Myopia: A Historical Perspective

1

Eugene Yu-Chuan Kang and Nan-Kai Wang

The word "myopia" is thought to be derived from New Latin, original Greek word "mŭopia" (μυωπία, from myein "to shut" + ops [gen. opos] "eye"), which means contracting or closing the eye. This is a description of the typical facial expression of the uncorrected myopia when a patient attempts to obtain clear distance vision. Before the introduction of spectacles, squinting the eyelids resulting in a horizontal stenopeic slit was the only practical way to achieve clearer distance vision. 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 myopia has been continually expanded.

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

E. Y.-C. Kang
Department of Ophthalmology, Chang Gung Memorial Hospital, Linkuo Medical Center, Kuei Shan, Taoyuan, Taiwan

N.-K. Wang (✉)
Department of Ophthalmology, Chang Gung Memorial Hospital, Linkuo Medical Center, Kuei Shan, Taoyuan, Taiwan

Department of Ophthalmology, Edward S. Harkness Eye Institute, Columbia University, New York, NY, USA

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 [1–6]. Aristotle (384–321 BC) was generally thought to be 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 myopia to blink the lids and write in small script [7]. Galen's (138–201 AD, Fig. 1.2) concepts 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, and the myopia had to wait a few more centuries before the introduction of minus lenses.

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 in myopia and flatter in hyperopia [9]. He did not mention the retina 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 seemed appropriate to address the subject because of his background in mathematics. In 1604, Kepler demonstrated 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, Kepler noted that parallel rays of light fell in front of

R. F. Spaide et al. (eds.), *Pathologic Myopia*, https://doi.org/10.1007/978-3-030-74334-5_1

Table 1.1 Historical landmarks in myopia

Year	Author	Description
384–321 BC	Aristotle	Difference between nearsighted and farsighted
138–201	Galen	First used the term "myopia" from the 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 the 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 Ritter 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-retinal pigment epithelium 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 the 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 pathologic myopia
1999	Morito Takano and Shoji Kishi	First illustrated foveal retinoschisis using optical coherence tomography (OCT) in eyes with posterior staphyloma
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 choroidal neovascularization using 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-magnetic resonance imaging (MRI) and intrachoroidal cavitation in pathologically myopic eyes using OCT
2013	Ohno-Matsui K.	Classification of posterior staphyloma based on MRI and wide-field fundus photos
2018	Panda-Jonas et al.	Hypothesis of myopization caused by production of Bruch's membrane

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

the retina in myopic eyes [11]. 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 aging, 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, and Boerhaave (1708) confirmed this lengthening and also reported the other cause of myopia: increased convexity of the refractive surfaces [12].

In the absence of the instruments necessary to measure corneal and lenticular variables, there were some studies confirming the variability of axial length. These included the studies of Morgagani (1761) [9], Guerin (1769) [9], Gendron (1770) [5], and Pichter (1790) [5]. Scarpa (Fig. 1.4 upper) is the first to describe anatomically posterior staphyloma (Fig. 1.4 lower) in two female eyes in 1801 [13]. He coined the Greek word "*staphylos*" which literally means "a bunch of grapes." It is of note that Scarpa described staphyloma but did not link it 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 a link either from posterior staphyloma to myopia [14].

1.2 Post-ophthalmoscopic Historical Landmarks in Myopia (1851)

Post-ophthalmoscopic development in myopia started from observations of the optic nerve, macula, and chorioretinal changes. Von Graefe (1854) first postulated the association between myopia and axial length in a combined ophthalmoscopic and anatomical study of two eyes measuring 29 mm and 30.5 mm in length [15]. However, it was Arlt's (1856) (Fig. 1.5) anatomical studies that convinced the scientific world of the intimate association of myopia with axial elon-

Fig. 1.4 Upper: Portrait of Antonio Scarpa; Lower: The earliest depiction of posterior staphyloma as contained in the text of Antonio Scarpa [13]

Fig. 1.5 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

gation of the globe at the expense of the posterior pole [16]. After Arlt made the connection between staphyloma and myopic refraction [16], clinical findings in pathologic myopia were investigated.

Von Jaeger (Fig. 1.6) was the first to describe and illustrate myopic conus and enlarged subarachnoid space around the nerve in 1861 [17]. 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 membrane which was exceedingly fine and delicately striated and contained a few vessels [17]. In 1862, Carl Friedrich Richard Förster (Fig. 1.7, upper) first observed sub-RPE choroidal neovascularization (CNV) (Fig. 1.7, lower) [18], and this is what we called "Forster spots." In 1901, Ernst Fuchs (Fig. 1.8, left) later discovered "central black spot in myopia" [19] (Fig. 1.8**,** right) [20], and this is what we called "Fuchs' spots." 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, but the etiology was obscure [12]. Henry Wilson described atrophy of choroidal epithelium in 1868 [21]. In 1902, Salzmann (Fig. 1.9, left) noted that cleft-shaped or branched defects were found in atrophic areas in the lamina vitrea that were concentric with the optic disc (Fig. 1.9, Right) [22, 23]. 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 [24].

Fig. 1.6 A portrait of Eduard Jäger von Jaxtthal by Adolf Dauthage in 1859

1.3 Modern Historical Landmarks in Myopia

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

The greatest efforts of the ophthalmologic community were concentrated on a search for the causes of increased axial length of the eye. Donders [1] appreciated that axial length was not the sole determinant of refraction. Schanbe and Harrneiser (1895) had found axial lengths varying from 22.25 to 26.24 mm in 35 emmetropic eyes and hypothesized that emmetropia could be determined by axial length and total refraction [25]. 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, deemphasized the importance of axial length as the only determinant of refraction. His biomathematical study was large (5000 children), but his experimental method was somewhat faulty in that he assumed lens power to be a constant and

Fig. 1.7 Upper: A portrait of Carl Friedrich Richard Förster. (Reprinted with permission from The Royal Library, The National Library of Denmark and Copenhagen University Library); Lower: Cross-section of the retina, choroid, and sclera from a myopic eye shows a circumscript inclusion in the choroidal stroma which encroaches into the anterior layer of the choroid. (Förster [18])

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 Donder's time [1]. In addition, actual lens power measurements, albeit in small samples, had been demonstrated by von Reuss (1887–1890), Awerbach (1900), and Zeeman

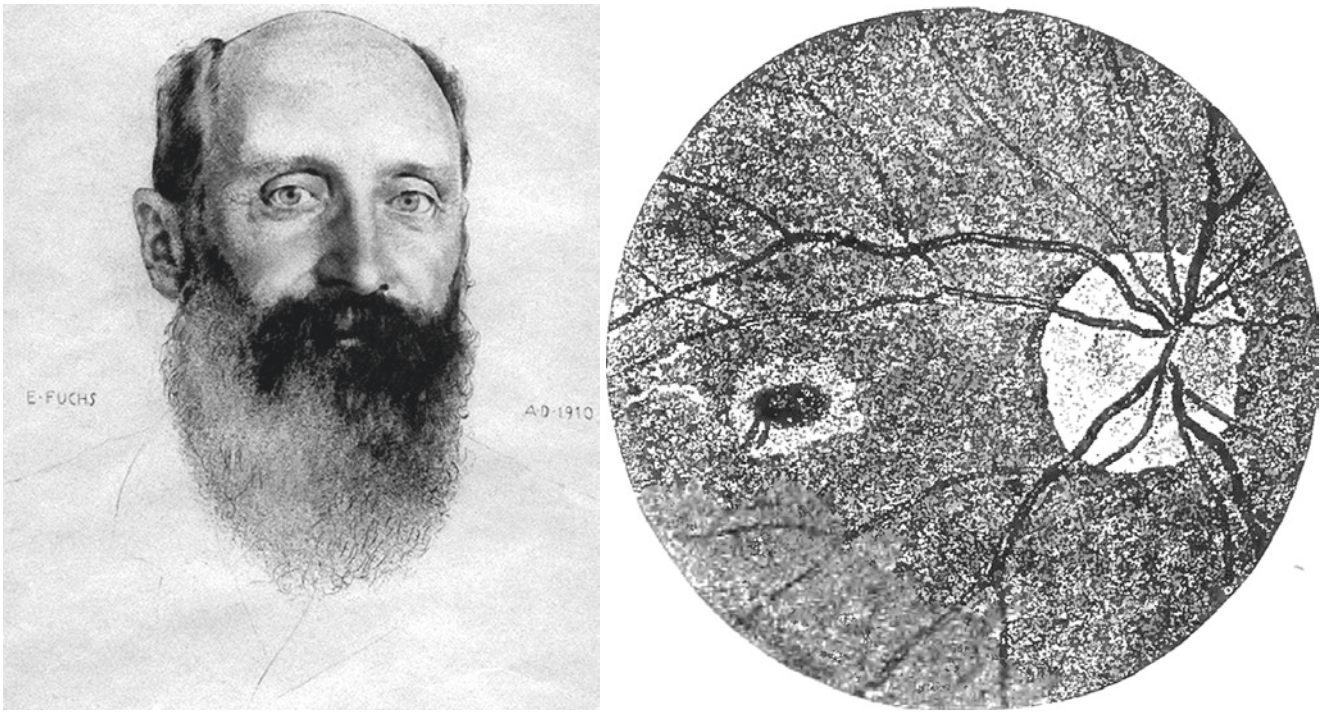

Fig. 1.8 Left: Portrait of Ernst Fuchs. Original etching by Emil Orlik, 1910. (Reprinted with permission from the Medical University of Vienna, Austria); Right: The earliest figure of "Fuchs' spot," which was described by Dr. Ernst Fuchs as "The central black spot in myopia." (Fuchs [20])

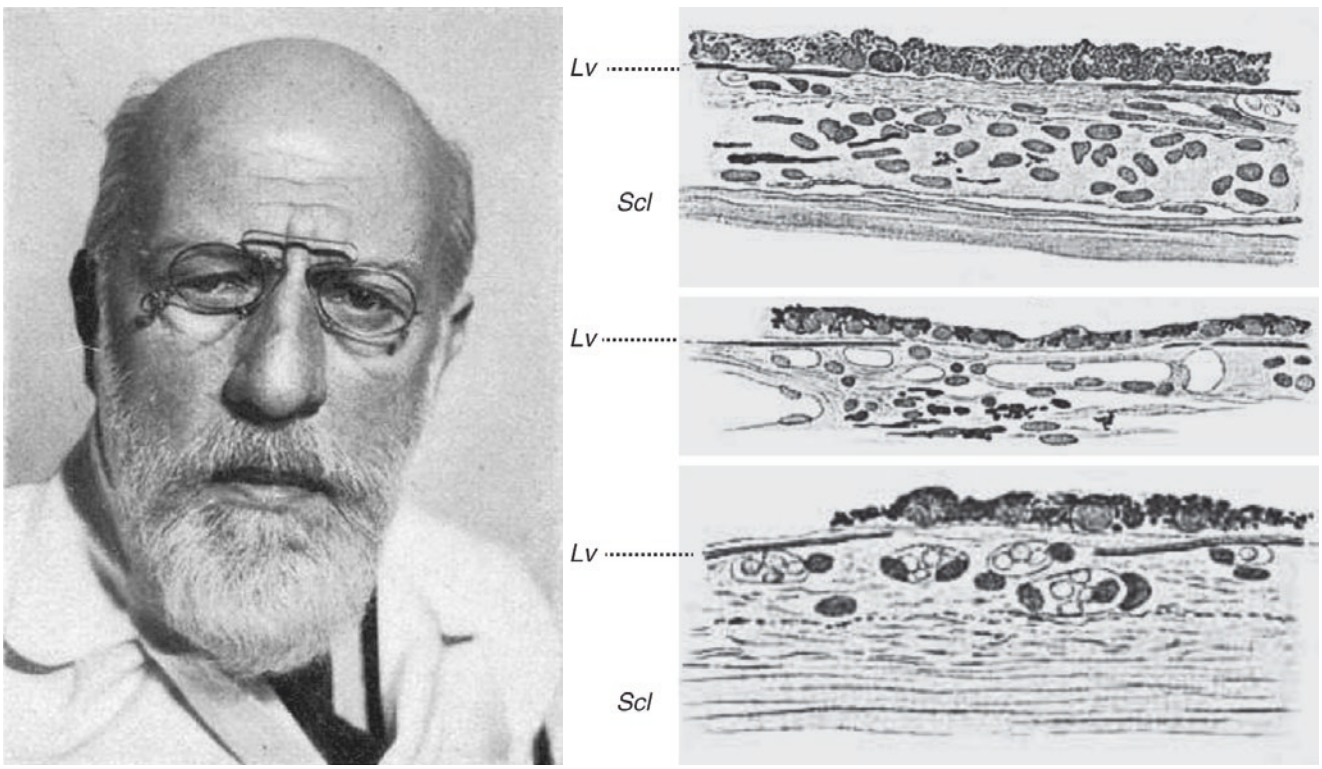

Fig. 1.9 Left: Photograph of Maximilian Salzmann, M.D. (Reprinted with permission from The Royal Library, The National Library of Denmark and Copenhagen University Library); Right: Top: Break in lamina of Bruch covered with the epithelium. Middle: Changes in the epithelium. Bottom: Break in lamina with epithelial covering and hyaline membrane. (Salzmann [23])

(1911) to show considerable variations [7]. Steiger's corneal measurements gave a Gaussian curve extending from 39 D to 48 D [26]. He did not note any set value of corneal power in emmetropia. He further made a distribution curve of +7 D to −7D using his corneal values and 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 [26]. Tscherning (1854–1939) was crucial in the understanding of optics in pathologic myopia [27], and he made many contributions in this area. In addition, he wrote a thesis about the frequency of myopia in Denmark [28]. Schnabel, Fuchs, Siegrest, and Elschnig were important to the studies 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.

Tron (1934–1935) followed with a study of 275 eyes and carefully avoided the pitfalls of Steiger's work [29, 30]. In his study, the only optical element not measured directly was 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) [29]. He also deduced that axial length was the determining factor for refraction only in the range beyond +4 D and −6 D [29]. 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) [31] directly measured axial length by using X-rays contributed by the development of this technique by Rushton (1938) [32]. Stenstrom studied 1000 right eyes and confirmed the results Tron had obtained in his smaller series. Both 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) [29, 31]. 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].

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 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 (1957) [9] later confirmed again the results of both Tron and Stenstrom and further explored 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 that 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 [33] after ultrasonography was pioneered in ophthalmology by Mundt and Hughes in 1956 [34].

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" [35]. Fick designed glass contact lenses to correct myopia and irregular astigmatism using lenses that were specially ground by Abbe, of Jena [36]. There were many early contact lens designs, but the first one to allow circulation of tear film was made by Tuohy in 1948; the lens was made by plastic.

1.4 Recent Historical Landmarks in Myopia

There are many recent contributors to pathologic myopia. No work has influenced and inspired the eye care field more than the published comprehensive textbook on myopia in 1985 by Brian J. Curtin, M.D. [7] (Fig. 1.10): *The Myopias: Basic Science and Clinical Management*. It increased the evidence that pathologic 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 and made clinical scientists to accelerate and intensify their research to expand our knowledge of the related embryological, epidemiological, molecular, biological, genetic, and clinical aspects of pathologic myopia. Curtin has many scientific contributions, and some of these will be briefly described here. Curtin and Karlin first used "lacquer cracks" and described the association between axial length and chorioretinal atrophy in 1970 [37]. In addi-

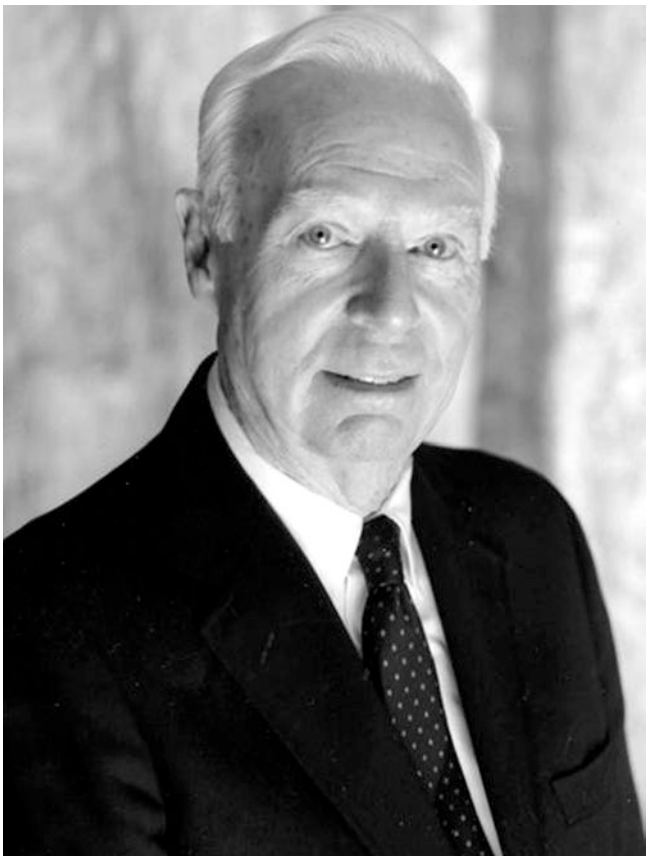

Fig. 1.10 Photograph of Brian Curtin, M.D.

Fig. 1.11 Photograph of Takashi Tokoro, M.D.

tion, Klein and Curtin discovered the formation of subretinal hemorrhage caused by lacquer cracks without choroidal neovascularization (CNV) in 1975 [38]. In 1977, Curtin created a classification scheme for staphyloma [39]. His textbook 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 the ocular changes putting myopic patients at risk for retinal detachment, early cataract formation, glaucoma, and a myriad of macular manifestations as its complications leading to severe vision loss [1].

The other important figure is Tokoro, (Fig. 1.11), and some of his accomplishments will be mentioned here. Tokoro described the mechanism of axial elongation and chorioretinal atrophy in high myopia [40]. In 1988, Tokoro defined pathologic myopia [41], which has been used for many myopic studies. Afterward, Tokoro classified chorioretinal atrophy in the posterior pole in pathologic myopia as tessellated fundus, diffuse chorioretinal atrophy, small patch atrophy, and small macular hemorrhage [42].

Some other recent landmarks in myopia were attributed to advanced technology and new treatments. Although fluorescein angiography (FA) is the main tool for diagnosing myopic 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 [43, 44]. Optical coherence tomography (OCT) is a powerful real-time imaging modality. Since its introduction, it has been utilized in understanding the ocular structure in many eye diseases. In 1999, Takano and Kishi reported foveal retinoschisis and retinal detachment in severely myopic eyes with posterior staphyloma [45]. Three years later, Baba et al. first used OCT to demonstrate characteristic features at each stage of myopic CNV [46]. As for other findings investigated using OCT, Spaide invented enhanced depth imaging spectral domain OCT to obtain images of choroid [47] and found thinner choroids in highly myopic eyes [48]. Excessive thinning of the choroid eventually leads to chorioretinal atrophy. Ohno-Matsui and Moriyama have furthered our understanding of the shape of pathologically myopic eyes and posterior staphyloma using high-resolution 3D magnetic resonance images and ultrawide-field fundus photos [49–51]. With the advent of swept-source OCT (SS-OCT), structural changes in myopic eyes could be studied more clearly. Ohno-Matsui et al. described intrachoroidal cavitation using SS-OCT [52]. Recently, Dr. Ohno-Matsui and her group used ultrawide-field SS-OCT and found that the sites of posterior staphyloma and myopic macular retinoschisis are spatially related to each other in high myopic eyes [53]. In 2017, Jonas et al. hypothesized that axial elongation is caused by production of Bruch's membrane in the retro-equatorial region, which plays an important role in myopization [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 endothelial growth factor to treat myopic CNV. Many details of diagnosis and treatment for myopic patients will be mentioned in later chapters.

Acknowledgment: 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.

References

1. Donders FC. On the anomalies of accommodation and refraction of the eye. London: The New Sydenham Society; 1864.
2. Sorby A, Benjamin B, Davey J, Sheridan M, Tauner J. Emmetropia and its aberrations. MRC special report series no 293. London: HMSO; 1957.
3. Alphen GWHMV. On emmetropia and ametropia. Basel, New York: S. Karger; 1961.
4. Blach RK. The nature of degenerative myopia: a clinico-pathological study. Master thesis. University of Cambridge; 1964.
5. Duke-Elder S. In: Duke-Elder S, editor. System of ophthalmology, vol. 1–15. St. Louis: Mosby; 1970.
6. Roberts J, Slaby D. Refraction status of youths 12–17 years, United States. Vital Health Stat. 1974;11(148):1–55.
7. Curtin BJ. The myopias: basic science and clinical management. Philadelphia: Harper & Row; 1985.
8. Wood CA. The American encyclopedia and dictionary of ophthalmology, vol. 11. Chicago: Cleveland Press; 1917.
9. Albert DM, Edwards DD. The history of ophthalmology. Cambridge, MA: Blackwell Science; 1996.
10. Kepler J. Ad Vitellionem Paralipomena (A Sequel to Witelo). C. Marnius & Heirs of J. Aubrius: Frankfurt; 1604.
11. Kepler J. Dioptrice. Augsburg; 1611.
12. Wood CA. The American encyclopedia and dictionary of ophthalmology, vol. 10. Chicago: Cleveland Press; 1917.
13. Scarpa A. Saggio di osservazioni e d'esperienze sulle principali malattie degli occhi. Pavia: Presso Baldessare Comino; 1801.
14. Ammon FAV. Histologie des Hydrophthalmus und des Staphyloma scleroticae posticum et laterale. Zeitschrift für die Ophthalmologie. 1832;2:247–56.
15. Graefe AV. Zwei Sektionsbefunde bei Sclerotico-chorioiditis posterior und Bemerkungen uber diese Krankheit. Arch Ophthalmol. 1854;1(1):390.
16. Arlt FV. Die Krankheiten des Auges. Prag Credner & Kleinbub; 1856.
17. Jaeger E. Ueber die Einstellungen des dioptrischen Apparates Im Menschlichen Auge. Kais. Kön. Hof- und Staatsdruckerei; 1861.
18. Förster R. Ophthalmologische Beiträge. Berlin: Enslin; 1862.
19. Fuchs E. Der centrale schwarze Fleck bei Myopie. Zeitschrift für Augenheilkunde. 1901;5:171–8.
20. Fuchs E. Text-book of ophthalmology. 5th ed. Philadelphia & London: Lippincott; 1917.
21. Wilson H. Lectures on the theory and practice of the ophthalmoscope. Dublin: Fannin & Co.; 1868.
22. Salzmann M. The choroidal changes in high myopia. Arch Ophthalmol. 1902;31:41–2.
23. Salzmann M. Die Atrophie der Aderhaut im kurzsichtigen Auge. Albrecht von Graefes Archiv fur Ophthalmologie. 1902;54:384.
24. Sym WG. Ophthalmic review: a record of ophthalmic science, vol. 21. London: Sherratt and Hughes; 1902.
25. Schnabel I, Herrnheiser I. Ueber Staphyloma Posticum, Conus und Myopie. Fischer's Medicinische Buchhandlung; 1895.
26. Steiger A. Die Entstehung der sphärischen Refraktionen des menschlichen Auges. Berlin: Karger; 1913.
27. Tscherning MHE. Physiologic optics: dioptrics of the eye, functions of the retina, ocular movements and binocular vision. Philadelphia: The Keystone Publishing Co.; 1920.
28. Norn M, Jensen OA. Marius Tscherning (1854–1939): his life and work in optical physiology. Acta Ophthalmol Scand. 2004;82(5):501–8.
29. Tron E. Uber die optischen Grundlagen der Ametropie. Albrecht Von Graefes Arch Ophthalmol. 1934;132:182–223.
30. Tron E. Ein Beitrag zur Frage der optischen Grundlagen der Anisound Isometropie. Albrecht Von Graefes Arch Ophthalmol. 1935;133:211–30.
31. Stenstrom SLHV. Untersuchungen über die Variation und Kovariation der optischen Elemente des menschlichen Auges. Uppsala: Appelbergs Boktr; 1946.
32. Rushton RH. The clinical measurement of the axial length of the living eye. Trans Ophthalmol Soc UK. 1938;58:136–42.
33. Gernet H. Biometrie des Auges mit Ultraschall. Klin Monatsbl Augenheilkd. 1965;146:863–74.
34. Mundt GH, W. Ultrasonics in ocular diagnosis. Am J Ophthalmol. 1956;41:488–98.
35. The "Kontaktbrille" of Adolf Eugen Fick; 1887.
36. Dor H. On contact lenses. Ophthal Rev. 1893;12(135):21–3.
37. Curtin BJ, Karlin DB. Axial length measurements and fundus changes of the myopic eye. I. The posterior fundus. Trans Am Ophthalmol Soc. 1970;68:312–34.
38. Klein RM, Curtin BJ. Lacquer crack lesions in pathologic myopia. Am J Ophthalmol. 1975;79(3):386–92.
39. Curtin BJ. The posterior staphyloma of pathologic myopia. Trans Am Ophthalmol Soc. 1977;75:67–86.
40. Tokoro T. Mechanism of axial elongation and chorioretinal atrophy in high myopia. Nippon Ganka Gakkai Zasshi. 1994;98(12):1213–37.
41. Tokoro T. On the definition of pathologic myopia in group studies. Acta Ophthalmol Suppl. 1988;185:107–8.
42. Tokoro T. Atlas of posterior fundus changes in pathologic myopia. 1st ed. Tokyo: Springer-Verlag; 1998. p. 5–22.
43. Brancato R, Trabucchi G, Introini U, Avanza P, Pece A. Indocyanine green angiography (ICGA) in pathological myopia. Eur J Ophthalmol. 1996;6(1):39–43.
44. Ohno-Matsui K, Morishima N, Ito M, Tokoro T. Indocyanine green angiographic findings of lacquer cracks in pathologic myopia. Jpn J Ophthalmol. 1998;42(4):293–9.
45. Takano M, Kishi S. Foveal retinoschisis and retinal detachment in severely myopic eyes with posterior staphyloma. Am J Ophthalmol. 1999;128(4):472–6.
46. Baba T, Ohno-Matsui K, Yoshida T, Yasuzumi K, Futagami S, Tokoro T, Mochizuki M. Optical coherence tomography of choroidal neovascularization in high myopia. Acta Ophthalmol Scand. 2002;80(1):82–7.
47. Charbel Issa P, Finger RP, Holz FG, Scholl HP. Multimodal imaging including spectral domain OCT and confocal near infrared reflectance for characterization of outer retinal pathology in pseudoxanthoma elasticum. Invest Ophthalmol Vis Sci. 2009;50(12):5913–8.
48. Fujiwara T, Imamura Y, Margolis R, Slakter JS, Spaide RF. Enhanced depth imaging optical coherence tomography of the choroid in highly myopic eyes. Am J Ophthalmol. 2009;148(3):445–50.
49. Ohno-Matsui K, Akiba M, Modegi T, Tomita M, Ishibashi T, Tokoro T, Moriyama M. Association between shape of sclera and

myopic retinochoroidal lesions in patients with pathologic myopia. Invest Ophthalmol Vis Sci. 2012;53(10):6046–61.
50. Moriyama M, Ohno-Matsui K, Modegi T, Kondo J, Takahashi Y, Tomita M, Tokoro T, Morita I. Quantitative analyses of high-resolution 3D MR images of highly myopic eyes to determine their shapes. Invest Ophthalmol Vis Sci. 2012;53(8):4510–8.
51. Ohno-Matsui K. Proposed classification of posterior staphylomas based on analyses of eye shape by three-dimensional magnetic resonance imaging and wide-field fundus imaging. Ophthalmology. 2014;121(9):1798–809.
52. Ohno-Matsui K, Akiba M, Moriyama M, Ishibashi T, Hirakata A, Tokoro T. Intrachoroidal cavitation in macular area of eyes with pathologic myopia. Am J Ophthalmol. 2012;154(2):382–93.
53. Shinohara K, Tanaka N, Jonas JB, Shimada N, Moriyama M, Yoshida T, Ohno-Matsui K. Ultrawide-field OCT to investigate relationships between myopic macular retinoschisis and posterior staphyloma. Ophthalmology. 2018;125(10):1575–86.
54. Jonas JB, Ohno-Matsui K, Jiang WJ, Panda-Jonas S. Bruch membrane and the mechanism of myopization: a new theory. Retina (Philadelphia, PA). 2017;37(8):1428–40.
55. Secretan M, Kuhn D, Soubrane G, Coscas G. Long-term visual outcome of choroidal neovascularization in pathologic myopia: natural history and laser treatment. Eur J Ophthalmol. 1997;7(4):307–16.
56. Verteporfin in Photodynamic Therapy Study Group. Photodynamic therapy of subfoveal choroidal neovascularization in pathologic myopia with verteporfin. 1-year results of a randomized clinical trial--VIP report no. 1. Ophthalmology. 2001;108(5):841–52.