Myopia: A Historical Perspective

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The word "myopia" is thought to be derived from New Latin, which in turn was derived from the original Greek word "mŭopia" (μυωπία, from myein "to shut" + ops [gen. opos] "eye"), which means contracting or closing the eye. This is an accurate description of the typical facial expression of the uncorrected myope as he or she attempts to obtain clear distance vision. Until the introduction of spectacles, squinting the eyelids, resulting in a horizontal stenopeic slit, was the only practical means by which clearer distance vision could be achieved. In ancient times, the myope was reliant upon others with normal vision for the spoils of the hunt and protection in war. In prehistoric times, this dependency must have been even greater. With the advent of civilization, the emergence of agricultural handicrafts, and the written word, the nearsighted at least found a place of more worth in society. As knowledge and fine skills have become increasingly important in our advancing culture, this place of the myope has been continually expanded.

It is interesting to trace the historical perspectives of pathologic myopia in the ophthalmic literature. First to consider is the evolution of our knowledge of myopia, which has been marked by occasional giant strides based on numerous careful investigations and their impartial analysis. However too often, conflicting observations on this subject have been bewildering by their varied and complex protocols, their results, and their conclusions. A tendency toward advocacy rather than investigatory curiosity can be seen to influence the early literature. Yet, myopia remains incredibly to this day one of the major causes of visual disability and blindness about which little is known. As a result, myopia continues to be a perplexing problem of major proportions worldwide. Table 1.1 lists some historical landmarks in myopia.

1.1 Pre-ophthalmoscopic Historical Landmarks in Myopia

Pre-ophthalmoscopic development in myopia started from light, optics, and anatomical studies. There are many reviews of the history of myopia that chronicalize the history of the disease [1–6]. Aristotle (384–321 BC) was generally thought to be one of the first to consider the problem seriously (Fig. 1.1). He described the difference between "long sight" and "short sight" and noted the tendency of the myope to blink the lids and write in small script [7]. Galen's (13–201 AD, Fig. 1.2) concepts very much dominated the early years of medicine. Galen thought that ocular refraction was dependent upon both the composition and quantity of the eye fluids (animal spirit), and he was the first to use the term myopia [7]. From Aristotle's time, it was believed that the eye itself was a source of vision rays, an idea finally dismissed by Alhazin (AD 1100) [8]. Optical correction and myopia evolved very slowly. Although Nero is believed to have watched gladiator battles through a concave ruby, correcting spectacles did not make their appearance until near the end of the thirteenth century. These lenses were convex, and the myope had to wait a few more centuries before the general introduction of minus lenses. Even then, there were few people who wore these minus lenses or advised wearing them [8].

The optics and image formation of refraction were poorly understood in those times. Porta (1558–1593) believed that the image fell on the anterior surface of the lens, whereas his contemporary, Maurolycus (1575), thought that the lens was involved in focusing the image, and that it was more convex

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Table 1.1 Historical landmarks in myopia

Year	Author	Description
384–321 BC	Aristotle	Difference between near sighted and far sighted
138–201	Galen	First used the term "myopia" from original Greek word: myein "to shut" + ops (gen. opos) "eye" Ocular refraction was dependent upon the composition and quantity of the eye fluids
1604	Johannes Kepler	Described retina as the site of vision, not the lens. Demonstrated concave lenses correct myopia and convex lenses correct hyperopia
1801	Antonio Scarpa	First anatomical description of posterior staphyloma, but did not make the link to myopia
1813	James Ware	Noted that people who were educated were often myopic
1854	Von Graefe	First postulated the association between myopia and axial length
1856	Carl Ferdinand von Arlt	First connected staphyloma and myopic refraction
1861	Eduard Jäger von Jaxtthal	First described and illustrated myopia "conus" and enlarged subarachnoid space around the nerve
1862	Carl Friedrich Richard Förster	First observed sub-RPE choroidal neovascularization; "Forster spot"
1887	Adolf Eugen Fick	First used the term "contact lens" and designed glass contact lenses
1901	Ernst Fuchs	"Central black spot in myopia;" "Fuchs' spot"
1902	Maximilian Salzmann	First described defect in lamina vitrea (Bruch's membrane); was later coined as "lacquer crack"
1913	Adolf Steiger	Myopic refraction depends on corneal refraction and axial length
1938	Rushton, R.H.	Measured axial length by x-rays
1965	Gernet, H,	Measured axial length by ultrasonography
1970	Brian J. Curtin and David B. Karlin	Discovered relationship between axial length and chorioretinal atrophy First used "lacquer crack" in this article
1977	Brian J. Curtin	Classification scheme for staphyloma
1988	Takashi Tokoro	Classification of chorioretinal atrophy in the posterior pole in pathologic myopia Definition of pathologic myopia
1996	Brancato R, et al.	Indocyanine green angiography (ICGA) in pathological myopia
1999	Morito Takano and Shoji Kishi	First illustrated foveal retinoschisis using optical coherence tomography (OCT)
2001	Verteporfin in Photodynamic Therapy Study Group	Treated myopic choroidal neovascularization with photodynamic therapy
2002	Baba T, et al.	First described different stages of myopic CNV using optical coherence tomography (OCT)
2005	Nguyen QD, et al.	Treated myopic choroidal neovascularization with bevacizumab
2008	Spaide RF, et al.	Enhanced depth imaging spectral domain OCT for choroidal imaging
2012	Ohno-Matsui K, et al.	Relationship between myopic retinochoroidal lesions with shape of sclera using 3D-MRI *Intrachoroidal cavitation in pathologically myopic eyes*

in myopia and flatter in hyperopia [9]. He did not mention the retina, however, and believed the focal plane was on the optic nerve. Adding to the confusion was the problem of obtaining an upright image in the eye, an accomplishment that early workers considered indispensable for normal vision. A dramatic step forward was made by Kepler (Fig. 1.3), who because of his background in both mathematics and myopia seemed appropriate to address the subject. In 1604, Kepler was able to demonstrate the image formation of the eye and the role played by the cornea and lens. He placed the inverted image at the retina and defined the action of convex and concave lenses upon this system [10]. Later, in

Fig. 1.1 Painting of Aristotle by Francesco Hayez (1791–1882)

Fig. 1.2 A portrait of Galen by Pierre Roche Vigneron (Paris: Lith de Gregoire et Deneux, ca. 1865) (Courtesy of the National Library of Medicine)

Fig. 1.3 A 1610 portrait of Johannes Kepler by an unknown artist

1611, Kepler noted that in myopic eyes, parallel rays of light fell in front of the retina [11]. He also wrote about accommodation, but in this era, there was much confusion as a result of an inability to appreciate the fact that presbyopia occurred in both myopia and hyperopia. Kepler further attributed the ability to see clearly at both distance and near to alterations in the shape of the eye. He went on to propose the "near-work" hypothesis for myopia by stating that study and fine work in childhood rapidly accustoms the eye to near objects [11]. With age and advancing years, this adaptive mechanism produces a permanent, finite far point such that distant objects were seen poorly, a theory that is still accepted today [9].

Newton (1704) wrote about the concept of hyperopia as a condition due to parallel rays of light converging behind the retina and set the stage for the acceptance of axial length of the eye as the sole determinant of refraction. Plempius (1632) [9] provided anatomical proof of increased axial lengthening of the eye was provided by. Boerhaave (1708) confirmed this lengthening while also finding another cause of myopia: increased convexity of the refractive surfaces [8]. Other causes that had been hypothesized were an increase in the

thickness of the lens, an increase in the refractive index, and a change in its position.

In the absence of the instruments necessary to measure corneal and lenticular variables, there were a number of studies confirming the variability of axial length. These included the studies of Morgagni (1761) [9], Guerin (1769) [9], Gendron (1770) [5], and Pichter (1790) [5]. Scarpa (Fig. 1.4) is the first to describe anatomically posterior staphyloma (Fig. 1.5) in two female eyes in 1801 [12]. He coined the Greek word *staphylos* literally means "a bunch of grapes." It is of note that Scarpa described staphyloma, but did not make the link to myopia. Von Ammon (1832) noted that posterior staphyloma was due to a distention of the posterior pole and was not a rare entity. However, he did not make the association between this posterior staphyloma and myopia [13]. It is possible he may have described incomplete closure of the fetal fissure and not a myopic staphyloma. Early epidemiological information on myopia was observed. In 1813, James Ware described the examinations of approximately 10,000 soldiers and made the observation that conscripts in the British Army were almost never myopic, while officers in the Queens' Guard, who were educated, were often myopic [14]. In the mid-nineteenth century, Cohn reported on his examination of 10,000 children in which he noted a link between amount of schooling and myopia [15]. In 1871 Erismann examined 4,358 scholars in St. Petersburg and found that 42.8 % of the highest classes were myopic [16]. In 1885, Randall collected statistics of 146,500 examinations and noted the proportion of myopia in general was very low, with only 1.5 % having simple myopia [17].

Fig. 1.4 Portrait of Antonio Scarpa

1.2 Post-ophthalmoscopic Historical Landmarks in Myopia (1851)

Post-ophthalmoscopic development in myopia started from observations of the optic nerve, the macula, and chorioretinal changes. Von Graefe (1854), in a combined ophthalmoscopic and anatomical study of two eyes measuring 29 and 30.5 mm in length, first postulated the association between myopia and axial length [18]. It was Arlt (1856) (Fig. 1.6), however, whose anatomical studies convinced the scientific world of the intimate association of myopia with axial elongation of

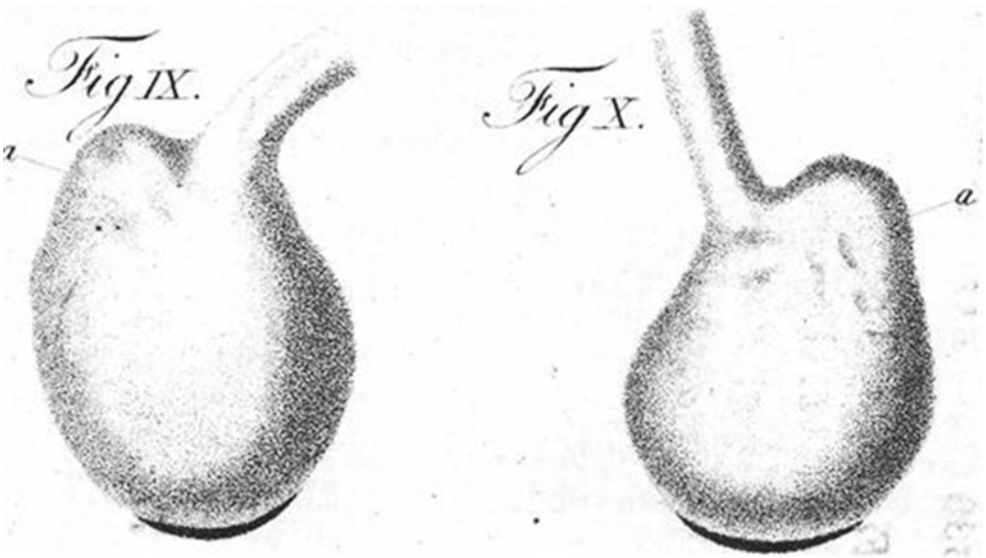

Fig. 1.5 The earliest depiction of posterior staphyloma as contained in the text of Antonio Scarpa (Scarpa [57])

Fig. 1.6 A portrait of Carl Ferdinand Ritter von Arlt by Fritz Luckhardt. The anatomical studies of Carl Ferdinand Ritter von Arlt convinced the scientific world of the intimate association of myopia with axial elongation of the globe at the expense of the posterior pole

Fig. 1.7 A portrait of Eduard Jäger von Jaxtthal by Adolf Dauthage in 1859

the globe at the expense of the posterior pole [19]. Arlt made the connection between staphyloma and myopic refraction [19]. After this, clinical findings in pathologic myopia were noted.

Von Jaeger (Fig. 1.7) was the first to describe and illustrate myopic conus and enlarged subarachnoid space around the nerve in 1861 [20]. He found that the choriocapillaris was sometimes absent within the limits of the conus, and that in extensive staphyloma the choroid over the conus presented the appearance of a glass-like, homogenous membrane which was exceedingly fine and delicately striated and contained a few vessels [20]. In 1862 Carl Friedrich Richard Forster (Fig. 1.8) first observed sub-RPE choroidal neovascularization (Fig. 1.9) [21], and this is what we called "Forster spots." In the same vein, in 1901 Ernst Fuchs (Fig. 1.10) later discovered "central black spot in myopia" [22] (Fig. 1.11) [23], and this is what we called "Fuchs' spots." Its typical course is enlargement to the size of the disc and beyond, growing lighter, and the formation of an atrophic peripheral zone. Fuchs concluded that the choroid is not destroyed, but is either converted into, or covered by, a callosity. It starts with sudden visual disturbances in the form of metamorphoses or positive scotomas, which in the course of years become more marked. Anatomically there is an intense proliferation of the pigment epithelium covered by a gelatinous acellular exudation (coagulum of fibrin), adherent to the retina. The etiology is obscure; certain only is its connection with myopia, or with its process of ectasia [8]. Henry Wilson described atrophy of choroidal epithelium in 1868 [24]. In 1902, Salzmann (Fig. 1.12) noted that cleft-shaped or branched defects were found in atrophic areas in the lamina vitrea that were concentric with the optic disc (Fig. 1.13) [25, 26]. The lamina vitrea is also referred to as "Bruch's membrane." He felt that these defects seemed to be the result of purely mechanical stretching. Later, the term "lacquer cracks" was used by Curtin and Kerlin to describe this lesion, which typically occurs as yellowish to white lines in the posterior segment of highly myopic eyes, resulting from progressive eyeball elongation. Salzmann believed that atrophic changes noted in the myopic choroid followed inflammation and the primary process driving this was stretching of the choroidal stroma [27].

1.3 Modern Historical Landmarks in Myopia

Modern historical landmarks include studies dedicated to explore the individual optical elements of myopia, axial length measurement (x-ray and ultrasound), and the development of contact lenses.

Fig. 1.8 A portrait of Carl Friedrich Richard Förster (Reprinted with permission from The Royal Library, The National Library of Denmark and Copenhagen University Library)

Because of these studies the greatest efforts of the ophthalmologic community were concentrated on a search for the causes of increased axial length of the eye. This central tenet of the older school would eventually be challenged. Donders [1] and undoubtedly many others appreciated that axial length was not the sole determinant of refraction. Schnabel and Herrnheiser (1895) had found axial lengths varying from 22.25 to 26.24 mm in 35 emmetropic eyes and hypothesized that emmetropia was determined by the relation between the axial length and total refraction [28]. Ludwig Hein (1899) thought that myopia was due to elongation of the globe [8]. Steiger (1913), in a large statistical study of corneal power in children, de-emphasized the importance of axial length as the only determinant of refraction. His biomathematical study was large (5,000 children), but his experimental method was somewhat faulty in that he assumed lens power to be a constant and therefore calculated the axial length of the eye from total refraction in this manner [9]. The variability of lens power had been alluded to as early as 1575 by Maurolycus [5] and variations in lens thickness, refractive index, and position had been considered as possible causes of myopia prior to Donders' time [1]. In addition, actual lens power measurements, albeit in small samples, had been demonstrated by von Reuss (1887–1890), Awerbach (1900), and Zeeman (1911) to show considerable variations [7]. Steiger's corneal measurements gave a Gaussian curve extending from 39 D to 48 D [29]. He did not note any set value of corneal power in emmetropia. He further made a distribution curve of from +7 D to −7 D using his corneal values and then calculated axial lengths found in emmetropia (21.5–25.5 mm). Steiger viewed emmetropia and refractive errors as points on a normal distribution curve, with corneal power and axial lengths as free and independent variables [29]. Tscherning (1854–1939) was crucial in the understanding of optics in pathologic myopia [30], and he made many contributions in this area. In addition, he wrote a thesis about the frequency of myopia in Denmark [31]. Schnabel, Fuchs, Siegrest, and Elschnig were important to the study of histopathology in myopic eyes, especially in relation to optic nerve changes in pathologic myopia [9]. These concepts brought an entirely new approach to the study of myopia.

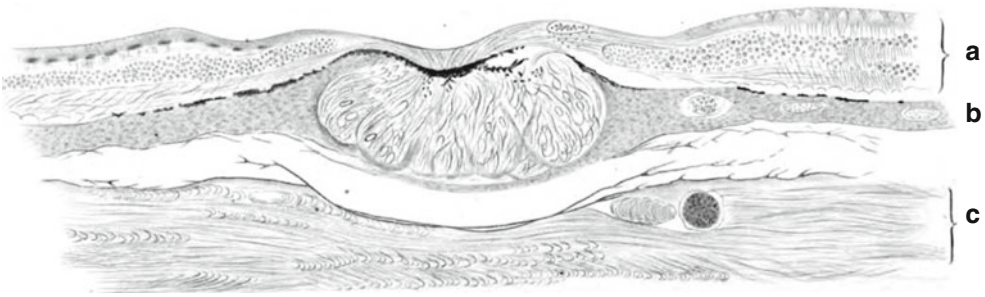

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Fig. 1.9 Cross section of the retina, choroid, and the sclera from a myopic eye shows a circumscript inclusion in the choroidal stroma which encroaches into the anterior layer of the choroid (Förster [21])

Fig. 1.10 Portrait of Ernst Fuchs. Original etching by Emil Orlik, 1910 (Reprinted with permission from the Medical University of Vienna, Austria)

Fig. 1.11 The earliest figure of "Fuchs' spot," which was described by Dr. Ernst Fuchs as "the central black spot in myopia" (Fuchs [23])

Tron (1934–1935) followed with a study of 275 eyes and carefully avoided the pitfalls of Steiger's work [32, 33]. In his study, the only optical element not measured directly was

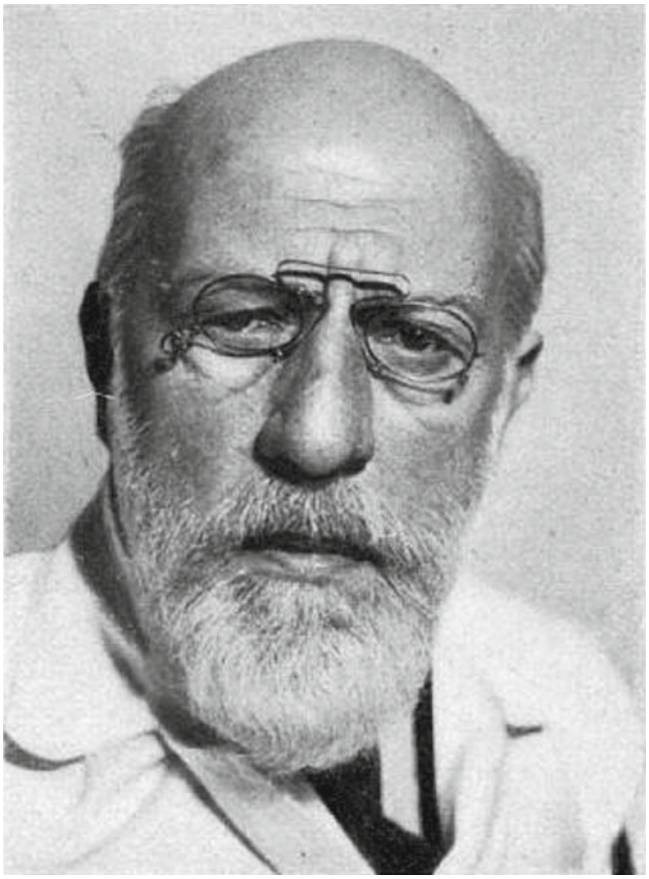

Fig. 1.12 Photograph of Maximilian Salzmann, MD (Reprinted with permission from The Royal Library, The National Library of Denmark and Copenhagen University Library)

axial length, which was calculated from the refraction, corneal power, lens power, and anterior chamber depth. Tron confirmed the wide range of axial lengths in emmetropia (22.4–27.3 mm) [32]. He also deduced that axial length was the determining factor for refraction only in the range beyond +4 D and −6 D [32]. He obtained essentially binomial curves for all the elements of refraction except for axial length. With the elimination of myopic eyes of more than 6 D, the curve for axial length also assumed a normal distribution [7]. Stenstrom (1946) [34] was able to directly measure axial length by using x-rays owing to the development of this technique by Rushton (1938) [35]. Stenstrom undertook a study of 1,000 right eyes and confirmed the results Tron had obtained in his smaller series. Both of these biometric studies found essentially normal distribution curves for corneal power, anterior chamber depth, lens power, and total refraction. Both also showed a peaking (excess) for axial length above the binomial curve as well as an extension of the limb toward increased axial length (skewness) [32, 34]. Stenstrom noted that the distribution curve of refraction had basically the same disposition as that of axial length, featuring both a positive excess at emmetropia and a skewness toward myopia [5].

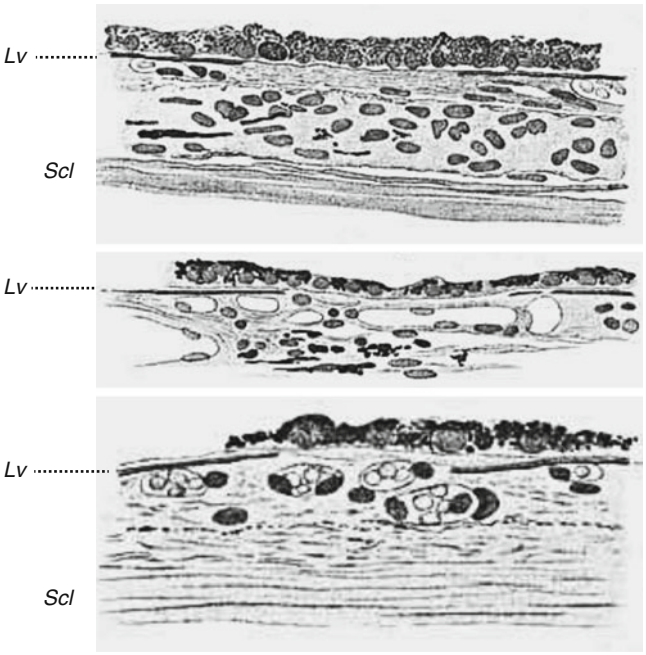

Fig. 1.13 *Top*: break in lamina of vitrea (*LV*) covered with epithelium. *Middle*: changes in epithelium. *Bottom*: break in lamina vitrea with epithelial covering and hyaline membrane. *Scl*: sclera (Salzmann [26])

This deviation in the population refraction curve had been noted previously by Scheerer and Betsch (1928–1929) [7], who had attributed this to the incorporation of eyes with crescent formation at the optic nerve. When these eyes were deleted from the data, a symmetric curve was obtained for the distribution of refraction. In the analysis of these data, it was pointed out that a positive excess still persisted in the "corrected" curve. Stenstrom's refractive curve [7] after the removal of eyes with crescent also demonstrated an excess. This central peaking was attributed to two factors: the first was the effect of the component correlation in the emmetropic range as postulated by Wibaut (1928) [5] and Berg (1931) [5], and the second was the direct effect of axial length distribution upon the curve of refraction [7]. Sorsby and co-workers (1957) [9] were later to confirm again the results of both Tron and Stenstrom and to further explore the variables in the correlations between the optical components in various refractions. This had been done to a limited extent by Berg [5]. Sorsby and co-workers demonstrated conclusively in their study of 341 eyes, the "emmetropization" effect was noted in distribution curves of refraction as a result of a correlation of corneal power and axial length. In ametropia +4 D and above, this correlation appeared to break down. Their study also indicated that neither the lens nor the chamber depth was an effective emmetropization factor [2]. Gernet (1965) proposed the use of ultrasound to measure the ocular axial length [36] after ultrasonography was pioneered in ophthalmology by Mundt and Hughes in 1956 [37].

In 1887, Adolf Eugen Fick submitted a very original paper entitled "Eine Contactbrille" (A contact spectacle) to the Archiv für Augenheilkunde. This was a report on his work, which led to the development of contact lenses. He published his paper in 1888 and coined the term "contact lens" [38]. Fick designed glass contact lenses to correct myopia and irregular astigmatism using lenses that were specially ground by Abbe, of Jena [39]. There were many early contact lens designs, but the first designed to allow circulation of tear film was made by Tuohy in 1948; this lens also was made of plastic. Contact lenses have proven to be useful by myopes.

1.4 Recent Historical Landmarks in Myopia

There are many recent contributors to the field of pathologic myopia. No body of work has influenced and inspired the eye care field more than the published comprehensive textbook on myopia in 1985 by Brian J. Curtin, MD [7] (Fig. 1.14): The Myopias: Basic Science and Clinical Management. It was becoming increasingly evident that pathological myopia represented an important cause for severe vision loss worldwide, particularly in selected racial populations. Curtin's textbook was an awakening on the importance of the disease. It was also an appeal to clinical scientists to accelerate and intensify their research to expand our knowledge of the related embryological, epidemiological, molecular, biological, genetic, and clinical aspects of pathological myopia, in hope of reducing the inherent visual disability. Curtin has many scientific contributions, and some of these will be briefly described here. Curtin and Karlin first used "lacquer cracks" and described the relationship between axial length and chorioretinal atrophy in 1970 [40]. In addition to this accomplishment, Klein and Curtin discovered that formation of lacquer cracks could cause subretinal hemorrhage in the absence of choroidal neovascularization in 1975 [41]. In 1977 Curtin created a classification scheme for staphyloma [42]. His textbook also emphasized the importance of the posterior staphyloma which was incriminated in the clinical manifestations associated with severe visual decline. In addition, Curtin helped to identify the optic nerve as an important cause of visual changes in myopia and described in detail the peripheral retinal myopic changes putting patients at risk for retinal detachment, early cataract formation, glaucoma, and a myriad of macular manifestations as adverse complications leading to severe vision loss in myopic patients [1].

The other important figure is Tokoro (Fig. 1.15), and some of his accomplishments will be mentioned here. Tokoro described the mechanism of axial elongation and

Fig. 1.14 Photograph of Brian Curtin, MD

Fig. 1.15 Photograph of Takashi Tokoro, MD

chorioretinal atrophy in high myopia [43]. In 1988, Tokoro defined pathologic myopia [44], and this definition has been used for many myopic studies. In 1998 Tokoro classified chorioretinal atrophy in the posterior pole in pathologic myopia as tessellated fundus, diffuse chorioretinal atrophy, small patch atrophy, and small macular hemorrhage [45].

Some other recent landmarks in myopia can be attributed to advanced technology and new treatment. Although fluorescein angiography (FA) is the main tool for diagnosing myopic choroidal neovascularization (CNV), indocyanine green angiography (ICGA) may better identify the CNV when large hemorrhages are present. ICGA also allows a better definition of lacquer cracks than FA [46, 47]. Optical coherence tomography (OCT) is a powerful real-time imaging modality. Since its introduction in ophthalmology, it has been utilized in understanding the ocular structure in many eye diseases. For example, in 1999, Takano and Kishi reported foveal retinoschisis and retinal detachment in severely myopic eyes with posterior staphyloma [48]. In 2002, Baba et al. first used OCT to demonstrate characteristic features at each stage of myopic CNV [49]. Spaide invented enhanced depth imaging spectral domain OCT to obtain images of choroid [50] and found thinner choroids in highly myopic eyes [51]. Ohno-Matsui et al. described intrachoroidal cavitation using swept-source OCT [52]. Ohno-Matsui and Moriyama have furthered our understanding of the shape of pathologically myopic eyes using high-resolution 3 D magnetic resonance images [53, 54]. Because of potential of visual loss from myopic CNV, several treatments have been tried, for example, thermal laser photocoagulation [55] and photodynamic therapy (PDT) with Visudyne [56]. In 2005, Nguyen et al. reported the effectiveness of bevacizumab in treating CNV secondary to pathologic myopia. After that, ophthalmologists started to use anti-vascular endothelium growth factor to treat myopic CNV. Many details of diagnosis and treatment for myopic patients will be mentioned in later chapters.

Acknowledgement: Dr. Brian J Curtin The early documentation of the history of myopia was based on his work. The update was incorporated in this perspective with his full consent.

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