time the data was tabulated and recorded (to be analyzed later) in a medical periodical.

Skeffington's visual training program employed the concept that, "... seeing is learned act and is therefore susceptible to training." Spheres, cylinders, prisms, and specially designed targets were used in order to improve visual skills and visual behavior patterns. No specifics regarding the actual training techniques were mentioned in any of the evaluations which followed the Baltimore Study.

Woods (1945) reported the official ophthalmological views concerning the Baltimore Study. He summarized the results by grouping the subjects according to percentage of acuity improvement. They are as follows: 1) 29.1% of the subjects showed an average improvement (on all four types of acuity charts) of 27 percentage points; 2) 30.1% showed inconsistent improvements of only 14.7 percentage points; 3) 31.1% showed a slight increase of 3.2 percentage points; and 4) 9.7% showed a slightly diminished visual acuity of 10.8 percentage points.

Despite a maximum average increase of one to three lines of Snellen acuity in the Group 1 individuals above, Woods believes that correct interpretation of the blurred retinal image was responsible for the improvement. Woods (1945) went on to say that, "With the possible exceptions of educating some patients to interpret blurred retinal images more carefully ... this study indicates that the visual training used on these patients was of no value for the treatment of myopia."

Hackman (1947) performed a statistical analysis of the Baltimore Study. He pointed out weaknesses in the study which made it very difficult to

accurately interpret the results statistically. For instance, the study was uncontrolled, the criteria for subject selection was not defined, it was assumed that each subject had the same amount and type of visual training, and the actual purpose of the study was never determined. Hackman concludes however that since a considerable number of subjects improved in visual acuity following the training, "... the Baltimore Myopia Study has made a very valuable contribution which will pave the way to future research." Unfortunately, critics of the study had convinced enough researchers that vision training could not control myopia, and Hackman's prediction was never fulfilled.

A few case reports of attempted myopia control through vision training did appear in the literature in the years following the Baltimore Study. Rowe (1947) reported an increase in unaided visual acuity from 20/200 to 20/40 in a student attempting to get into the naval reserve, however no change in refraction was noted. Preble (1948), using techniques recommended by the Optometric Extension Program and A. M. Skeffington, also found an improvement in unaided visual acuity from 20/200 to 20/40 in a 13 year old. Paradoxically, during the six month training period the lens needed to achieve 20/20 acuity actually increased from -2.00 to -3.00D, despite the improved visual acuity. Training consisted of a correction of +1.00D over one eye and a frosted lens over the other. Preble (1948) concludes that the case is, "... typical of my training experience with myopic patients. The vision improves but the myopia does not reduce in amount."

Kennedy (1951) reported a case of an uncorrected eight year old myope showing an actual progression in the presence of training over a 14 month period. The training consisted of poloroid projections, base-out fusion cards, accommodative rocks, and spatial projection training on A N series. The myopia reportedly increased from -1.00 to -1.50D while the visual acuities dropped from 20/60 to 20/200.

A factor consistently ignored in the above mentioned studies is the differentiation of the type of myopia being dealt with. For instance, a study involving subjects with diagnosed functional myopia might reveal very different results than a similar study involving purely hereditary myopia. In the case of Kennedy's study above, it is highly probable that he was dealing with something other than functional myopia due to the early onset of the case at hand.

Little has been written solely on vision training and myopia control since the group of studies which were conducted in the 1940's. Currently practiced training procedures frequently combine the use of bifocal lenses and accommodative rock techniques in order to prevent or slow down progressive myopia. Birnbaum (1979) emphasizes vision training as a means for creating a visual system capable of withstanding environmental stress. An advocate of the near-point stress hypothesis, Birnbaum states that, "Vision training may also be effective in more actively reducing accommodative stress through the use of plus acceptance training, accommodative inhibition training, and supportive general relaxation, stress reduction, and imagery techniques."

The desire by military personnel to pass the visual acuity requirements needed to become officers prompted many of the studies which were reported during World War II. Unfortunately however, few of the studies actually documented training procedures, and changes in visual acuities were stressed as measures of success rather than refractive changes. Although little evidence to support the validity of visual training in reducing myopia surfaced from the studies in the 1940's, it is surprising that the concept was laid to rest so abruptly. It is conceivable however, that with the advent of numerous other methods offered as controls for myopia, vision training has been left by the wayside.

IV. BIFOCALS

Following the apparent failure of vision training to produce sound results in the area of myopia control, researchers turned to other theories in search of the ultimate technique. The application of multifocals as an effective means for curbing myopia progression gained credibility in the mid-1950's. Although some studies have attempted to prove otherwise, there is much evidence in the literature in favor of bifocals. However, current beliefs emphasize the fact that bifocals are directed at prevention of, rather than the reduction of myopia. From a functional standpoint, the purpose of bifocals is in effect to optically control the near point environment.

An early account describing the use of bifocals in myopia was reported by Wick (1947). He prescribed bifocals for an 18 year old male with 3.00D of myopia. Based on the fused cross-cylinder findings, Wick selected an add of +1.25D in hopes of reducing fatigue and headaches associated with complaints

of near point eyestrain. Although the add provided greater comfort initially, Wick made no inferences concerning the effects of the bifocals on the patient's myopia.

A "protective-corrective" program to control myopia was presented by Parker (1958) which utilized the concept of a combination lens. In Parker's program, the corrective part involved the use of a lens which provided adequate visual acuity for distance and near, and the protective part was aimed at protecting what acuity the patient had left. The reading portion of the bifocal was determined by adding +0.25 or +0.50D to the near nets. Parker randomly chose clinical records from his own files and compared the progressive tendency of 19 myopes corrected for distance only with 12 myopes receiving the protective-corrective lenses. A constantly progressive trend was noted in the former group, while a small decrease in myopia was noted in some cases in the P-C group. Parker credited the success of the P-C lens to the maintenance of a relaxed accommodative system which provided a range of performance able to withstand the demands of the near point environment.

Although significant, the Parker study is by no means conclusive. Examination of the graphs reveals that the myopes receiving the distance lens only progressed at an annual rate of about 0.50D, while in the P-C group, myopic progression was essentially zero. However, Parker leaves too much information to the reader's imagination for the study to be clear cut proof of the validity of bifocals. For instance, essential variables such as entering myopia, sex, and age were completely ignored in the study.

Watkins (1959) designed a simplified "check list for and against myopia control lenses". He described the existence of the myopia-bifocal problem as a result of professional disagreement over the etiology and description of types of myopia. Watkins suggested indications for prescribing bifocals such as: esophoria at far and near, binocular cross cylinder findings 1.00D or more above the best subjective lens, a myopia progression rate of 0.50D to 1.00D per year, absence of a myopic crescent, myopia of short standing, a high degree of myopia seen in a young patient, dietary deficiencies, and verbal approval by the patient. Contraindications for bifocals included: rigid retinoscopy findings, exophoria at all distances, low cross cylinder findings, long standing myopia, presence of myopic crescent, and a small amount of myopia seen in an older patient.

Mandell (1959) conducted a fairly extensive eleven year study which compared the progression rate of 175 myopes, 59 of which had at some time

received bifocals. Mandell concluded from his study that the bifocals had not eliminated or reduced the progression of myopia. Indeed, his results even showed a progression of myopia in 91% of the subjects in the bifocal group, while only 76% of the non-bifocal group showed any appreciable progression. However, inspection of the bifocal group reveals an initial average refractive error of 2.75D with an average initial age of 14.3 years, while the non-bifocal group showed an initial refractive error of only 1.48D and a significantly different average initial age of 17.1 years. In an editorial footnote to the Mandell study, Hirsch accurately points out that, "The age and initial refraction seemingly determined which patients received bifocals, and which patients did not."

An attempt to compare the effects of bifocals on myopia progression to non-bifocal wearers of similar age and refractive error was reported by Roberts and Banford (1967). Analysis was performed on fourteen years of case records involving 85 bifocal wearers and 396 non-bifocal wearers from their own partnership practice. A very comprehensive statistical investigation revealed the following major points: 1) with age differences factored out, the bifocal group showed a mean annual rate of myopia of -.314D, while the rate for the single vision group was -.407D; 2) the apparent retardation in the progression rate was 22.8% more effective for the bifocal group; 3) bifocals appeared to benefit girls considerably more than boys; 4) a 37% reduction in rate of myopia progression occurred during periods between the first and second refractions, while only a 3% reduction was noted in subsequent refractions; and 5) children fit with bifocal adds of 1.25D to 2.00D changed more rapidly than those with 0.75D to 1.00D adds.

Although the study suggests that bifocals do indeed retard the progression rate of myopia to some extent, Roberts and Banford were cautious to point out that, "There is no reason to believe that bifocals could affect the progression of the structural myopia, but it is reasonable that bifocals, by altering certain environmental factors known to contribute to ciliary hypertonus, could alter this component of the manifest refractive error."

The most recent large-scale controlled study on bifocal control of myopia was performed by Oakley and Young (1975). The study was designed with an attempt to control confounding variables such as sex, age, and initial refraction while following the subjects for a number of years. A native American sample of 156 subjects ranging in age from 6 to 21 was contrasted with 441 Caucasian subjects. Flat top bifocal adds were prescribed

so that the add intersected the pupil center. Fifty-four Native Americans and 226 Caucasians were fit with bifocals based on cycloplegic determination of the initial myopia present.

Combined results from the two samples indicated the following: 1) the bifocal group showed a mean annual rate of progression of 0.04D; 2) the control subjects showed a mean annual rate of 0.50D; and 3) the overall annual rate of progression for the bifocal group was 8% of that shown by the non-bifocal group.

From the results of the study, Oakley and Young concluded that, "The annual rate of progression of -0.04 diopters per year found among the bifocal subjects is uncommonly found among myopes at these age levels and suggests that bifocals are having a controlling and reducing effect upon the rate of progression. The effectiveness ... may well depend upon the very high position of the add fitted to the child."

Birnbaum (1979) has most recently suggested the application of plus lenses for children showing signs of accommodative dysfunction. He postulated that virtually all cases of incipient myopia show signs of accommodative insufficiency. Birnbaum emphasizes that the decision as to whether bifocals or single vision plus lenses should be given is dependent on the distance acuity through the nearpoint correction. For instance, for school children a bifocal is indicated, since a single vision lens will not afford clear vision for nearpoint as well as black-board work.

Based on discussion of the above mentioned studies, it appears that there is sound evidence in favor of the application of bifocals for the control of myopia. The importance of early screening of young myopes for degree of myopia, rate of progression and environmental influences becomes apparent. In order to justify the use of bifocals, these factors are essential in the diagnosis of the type of myopia being dealt with. In conclusion, it is evident that when used properly, bifocals present a safe and effective means for curbing the progression of myopia.

V. PHARMACEUTICALS

As stated earlier, it is believed that functional myopia is caused by an overaction of the ciliary muscle in the presence of near-point stress. A logical means for counter-balancing the accommodative spasm would be to

inhibit the action of the ciliary muscle with an appropriate pharmacological agent. Atropine, tropicamide, scopolamine, and other similar acting drugs have been utilized in the past as means for attempting to prevent the progression of myopia.

Abraham (1965) reported considerable success in reducing the degree of myopia progression with nightly instillations of 1% tropicamide. He used a carefully selected experimental group of 136 subjects who were initially matched with 164 control subjects for average age ($\bar{x}$=12 years), sex, and family history of myopia. Average entering myopia was unmatched however, being -2.27D in the experimental group and -1.59 in the control group.

Periodic examinations were performed on each subject throughout the 18 month study with the following strikingly different results reported by Abraham (1965). Of those subjects treated with 1% tropicamide, 52.9% showed no progression, 70.6% showed 0.50D or less, and 29.4% showed a progression of greater than 0.50D. In the control group, on the other hand, 16.4% showed no progression, 37.2% showed 0.50D or less, and 62.8% increased by more than 0.50D. Further, Abraham noted an increase of myopia in the untreated control subjects of 0.85D, while the experimental subjects increased by only 0.44D over the same 18 month period.

Young (1965) cited an unpublished study reported by Bedrossian at the First International Conference on Myopia in 1964 concerning the effects of atropine instillation on the development of myopia in children. Bedrossian chose twenty-four myopic children who were progressing at an average rate of 0.67D per year and compared them with twenty control subjects matched for age and progression rate. Atropine was instilled in one eye only on a daily basis for a period of one year. The treatment was then reversed so that the opposite eye received the identical atropine treatment during the following one year period. Based on refractions every four to six months, Bedrossian found that when under atropine treatment the degree of myopia either stabalized or regressed approxiamtely 0.25D. In other words, the eye being treated with atropine showed no further myopic progression, while the untreated eye progressed at a rate equivalent with the control group.

Unfortunately, no figures were given for the average two year progression rate in the control group. Young reported however that, "When the experimental group was compared with the control group, in terms of amount of myopia developed over the two year period, the control group had significantly more myopia than the experimental group."

In another study reported by Young (1965) from the same conference, Gostin used a combination of scopolamine and bifocals to control myopia progression. Daily instillation of one drop of scopolamine, given to 106 children (no age was given), was coupled with bifocal prescriptions so that near work might be maintained during the one year study. Although exact figures were undocumented, Gostin reported that no progression was noted during the scopolamine treatment, whereas all subjects exhibited progression prior to the study. An important point emphasized in the two studies described by Young was the fact that myopia progression appeared to stabilize despite the intact convergence mechanism which remained in play during periods of near work. Further, he concludes, "...it appears that convergence as such, does not contribute to the development of myopia."

More recently, Kelly et. al. (1975) conducted a thorough study which compared the effects of various methods on myopia. A control group, consisting of 86 myopic subjects (Group I) was compared with 77 subjects receiving a combination of atropine, bifocals, and phenylephrine (Group II) and a third group of 38 subjects receiving atropine, bifocals, and phenylephrine (Group III). Each subject was initially examined at age 11 and again at one year ($\pm$ 3 months) intervals until age 14. Subjects in Group II were administered 1% atropine three times daily for seven days, at which time bifocals were prescribed based on the level of atropine refraction with no addition. Phenylephrine (5%) drops were then instilled nightly. Group III subjects were given 1% atropine drops once or twice daily, with the duration dependent on the level of myopia reduction attained.

Results from the Kelly study were as follows: 1) in the control group, 15% of the subjects showed an arrest of myopia progression, while the average change in myopia was +0.52D after one year; 2) in Group II, 66% of the subjects showed an arrest of myopia progression, while the rate of change was -0.58D over six months. From this study then, it appears that atropine and phenylephrine have dramatic halting effects on myopia progression. This strongly suggests, as Young (1965) points out, "... that accommodation plays a major role in the development of simple or school myopia."

Before the decision is made to use an appropriate pharmacological agent in treating myopia, it is essential that adequate precautions be taken. The physician must be aware of the side effects that may develop, as well as the

possibility of allergic reactions which may be encountered. For instance, in the Abraham study, the side effects of tropicamide were compromised by nightly instillations of the drug followed by cold compresses. In this manner, the treated eye was seldom dilated in the morning and complaints of stinging were usually eliminated.

Although pharmaceuticals have been shown to be effective in treatment of myopia, their efficacy for clinical use is still debatable. Long term safety of such an approach remains questionable, a fact which may influence the desired course of action taken by the clinician.

VI. CONTACT LENSES AND ORTHOKERATOLOGY

The use of contact lenses in controlling myopia had its rather unique origin in the late 1950's when practitioners began to notice the apparent arrest of myopia progression in the presence of hard contact lens wear. Morrison (1958) produced one of the earliest reports of success with contact lenses at a time when lenses were thick, of large diameter, and fit flatter than the flattest corneal meridian. Bailey (1958) believed that the lens produced a mechanical pressure which flattened the cornea, thus reducing the need for more minus power.

The subsequent conception of the field of orthokeratology in 1962, as described by Grant and May (1971) was based on the corrective aspects of contact lenses with corresponding changes in visual acuities. They defined orthokeratology as "... the reduction, modification or elimination of a visual defect by the programmed application of contact lenses or other related procedures." Although the techniques utilized by orthokeratologists may vary considerably, most clinicians agree that flattening the cornea results in an alteration of the refractive status in a less myopic direction.

Although orthokeratology bases its success in myopia reduction on corneal changes, many other studies have attempted to explain the cessation of myopia progression in terms of non-corneal changes. Morrison (1958) feels that there are several factors involved in myopia control by contact lenses: 1) the lens retains the curvature of the cornea; 2) the lens produces a holding effect on any stretching of the eyeball; 3) contact lenses do not have the same prismatic effects and accommodation-convergence relationships that spectacle lenses have; 4) contact lenses produce a larger

retinal image size than do spectacles; 5) contact lenses afford a better depth of focus due to smaller pupil size; and 6) they provide a wider field of view than spectacles. Morrison suggests that daily wear of contact lenses has an arresting effect on the progression of non-pathological myopia.

Silbert (1962) classified non-pathological myopia and summarized the possible roles of contact lenses in reducing each type. Axial myopia is believed to be controlled by way of the gentle pressure exerted by the lens, thus retarding any possible axial length changes. Refractive myopia is greatly influenced by the cornea since it accounts for approximately 80% of the total refractive power of the eye. Silbert states that refractive myopia is controlled by a contouring effect on the cornea provided by the contact lens which prevents permanent curvature changes. In cases of functional myopia, it is believed that the contact lens provides the wearer a different spatial orientation which removes the original functional cause of the myopia.

Numerous studies have been published which support the beliefs of Morrison, Silbert, and others advocating the use of contact lenses to halt the progression of myopia. Rengstorff conducted extensive research involving changes in corneal curvature associated with contact lens wear and its effects on myopia. Rengstorff (1979) reported long-term changes upon removal of contact lenses in a direction of corneal flattening. An average change of 0.75D corneal flattening was seen in over 100 eyes of men 18 to 26 years old. Progressive corneal flattening was shown in some individuals for one, three, or seven days. He also noted that the most common changes in corneal astigmatism were increases in with-the-rule astigmatism, however he made no mention as to the magnitude of the changes. The corneas did not revert to their former curvatures after the lenses had been removed for more than 30 days.

Rengstorff suggests that changes in corneal curvature are "... probably a combination of mechanical, physiological, and anatomical factors, and not singly a result of mechanical pressure from a contact lens." He feels that the mechanism for long-term structural changes may have a chemical basis involving the variability of available oxygen to the cornea, which may induce alterations of corneal curvature.

Stone (1973), in a study on contact lens wear in young myopes, believed that Rengstorff was dealing with adults whose myopia had probably stabilized. She concluded that after two years of corneal contact lens wear, myopia in young children appeared to stabilize, although it increased up to that time.

In an impressive five year study, Stone(1974) compared the progression of myopia in eighty myopic contact lens wearers with that in forty myopic spectacle lens wearers. Using conventional apical clearance fit hard corneal lenses, she monitered the myopia in growing children after two years of wear.

The essential results of Stone's research are as follows: 1) the myopia of spectacle lens wearers increased by an average of 1.75D over the five year period; 2) with-the-rule astigmatism also showed a slight myopic increase of 0.38D in the spectacle lens wearing group; 3) the myopia of the contact lens wearing group showed an average decrease of 0.12D over this same period; while 4) with-the-rule astigmatism increased by 0.87D in the contact lens wearing subjects.

It appears from Stone's study that contact lens wear has not only a stabilizing effect, but a slight reduction effect on myopia. This apparent change however, seems to be at the probable expense of an increase in refractive astigmatism.

Stone concludes that the mechanisms involved in the myopic progression of spectacle lens wearers are an increase in axial length, as well as a possible increase in crystalline lens power. Corneal power as measured by keratometry did not show a significant change. The corneal curvature in the contact lens wearers flattened however, which resulted in decreased corneal power and a subsequent reduction in the degree of myopia. Yet, the total reduction in myopia could not mathematically be explained by the change in corneal curvature alone. Stone suggests that contact lenses may have additional effects on the eye, such as inhibition of axial length elongation or increases in crystalline lens power, to account for this.

While there are those practitioners who advocate the use of conventional fit hard contact lenses to arrest the progression of myopia, there are those who carry the concept further by designing specific fitting procedures aimed at reducing or completely eliminating myopia. As stated earlier, orthokeratology techniques vary considerably among different clinicians, however most techniques utilize lenses designed to flatten the cornea, thus reducing the myopia.

Grant and May (1970) outlined a procedure for orthokeratology in which the initial lens is fit basically the same as any new contact lens patient, that is, parallel or no more than 0.37D flatter than the cornea. New lenses are then fit as soon as any measurable changes (0.50D or more) are recorded

in either corneal curvature or plus acceptance in lenses. The base curve of the newly fit lenses are computed on the basis of the new flattest corneal curvature, or slightly flatter. The procedure is repeated until plano lens power is achieved and 20/20 visual acuity is maintained with and without the contact lenses. Grant and May report using orthokeratology techniques on 300 patients, with 3.00D being the maximum change in myopia reduction.

Nolan (1971) feels that those myopes under 14 years of age and with less than 2.00D of myopia are the group most amenable to orthokeratology. Nolan uses a procedure in which a plano power contact lens is fit sufficiently flat so that the lacrimal lens corrects the refractive error. For instance, a 1.00D myope is fit with a lens having a base curve 1.00D flatter than the flattest corneal meridian. Wearing time is gradually increased until the desired improvement in unaided acuity is achieved, at which time a minimum wearing schedule is determined which will sustain the improved condition.

Kerns(1976a) conducted a comprehensive exploratory study designed to challenge the validity of orthokeratology and examine the interrelationships among the known variables. Kerns (1976b) monitered changes in corneal curvature, refractive error, and corneal topography in three groups of subjects between 10 and 30 years of age. The tests groups (expressed in number of eyes) were as follows: 1) non-contact lens wearers (N=6); 2) "conventional" contact lens wearers (N=26); and 3) "orthokeratology" subjects (N=36).

The "conventional" contact lens wearers were fitted with rigid lenses within $\pm$ 0.25D of the flattest corneal meridian. The"orthokeratology" subjects were initially fitted with conventional lenses until adaptation and full time wear was achieved. The actual orthokeratology procedure was implemented when the subjects showed a post-refraction of 0.50D less minus as compared to their initial refraction. Lenses were adjusted accordingly with each 0.50D change until a plano post-refraction and 20/20 unaided visual acuity was demonstrated. An average of 1000 days of contact lens wear preceded lens removal.

Results and observations reported by Kerns (1976c) provide photokeratoscopic evidence of corneal contour changes accompanying orthokeratology. A rather dramatic change towards sphericalization was observed in both corneal meridians during the first 300 days of lens wear. Refractive changes

in the orthokeratology group were as follows: 1) a mean of 1.06D decrease in myopia (S.D. = $\pm$ 0.98D) with a range from 0.75D increase to 3.00D decrease in the horizontal meridian; 2) a mean of 0.68D decrease in myopia (S.D. = $\pm$ 0.90D) with a range from 1.00D increase to 3.25D decrease in the vertical meridian; 3) a mean change in refractive astigmatism of 0.42D with-the-rule (S.D. = $\pm$ 0.74D) with a range from 0.25D against-the-rule to 2.00D with-the-rule.

In his analysis of the results, Kerns (1978a) was cautious to point out that corneal modification with contact lenses is still not clearly understood. However, the following trends were revealed upon detailed examination of the study: a) regardless of the base curve-cornea relationship, the horizontal corneal curvature showed a tendency to flatten; b) in the vertical corneal meridian, a flattening occurred when lenses were fit on "K" to 0.50D flatter than "K" and a steepening occurred when lenses were fit greater than 0.50D flatter than "K"; c) the flatter the fit, the greater was the probability of observing increased corneal toricity; d) the resulting with-the-rule astigmatism appeared to be an uncontrollable consequence of the orthokeratology; e) the limits of myopia reduction were affected by the ocular rigidity of the cornea; f) sphericalization of the cornea due to orthokeratology resulted in loss of lens centration which indicated that ocular rigidity had occurred.

In conclusion, Kerns (1978b) emphasizes that, "... Orthokeratology is very much an individualized process and is likely to remain so until factors important to the process are positively identified and quantified. Only when the mechanism for corneal change following contact lens wear are fully understood will there be less myopic views of orthokeratological procedures."

VII. SURGERY

Surgery has most recently gained widespread attention as an alternative for the correction of myopia. Following World War II, Sato (1953) introduced a surgical technique designed to flatten the cornea and thus reduce myopia of high to moderate degree. In the 1970's, Fyodorov expounded on Sato's technique by refining the length and number of incisions in order that it may be useful for minimal to moderate myopia. Fyodorov and Durnev (1979) reported great success in reducing the myopia in 60 eyes with this

technique, which has now become known as radial keratotomy.

Grosvener (1981) outlined the various surgical procedures which have been used to reduce myopia. The techniques and the indications for their use are as follows: 1) scleral resection and scleral reinforcement for progressive myopia of high degree; 2) crystalline lens removal for high degree myopia; 3) refractive keratoplasty for hyperopia (aphakia) and myopia; and 4) radial keratotomy for small amounts of myopia. Since this paper is intended to report the various means of control for non-pathological myopia, only radial keratotomy will be discussed further.

Sato (1953) designed a technique in which radial incisions were made into the posterior two-thirds of the cornea from the endothelial side. Sato claimed a 95% success rate with 32 myopic cases in Japan. The reduction of myopia ranged from 1.50 to 7.00D with an average of 3.00D. Degree of reduction was controlled by the distribution and number of incisions made. An outline of the methods advocated by Sato is as follows: 1) the exact refractive error is determined; 2) following anestheia, incisions are made in the superior and inferior limbal areas; 3) the corneal knife is then inserted through these incisions into the anterior chamber, whereupon posterior incisions are made through the endothelium, Descemet's membrane and two thirds of the corneal stroma; and 4) the eye is then atropinized and penicillin is instilled into the conjunctival sac. It is noteworthy that the limbal incisions were described as self-sealing, therefore aqueous seapage is rare.

Sato claimed a stable refraction within two months. Unaided visual acuities improved in all cases, and 20/20 acuity was obtained in nine cases. From the study, Sato (1953) concluded that, "We feel safe in saying that eyes with four diopters of myopia can be made emmetropic, or so nearly so that only slight correction is necessary to acquire full corrected vision".

In analyzing the last statement, one might conclude that because a slight correction may still be needed, most myopes of 4.00D or less would be dissuaded from undergoing such surgical treatment. It is probable that most myopes in this range are content with the less drastic choices available. Another fault in Sato's logic as pointed out by Ranani (1981) was the fact that the incisions were made from the endothelial side. Indeed, many patients in time soon developed corneal disruption of the delicate physiological balance of the endothelial structure.

Using keratometric data, Fyodorov and Durnev (1979) showed that considerable degrees of myopia could be reduced by making sixteen radial

incisions into the cornea from the epithelial side. Radial keratotomy was performed on 60 eyes with 0.75 to 3.00D of initial myopia. The age of the subjects ranged from 17 to 43 years and the myopia was regarded as stable. Post-operative visual acuities showed improvements in all cases.

The method used by Fyodorov and Durnev is outlined as follows: 1) the cornea is anesthetized with 1% tetracaine hydrochloride; 2) the central optic zone is delineated by a marker of a pre-determined diameter; 3) sixteen radial incisions are cut from the epithelial side with a depth of about three-fourths the corneal thickness; 4) the eye is then irrigated with physiological saline; and 5) an antibiotic is injected under the conjunctiva and the eye is patched.

During the first 3 to 4 days, hyperopia of 2.00 to 3.00D occurred, followed by a gradual decrease so that stabilization was attained by the third month. Following stabilization, refractions revealed 29 cases of emmetropia, 21 cases of myopia of lesser degree than pre-operatively, and 10 cases of hyperopia. A direct dependence between length of incision and the degree of reduction of myopia was reported. For example, for a central zone of 4.5mm the reduction of myopia averaged 1.25D, while an average reduction of 2.65D occurred for a central zone of 3.0mm. In other words, the longer the incision, the greater was the degree of myopia reduction. An average uncorrected visual acuity of 0.86 was reported following the post-surgical stabilization period.

Fyodorov and Durnev hypothesized the mechanism of action in radial keratotomy as being a process by which dissection of the circular ligament of the cornea leads to a weakening of the corneal periphery. As this weakening occurs, that portion of the cornea bulges outward due to the intraocular pressure. Since the periphery of the cornea is now more curved, the central portion compensates by flattening, and therefore the power of the cornea is decreased.

A crucial point is raised when one questions the safety and effectiveness of radial keratotomy, that being the long-term integrity of the cornea. Fyodorov and Durnev (1979) state that, "... 3 to 4 months after the operation the obtained effect was preserved unchanged in the initial follow-up period fo 3 years, therefore there is no reason to believe that it will change in the future." This it seems, is a very strong statement to make based on the rather limited sample at hand.

Ranani (1981) is cautious to point out that surgery may be a viable solution for patients with occupational needs who are dissatisfied with spectacles, providing that contact lens wear has been ruled out first. Ranani also recommends that surgeons emphasize, "... the experimental nature of the procedure and the possibility of fluctuating vision, glare and inadequate correction." He reports that the safety and effectiveness of radial keratotomy is presently being examined by at least four study groups in this country alone. The long-term results of these groups may ultimately determine whether or not the widespread use of radial keratotomy becomes a reality in treatment of myopia of low degree.

VIII. SUMMARY

The following is a summarization of some of the major studies for each method of myopia control as described in this paper.

(KEY: E = Experimental Group; C = Control Group
NA = Not Applicable; and (?) = Not Documented)

STUDY	SUBJECTS	MEASURE OF MYOPIA CONTROL	PERCENTAGE AFFECTED
PART III. VISION TRAINING			
Baltimore Study (1944)	111	VA Improvement (1-3 lines)	59.1%
Rowe (1947)	1	VA Improvement (20/200-20/40)	NA
Preble (1948)	1	VA Improvement (20/200-20/40)	NA
Kennedy (1951)	1	VA Decrease (20/60-20/200)	NA
PART IV. BIFOCALS			
Parker (1958)	E=19 C=12	Progression (D/yr) E=zero(?) C=0.50	NA
Mandell (1959)	E=59 C=116	Myopia Progression Halted	E= 9% C=24%

(Continued)

STUDY	SUBJECTS	MEASURE OF MYOPIA CONTROL	PERCENTAGE AFFECTED
PART IV. BIFOCALS (Continued)			
Roberts and Banford (1967)	E=85 C=396	Progression (D/yr) E=0.314 C=0.407	NA
Oakley and Young (1975)	E=280 C=317	Progression (D/yr) E=0.04 C-0.50	NA
PART V. PHARMACEUTICALS			
Abraham (1965)	E=136 C=164	Zero Myopia Progression	E=52.9% C=16.4%
Bedrossian (1964)	E=24 C=20	Zero Myopia Progression	Greater (?) In E Group
Gostin (1964)	E=106	Progression (D/yr)	None (?)
Kelly, et. al. (1975) E_p(phenylephrine) E_a(Atropine)	E_p=77 E_a=38 C =86	Zero Myopia Progression	E_p=66% E_a=97% C =15%
PART VI. CONTACT LENSES			
Grant and May (1970)	300	Myopia Reduction (up to 3.00D)	100%
Rengstorff (1971)	100	Corneal Flattening ($\bar{x}$=0.75D	NA
Stone (1974)	E=80 C=40	Increase over 5 yrs E=0.12D C=1.75D	NA
Kerns (1976-1978)	36	Myopia Reduction $\bar{x}$= 1.06D Horiz. Mer. $\bar{x}$=0.68D Vert. Mer.	NA
PART VII. SURGERY			
Sato (1953)	E=32	Myopia Reduction ($\bar{x}$=3.00D)	NA
Fyodurov and Durnev (1979)	E=60	Myopia Reduction a) overall b) to emmetropia c) to hyperopia	 100% 48.3% 16.7%

CONCLUSION

Current theories on the development of myopia were discussed in Part I. These include the Nutritional-Disease, Mechanical-Anatomical, Environmental, and Genetic theories. At the present time it appears that there is no single causative agent of myopia. Rather, it is a combination of processes which occurs. We cannot rule out any of the topics previously discussed. Therefore, we believe, a broader more generalized theory is needed to describe what actually occurs in the development of myopia.

Such a theory might be explained as follows. At birth a person would inherit certain anatomical and mechanical characteristics. These would include the points of insertion of the extra ocular muscles. As we have discussed, narrow attachment lines of the superior and inferior obliques cause greater scleral stress, as do obliques, which attach to the globe nearer each other. This could predispose a person to myopia and much like cancer, if environmental factors are present, these will become manifest.

These environmental factors also cause scleral stress but do so in two different ways. Near work or accommodation has been shown to increase the I.O.P. thereby causing stress. Disease and improper nutrition may also weaken the scleral coat causing an axial elongation and myopia.

Young's Eskimo study has given solid evidence that environmental factors and near work are involved in myopia development however, at the same time Eskimo children were doing more near work, their diets during the growth years were changed drastically from that of their ancestors. Perhaps myopia is the by-product of refined foods, sugars, and preservatives which accompany industrialization as well as the increased near visual demands.

The controversy between Genetic and Environmental theories will never be solved because, we believe, both are correct and part of a larger macroscopic theory on the development of myopia.

Current theories on the control of functional myopia were discussed in Part II. Areas of emphasis included orthokeratology, vision training, surgery, pharmaceuticals and plus lens approaches.

In analysis of the results for the major studies discussed in the area of myopia control, it appears that pharmaceuticals and surgery produced the greatest effect on the reduction of myopia. The question of safety, as well as effectiveness is raised however when methods such as radial keratotomy

and atropine instillation are concerned. In addition, obvious limitations exist for individual eye care practitioners. For instance, many pharmaceuticals are not currently available to optometrists in many ststes, while it is doubtful that radial keratotomy would be practiced by every ophthalmologist.

It is our opinion that the method which has the greatest potential in terms of both safety and effectiveness in the control of functional myopia is bifocals. When properly prescribed, myopia progression is essentially zero. In addition, worries of corneal trauma, pupillary dilation, and other side effects are eliminated with this method.

The application of bifocals in the treatment of functional myopia would alleviate the environmental stress factor responsible for axial elongation. It is conceivable therefore that an individual anatomically predisposed to myopia would not manifest the condition due to the optically controlled near point environment.

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