

Refractive Error Characteristics and Influence on Ocular Parameters in Patients with Unilateral Congenital Ptosis

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Abstract

Background

The study aimed to investigate the difference of refractive status and ocular parameters between ptotic and fellow eyes in patients with unilateral congenital ptosis.

Methods

25 patients (52% males, age 22.04 ± 10.96) with unilateral congenital ptosis diagnosed and treated at the First Affiliated Hospital of Sun-yet Sen University were enrolled and underwent detailed refractive examination from March 2019 to November 2021. Ocular biometric measurements were performed by IOL Master 700 biometer. The difference of refractive error characteristics, best-corrected acuity (BCVA), as well as ocular parameters including axial length (AL), central corneal thickness (CCT), aqueous depth (AQD), anterior chamber depth (ACD), lens thickness (LT), and keratometry values between ptotic and fellow eyes were analyzed.

Results

Lower BCVA ($p=0.01$) and higher incidence of amblyopia ($p=0.018$) were observed in ptotic eyes. Among patients with amblyopia, the presence ($p=0.038$) and magnitude ($p=0.044$) of astigmatism were higher in ptotic eyes. The CCT of ptotic eyes was thicker than that of fellow eyes ($p \leq 0.001$). The keratometry values at the flat axis (K1), and mean corneal power (Km) were smaller (both $p \leq 0.001$). There was no statistical difference of AL between ptotic and fellow eyes.

Conclusions

Congenital ptosis influenced ocular parameters, mainly causing a thicker and flatter cornea. Patients with unilateral congenital ptosis might have lower BCVA in ptotic eyes, while higher amblyogenic astigmatism was observed.

Introduction

Congenital ptosis is a quite normal ocular abnormality caused by drooping eyelid within the first year of life, characterizing as a narrow palpebral fissure and more coverage by upper eyelid on cornea. Several studies have reported that congenital ptosis can increase the incidence of myopia, hyperopia, astigmatism, anisometropia and amblyopia.^[1, 2, 3, 4, 5] Previous studies have shown that pressure from upper eyelids may alter the corneal shape.^[6] In animal experiments, axial elongation induced by covering the pupil was observed.^[7, 8, 9] And it was also proved in human infants.^[10] However, under long-term pressure and coverage from upper eyelids, the effect of congenital ptosis on ocular structure remains unclear.

Recently, IOL Master 700 (Carl Zeiss AG, Jena, Germany), a more advanced, non-contact biometer, allows us to perform accurate ocular parameter testing on patients with congenital ptosis.^[11] It is based on the technique of swept-source optical coherence tomography (SS-OCT), providing fixation points for the patients and retinal OCT images for technicians when testing, so as to gain accurate axial measurements from posterior cornea to fovea.^[12] Besides, it also provides us with the measurements of central corneal thickness, lens thickness, anterior chamber depth, and corneal power.

In this study, we focus on both refractive status and the ocular biological parameters of unilateral congenital ptosis, in order to compare the refractive error between ptotic eyes and fellow eyes and to investigate the influences of congenital ptosis on the ocular structure during eyeball growth.

Materials And Methods

Patients

We enrolled 25 patients with unilateral congenital ptosis who were diagnosed and treated at the Ophthalmology Department of the First Affiliated Hospital of Sun Yat-sen University between March 2019 and November 2021. This study was approved by the Ethics Committee of the first Affiliated Hospital of Sun Yat-sen University in China ([2021]544) and complied with the tenets of the Declaration of Helsinki for biomedical research involving human subjects.

The normal position of the upper lid covers the cornea by less than 2mm, and ptosis can be diagnosed when the upper lid is below its normal position. The severity of the ptosis can be classified as mild (1–2 mm) when the upper lid does not cover the pupil, moderate (3–4 mm) when the upper lid covers partially the pupil and severe (more than 4 mm) when the pupil is completely covered by the lid.^[13]

The inclusion criteria included unilateral congenital ptosis older than 5 years, absence of other ocular pathology, patients older than five years, and reliable measurement results. The exclusion criteria were: acquired ptosis, patients younger than 5 years old unable to cooperate with the examination, a history of eye trauma, previous ocular surgery (including ptosis correction surgery), corneal opacity, wearing of eye contact lens in the last 3 months, and other ophthalmic or systemic disorders.

Ophthalmic examination

All the patients were examined by the same trained examiner. Routine ophthalmic examinations were performed in all patients, including best-corrected visual acuity measurements(BCVA) using snellen chart, detailed slit-lamp examination of the anterior segment and fundus examination with dilated pupil, and measurement of intraocular pressure (Topcon CT-80A, Tokyo, Japan). Complete evaluation of ptosis including measurement of the height of palpebral fissure (HPF), and levator functions (LF) was done by the lid excursion method, which was a measurement of the upper lid from extreme downgaze to extreme upgaze with the frontalis muscle fixed.

Refractive error examination

Cycloplegic refraction was done for all patients. Children younger than 10 years were given ointment atropine 1%, once a day for three days and were examined on the fourth day. Patients older than 10 years were examined following administration of combined 0.5% phenylephrine hydrochloride and 0.5% tropicamide eye drops, three times every 5 minutes. We calculated the refraction by spherical equivalent refraction (SER), and defined myopia as SER of at least $-0.50D$ and hyperopia as $+2.00D$ or more. Astigmatism was defined as $0.75DC$ or more. Anisometropia was defined as at least $1.00D$ difference compared with the opposite eye. We defined amblyopia as visual acuity of 6/9 or less and difference of more than two Snellen lines between the fellow and ptotic eyes excluding any other ocular abnormalities. [1, 14]

Ocular biometric measurements

Biometric measurements were performed using IOL Master 700 three times by the same well-trained technician before pupillary dilatation in all patients. Each time before the examination, the device was recalibrated. During the measurement, the examinees were asked to position their heads on a headrest and look at the fixation point for foveal scans. The poor-quality result would be deleted and remeasured until the readings were reliable.

The axial length (AL), central corneal thickness (CCT), aqueous depth (AQD), anterior chamber depth (ACD), lens thickness (LT), keratometry values at the flat (K1) and steep (K2) axis, and mean corneal power (Km) were obtained. AL was defined as the distance from the tear film to the retinal pigment epithelium (RPE) of the fovea. CCT was defined as the distance from the anterior to the posterior cornea. [15] AQD was measured using signals from the posterior face of the cornea to the anterior surface of the lens. The ACD was defined as the distance from the central corneal epithelium to the anterior surface of the lens and same as adding value CCT and AQD (CCT + AQD). [16] LT was defined as the distance between the anterior and posterior lens surfaces. Corneal power included K1, K2 and Km, which K1 was defined as the corneal power at the flat axis, and K2 at the steep axis, and Km was the average value of the K1 and K2. [17, 18]

Statistical analysis

Statistical analysis was performed by using SPSS version 22.0. Snellen VA was converted to a logarithm of the minimum angle of resolution (logMAR) value. Comparisons between the fellow and ptotic eyes were performed using Paired t tests. Chi-square tests were used to compare the frequency of refractive types between the fellow and ptotic eyes. A *P* value less than 0.05 was considered statistically significant.

Results

In total, 25 eyes of 25 patients with unilateral congenital ptosis in this study were analyzed. 25 fellow eyes without ptosis served as controls. The distribution of gender was equal of 13 (52%) males and 12 (48%) females. Their average age was $21.43 \pm 10.28y$, ranging from 5 to 42 years old. Out of 25 patients,

7 (28%) patients had mild ptosis, 13 (52%) had moderate ptosis, and 5 (20%) had severe ptosis. 12 (48%) had the right eye affected, while 13 (52%) the left. Table 1 shows the demographics of the patients.

Table 1
Demographic characteristics of participants
(N = 25)

Characteristics	
Age(year, Mean ± SD)	22.04 ± 10.96
Sex, n(%)	
Male	13(52%)
Female	12(48%)
Side of affected eye n(%)	
Right	12(48%)
Left	13(52%)
Severity of ptosis, n(%)	
Mild	7(28%)
Moderate	13(52%)
Severe	5(20%)

Abbreviation: SD=standard deviation

There was a significant difference in BCVA between ptotic and fellow eyes, that BCVA of ptotic eyes was lower (logMAR, Mean±SD, -0.059±0.107, p=0.01). Incidence of myopia, hyperopia and astigmatism did not differ significantly between ptotic eyes and fellow eyes. However, it was observed that the prevalence of amblyopia in the ptotic eyes (n (%), 5(20%), $\chi^2=5.556$, p=0.018) was higher than that in fellow eyes (n (%), 0). All 5 amblyopia occurred in the ptotic eyes, and 3 of them were severe ptosis while 2 were moderate. The results are shown in Table 2. What's more, as is summarized in Table 3, among the 5 patients with amblyopia, the incidence of astigmatism in ptotic eyes (n(%), 5(100%), $\chi^2=4.286$, p=0.038) is higher than fellow eyes (n(%), 2(40%)), and the magnitude of astigmatism is larger (D, Mean±SD, -0.35±0.69 v.s. -2.25±1.35, p=0.044).

Table 2. Difference of BCVA and refractive status between the fellow and ptotic eyes (N=25)

	Fellow eye	Ptotic eye	χ^2	P value
BCVA(logMAR, Mean±SD)	-0.006±0.046	-0.059±0.107		0.010*
DS (D), Mean±SD	-1.24±2.44	-1.56±2.30		0.181
DC (D), Mean±SD	-0.94±1.04	-0.57±0.61		0.098
SER (D), Mean±SD	-1.73±2.48	-1.86±2.43		0.576
Refractive type (number, %)				
Myopia	16(64%)	14(56%)	0.333	0.564
Hyperopia	0	2(8%)	2.083	0.149
Astigmatism	9(36%)	13(65%)	1.299	0.245
Amblyopia	0	5(20%)	5.556	0.018**

* Paired t test, P < 0.05 means statistically significant

** Chi-Square test, P < 0.05 means statistically significant

Abbreviation: BCVA=best-corrected visual acuity, DS=dioptr of spherical power, DC=dioptr of cylindrical power, SER=spherical equivalent refraction

Table 3. Difference of refractive status in patients with amblyopia between the fellow and ptotic eyes (N=5)

	Fellow eye	Ptotic eye	X2	P value
DS (D), Mean±SD	-0.05±1.16	0.40±2.66		0.550
DC (D), Mean±SD	-0.35±0.69	-2.25±1.35		0.044*
SER (D), Mean±SD	-0.20±1.32	-0.70±2.43		0.390
Refractive type (number, %)				
Myopia	2(40%)	1(20%)	0.476	0.490
Hyperopia	1(40%)	0	1.111	0.292
Astigmatism	5(100%)	2(40%)	4.286	0.038**

* Paired t test, P < 0.05 means statistically significant

** Chi-Square test, P < 0.05 means statistically significant

Abbreviation: DS=dioptr of spherical power, DC=dioptr of cylindrical power, SER=spherical equivalent refraction

Ocular biometric parameters in eyes with ptosis were compared with that of fellow normal eyes, which are summarized in Table 4. The CCT was greater in ptotic eyes than in fellow eyes ($541.72 \pm 28.48 \mu\text{m}$ v.s. $532.16 \pm 29.31 \mu\text{m}$, $p = 0.001$). And the K1 (42.01 ± 1.50 D v.s. 42.53 ± 1.43 D, $p = 0.001$) and SE (42.63 ± 1.54 D v.s. 43.04 ± 1.43 D, $p = 0.001$) of ptotic eyes were lower than that of fellow eyes, which means that the corneas of ptotic eyes were thicker and flatter. The parameters of AL, AQD, ACD, LT, K2 and IOP were not statistically different between the ptotic and fellow eyes.

Table 4. Comparison of ocular biometric parameters between ptotic and fellow eyes

	Ptotic eyes [mean \pm SD]	Fellow eyes [mean \pm SD]	P-value
IOP [mmHg]	15.10 \pm 3.69	15.28 \pm 2.73	0.813
AL [mm]	24.29 \pm 1.45	24.19 \pm 1.34	0.272
CCT [μm]	541.72 \pm 28.48	532.16 \pm 29.31	0.001*
AQD (mm)	2.87 \pm 0.30	2.86 \pm 0.27	0.548
ACD [mm]	3.40 \pm 0.27	3.40 \pm 0.31	0.930
LT [mm]	3.76 \pm 0.36	3.77 \pm 0.37	0.177
K1 [D]	42.01 \pm 1.50	42.53 \pm 1.43	0.001*
K2 [D]	43.29 \pm 1.78	43.57 \pm 1.51	0.083
Km [D]	42.63 \pm 1.54	43.04 \pm 1.43	0.001*

* Paired t test, $P < 0.05$ means statistically significant

Abbreviation: IOP=intraocular pressure, AL=axial length, CCT=central corneal thickness, AQD=aqueous depth, ACD=anterior chamber depth, LT=lens thickness, K1=Keratometry values at the flat axis, K2=Keratometry values at the steep axis, Km=Mean corneal power

Discussion

It's established that congenital ptosis may increase the incidence of refractive error including myopia, hyperopia, and astigmatism, as well as the incidence of amblyopia and anisometropia.^[1,19,20] Besides, due to amblyopia or deprivation caused by drooping eyelid covering the pupil, congenital ptosis may have a bad influence on visual acuity.^[21] Our study revealed that patients with congenital ptosis are more likely to suffer from visual impairment. Among the 25 patients with unilateral ptosis, the BCVA in ptotic eyes is lower than that in fellow normal eyes, which indicated that congenital ptosis may lead to visual defects.

In the present study, the incidence of amblyopia in patients with congenital ptosis is 20%, similar to previous reports.^[21,22,23] Besides, all cases of amblyopia in this study were ptotic eyes, revealing that in cases with unilateral congenital ptosis, amblyopia was found more often in ptotic eyes. When it comes to

the causes of amblyopia in patients with congenital ptosis, astigmatism made up to the largest proportion. Ugurbas et al observed that among patients with congenital ptosis, ptotic eyes had more asymmetric and irregular cornea, resulting in the increased magnitude of astigmatism, which may associate with higher incidence of amblyopia.^[3] Both reports individually by Huo et al and Paik et al found that in patients with congenital ptosis, ptotic eyes had a higher frequency of astigmatism and amblyopia.^[1,23] In our study, out of 5 cases with amblyopia, a higher incidence of astigmatism were found in ptotic eyes, and the magnitude of astigmatism in ptotic eyes was statistically larger than that in fellow eyes. These findings were in accordance with previous studies.

In normal human eyes, the upper eyelid covers 1-2 mm of the cornea while the lower eyelid covers zero. A previous study had shown that the superior cornea is thicker than the inferior cornea.^[24] Under the coverage and pressure of the upper eyelid, the superior cornea is chronically hypoxic. This can be explained by research that compared with the central and inferior cornea, the superior cornea demands more oxygen after lifting the upper eyelid.^[25,26] Consider this in mind, we presumed that chronic hypoxia induced by eyelids may cause a chronic corneal edema, leading to the thickening of the cornea. In the current study, thicker central cornea was found in ptotic eyes, which was consistent with the finding by LiX et al.^[27] We speculated that in congenital ptosis, ptotic eyes are covered more by the drooping eyelids and are subject to mechanical stress, bringing about chronic hypoxia of the cornea, and thus leading to a thicker cornea.

In our study, among 25 patients with unilateral congenital ptosis, no difference of axial length between ptotic eyes and fellow normal eyes was observed, which was consistent with the reports individually by von Noorden et al and Takahashi et al.^[28,29] One possible explanation is that by adjust head posturing, patients with congenital ptosis are able to offset the covering of upper eyelid, thus avoiding being occluded when looking. This indicated that in human eyes, the ocular axial elongation does not similar to the mechanism of that in animal model.^[7,8,9]

The previous study has shown that the pressure from the upper eyelids may have an influence on the shape of cornea, that the upper eyelid pressure may cause the cornea to become steep.^[30] In another research, they stated that at primary gaze the wider horizontal palpebral fissure, the flatter the cornea.^[31] However, in the current study, we found that both horizontal corneal power and mean corneal power of ptotic eyes are lower than that of fellow eyes, which indicated that congenital ptosis, with a narrower palpebral fissure and under more pressure from upper eyelid, may lead the cornea to become flatter.

Several limitations of this research need to be considered. First, the average age of these patients (21.43±10.28 years) is quite old, since people among these ages in China are mostly myopic. Maybe, different results of refractive errors and axial length could be found in cases during younger periods. The second limitation is that the number of cases included in this study was little although we had in advance calculated the minimum required sample size. But in such a small sample, parameters such as CCT were significantly different between ptotic and fellow eyes, which confirmed the reliability of the results. Future

research needs to be done to find out more abnormalities induced by congenital ptosis, providing more useful information in treatment. Third, ocular parameters in this study seem to be limited, more parameter testing and more accurate technique await us to apply in patients with congenital ptosis. At last, for well-controlled, only patients with unilateral congenital ptosis were enrolled, ocular changes caused by acquired ptosis remains unknown, therefore further studies are needed.

In summary, we confirmed that congenital ptosis may not only have an influence on refractive errors and raise the prevalence of amblyopia, but also cause the cornea to become thicker and flatter. The impact of congenital ptosis on axial length seems to be little in human beings.

Abbreviations

SS-OCT	Swept-source optical coherence tomography
BCVA	Best-corrected visual acuity
HPF	Height of palpebral fissure
LF	Levator functions
logMAR	logarithm of the minimum angle of resolution
SER	Spherical equivalent refraction
DS	Diopter of spherical power
DC	Diopter of cylindrical power
AL	Axial length
CCT	Central corneal thickness
AQD	Aqueous depth
ACD	Anterior chamber depth
LT	Lens thickness
K1	Keratometry values at the flat axis
K2	Keratometry values at the steep axis
Km	Mean corneal power
RPE	Retinal pigment epithelium

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the first Affiliated Hospital of Sun Yat-sen University in China ([2021]544) and conducted in compliance with Declaration of Helsinki.

Consent for publication

The written informed consent for publication was obtained from the participant.

Availability of data and materials

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

HLJ, and CTT designed the study; LYL, HJW, CYL and LWT collected data. LYL managed data and wrote the manuscript; CTT analyzed data; HLJ and CTT reviewed the manuscript. LYL and CTT share first authorship. All authors read and approved the final manuscript.

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References

1. Huo L, Cui D, Yang X, et al. A retrospective study: form-deprivation myopia in unilateral congenital ptosis[J]. *Clinical & experimental optometry*, 2012, 95(4): 404–409. doi: 10.1111/j.1444-0938.2012.00716.x.
2. Gusek-Schneider GC, Martus P. Stimulus deprivation myopia in human congenital ptosis: a preliminary report of 50 unilateral cases[J]. *Strabismus*, 2000, 8(3): 169–177. doi:
3. Uğurbaş SH, Zilelioğlu G. Corneal topography in patients with congenital ptosis[J]. *Eye (London, England)*, 1999, 13 (Pt 4): 550–554. doi: 10.1038/eye.1999.136.
4. Merriam WW, Ellis FD, Helveston EM. Congenital blepharoptosis, anisometropia, and amblyopia[J]. *American journal of ophthalmology*, 1980, 89(3): 401–407. doi: 10.1016/0002-9394(80)90011-2.
5. Griepentrog GJ, Diehl N, Mohny BG. Amblyopia in childhood eyelid ptosis[J]. *American journal of ophthalmology*, 2013, 155(6): 1125–1128.e1121. doi: 10.1016/j.ajo.2012.12.015.

6. Lieberman DM, Grierson JW. The lids influence on corneal shape[J]. *Cornea*, 2000, 19(3): 336–342. doi: 10.1097/00003226-200005000-00016.
7. Smith EL, 3rd, Bradley DV, Fernandes A, et al. Form deprivation myopia in adolescent monkeys[J]. *Optometry and vision science: official publication of the American Academy of Optometry*, 1999, 76(6): 428–432. doi: 10.1097/00006324-199906000-00023.
8. Troilo D, Nickla DL, Wildsoet CF. Form deprivation myopia in mature common marmosets (*Callithrix jacchus*)[J]. *Investigative ophthalmology & visual science*, 2000, 41(8): 2043–2049. doi:
9. Howlett MH, McFadden SA. Form-deprivation myopia in the guinea pig (*Cavia porcellus*)[J]. *Vision research*, 2006, 46(1–2): 267–283. doi: 10.1016/j.visres.2005.06.036.
10. Hoyt CS, Stone RD, Fromer C, et al. Monocular axial myopia associated with neonatal eyelid closure in human infants[J]. *American journal of ophthalmology*, 1981, 91(2): 197–200. doi: 10.1016/0002-9394(81)90173-2.
11. Huang J, Chen H, Li Y, et al. Comprehensive Comparison of Axial Length Measurement With Three Swept-Source OCT-Based Biometers and Partial Coherence Interferometry[J]. *Journal of refractive surgery (Thorofare, N.J.: 1995)*, 2019, 35(2): 115–120. doi: 10.3928/1081597x-20190109-01.
12. Yang JY, Kim HK, Kim SS. Axial length measurements: Comparison of a new swept-source optical coherence tomography-based biometer and partial coherence interferometry in myopia[J]. *Journal of cataract and refractive surgery*, 2017, 43(3): 328–332. doi: 10.1016/j.jcrs.2016.12.023.
13. Díaz-Manera J, Luna S, Roig C. Ocular ptosis: differential diagnosis and treatment[J]. *Current opinion in neurology*, 2018, 31(5): 618–627. doi: 10.1097/wco.0000000000000600.
14. Anderson RL, Baumgartner SA. Amblyopia in ptosis[J]. *Archives of ophthalmology (Chicago, Ill.: 1960)*, 1980, 98(6): 1068–1069. doi: 10.1001/archophth.1980.01020031058009.
15. Hughes RPJ, Read SA, Collins MJ, et al. Changes in ocular biometry during short-term accommodation in children[J]. *Ophthalmic & physiological optics: the journal of the British College of Ophthalmic Opticians (Optometrists)*, 2020, 40(5): 584–594. doi: 10.1111/opo.12711.
16. Fukuda S, Ueno Y, Fujita A, et al. Comparison of anterior segment and lens biometric measurements in patients with cataract[J]. *Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie*, 2020, 258(1): 137–146. doi: 10.1007/s00417-019-04482-0.
17. Akman A, Asena L, Güngör SG. Evaluation and comparison of the new swept source OCT-based IOLMaster 700 with the IOLMaster 500[J]. *The British journal of ophthalmology*, 2016, 100(9): 1201–1205. doi: 10.1136/bjophthalmol-2015-307779.
18. Asena L, Akman A, Güngör SG, et al. Comparison of Keratometry Obtained by a Swept Source OCT-Based Biometer with a Standard Optical Biometer and Scheimpflug Imaging[J]. *Current eye research*, 2018, 43(7): 882–888. doi: 10.1080/02713683.2018.1458881.
19. Gusek-Schneider GC, Martus P. Stimulus deprivation myopia in human congenital ptosis: a study of 95 patients[J]. *Journal of pediatric ophthalmology and strabismus*, 2001, 38(6): 340–348. doi:

20. Srinagesh V, Simon JW, Meyer DR, et al. The association of refractive error, strabismus, and amblyopia with congenital ptosis[J]. *Journal of AAPOS: the official publication of the American Association for Pediatric Ophthalmology and Strabismus*, 2011, 15(6): 541–544. doi: 10.1016/j.jaapos.2011.08.006.
21. Ural O, Mocan MC, Erdener U. Evaluation of All Causes of Visual Function Loss in Children With Congenital Blepharoptosis[J]. *Journal of pediatric ophthalmology and strabismus*, 2020, 57(2): 97–102. doi: 10.3928/01913913-20200120-01.
22. Dray JP, Leibovitch I. Congenital ptosis and amblyopia: a retrospective study of 130 cases[J]. *Journal of pediatric ophthalmology and strabismus*, 2002, 39(4): 222–225. doi:
23. Paik JS, Kim SA, Park SH, et al. Refractive error characteristics in patients with congenital blepharoptosis before and after ptosis repair surgery[J]. *BMC ophthalmology*, 2016, 16(1): 177. doi: 10.1186/s12886-016-0351-9.
24. Haque S, Jones L, Simpson T. Thickness mapping of the cornea and epithelium using optical coherence tomography[J]. *Optometry and vision science: official publication of the American Academy of Optometry*, 2008, 85(10): E963-976. doi: 10.1097/OPX.0b013e318188892c.
25. Benjamin WJ, Hill RM. Human cornea: superior and central oxygen demands[J]. *Graefe's archive for clinical and experimental ophthalmology = Albrecht von Graefes Archiv fur klinische und experimentelle Ophthalmologie*, 1988, 226(1): 41–44. doi: 10.1007/bf02172716.
26. Benjamin WJ, Ruben CM. Human corneal oxygen demands at superior, central, and inferior sites[J]. *Journal of the American Optometric Association*, 1995, 66(7): 423–428. doi:
27. Li X, Liu C, Mao Z, et al. Effect of congenital blepharoptosis on corneal biomechanical properties and changes after ptosis surgery[J]. *Eye (London, England)*, 2020, 34(6): 1055–1062. doi: 10.1038/s41433-019-0586-9.
28. von Noorden GK, Lewis RA. Ocular axial length in unilateral congenital cataracts and blepharoptosis[J]. *Investigative ophthalmology & visual science*, 1987, 28(4): 750–752. doi:
29. Takahashi Y, Kang H, Kakizaki H. Axial Globe Length in Congenital Ptosis[J]. *Journal of pediatric ophthalmology and strabismus*, 2015, 52(3): 177–182. doi: 10.3928/01913913-20150326-11.
30. Read SA, Collins MJ, Carney LG. A review of astigmatism and its possible genesis[J]. *Clinical & experimental optometry*, 2007, 90(1): 5–19. doi: 10.1111/j.1444-0938.2007.00112.x.
31. Read SA, Collins MJ, Carney LG. The influence of eyelid morphology on normal corneal shape[J]. *Investigative ophthalmology & visual science*, 2007, 48(1): 112–119. doi: 10.1167/iovs.06-0675.

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