RESEARCH

Intraocular pressure variation from ocular compression in low and high myopia

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Running title
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Abstract

Clinical relevance

Change in intraocular pressure during acute ocular compression is related to aqueous humour dynamics. Monitoring IOP change throughout ocular compression has potential to evaluate aqueous outflow facilities.

Background

Recent studies have monitored lamina cribrosa deformation using optical coherence tomography during ocular compression. Intraocular pressure (IOP) was measured only once immediately after ocular compression. This study aimed to evaluate IOP changes during and after ocular compression and compare the differences between low and high myopia.

Methods

Two groups of young, healthy adults were age-matched and underwent ocular compression. IOP was measured at baseline and monitored during a 2-min ocular compression followed by a 10-min recovery phase. Rebound tonometry was used and applied at 30-s intervals.

Results

Thirty low and 30 high myopes (60 right eyes) were included in the study. They had similar baseline IOP at 14.9mmHg. IOP was elevated to 21.7 $\pm$ 3.8mmHg and 22.3 $\pm$ 4.2mmHg for the low and high myopic group, respectively ($p = 0.877$). Low myopes had faster IOP decay during ocular compression at -3.24mmHg/min than high myopes at -2.58mmHg/min ($p = 0.0528$). The IOP dropped below the baseline level after the release of the compressive force. Low myopes had IOP that returned to baseline levels faster (at 360 s) than high myopes (at 510 s).

Conclusion

Measuring IOP once immediately after ocular compression could under-estimate the effect of IOP elevation during ocular compression. Further studies are required regarding IOP changes from ocular compression in aqueous humour dynamics.

Introduction

Myopia is a known risk factor for high-pressure glaucoma.[1-3] Lamina cribrosa (LC) is the site of glaucoma damage.[4,5] Studies on LC can enrich the understanding of the pathophysiology of glaucoma. With the development of spectral-domain optical coherence tomography (SD-OCT), the morphology of LC can be studied, including the anterior and posterior LC surfaces, as well as its thickness. Acute intraocular pressure (IOP) elevation through ocular compression is commonly used to study the biomechanical properties of the LC in glaucoma research.[6-9] Any morphological changes in the LC were monitored before and during ocular compression. Previous studies have measured the IOP immediately after ocular compression only. Investigators assumed IOP to be constant during ocular compression.[8,9]

The recent pilot study demonstrated an IOP drop during a 1-min ocular compression. IOP recovery after ocular compression took longer than 5 min (Lam, AKC & Xu, FY. IOVS 2020;61:ARVO E-Abstract 4627). Chen et al.[10] applied ocular compression and used rebound tonometry to monitor IOP change. They found IOP drop in the first few minutes which was related to changes of Schlemm's canal dimensions. Both IOP drop and recovery should be related to aqueous humour dynamics, which requires further investigation.

Since high myopia is a risk factor for primary open-angle glaucoma (POAG), the evaluation of LC has important implications for understanding the correlation between high myopia and POAG.[11] It is inappropriate to assume a constant IOP when the LC is scanned using OCT during ocular compression, especially when OCT scanning lasts for ≥2 min.[9] In the current study, IOP was monitored during a 2-min ocular compression, and the IOP recovery phase was monitored after the release of the compressive force. It was speculated that there was a continuous drop in IOP during ocular compression. The current study further investigated whether IOP variation could be different between eyes with low and high myopia.

Methods

Healthy low and high myopes were recruited from the university campus by hanging posters at various places. Written informed consent was obtained from each participant before ophthalmic assessment was initiated. The present study adhered to the tenets of the Declaration of Helsinki and was approved by the Institutional Review Board of The Hong Kong Polytechnic University (reference: HSEARS20200211003).

Low myopes should have spherical equivalent (sphere plus half the amount of negative cylinder) ≤ -0.50D up to -3.00D. High myopes had spherical equivalent ≤ -6.00D. Distance visual acuity was at least logMAR 0.00 in each eye. Participants with history of ocular disease, ocular trauma or surgery, who were currently taking any medication, were excluded. Participant with an IOP ≥ 21mmHg at baseline were also excluded. The workflow included rebound tonometry at baseline, followed by acute IOP elevation for 2 min through ocular compression. Tonometry was conducted at 30-s intervals during ocular compression and during a 10-min recovery phase. Only the right eye was used in this experiment.

<u>Ocular compression</u>

A phosphene pressure tonometer (Proview) was used to increase the IOP. The Proview was designed for patients to monitor IOP by themselves.[12] The spring compression device consists of a spring-loaded plunger with a flat applicator. The Proview was modified with an extension long enough for easy ocular compression (Figure 1). To ensure similar force was applied to each subject: 1) the modified Proview was calibrated which showed linear relationship between the force provided at different scales between 14 and 40 (Figure 2); 2) an electronic circuit was added to the modified Proview. A beep tone was given when compression reached the pre-set scale (that was 20,

equivalent to around 47g); 3) another beep tone at higher pitch was given when the force exceeded 47g, at around 48g, to remind examiner too strong the force was applied; 4) the examiner was well trained to keep the scale reaching the pre-set level for ocular compression. Thus, the force created should be correct and could be maintained consistently. Previous studies applied 30g,[13] 40g,[14] or up to 95g forces[6,7] using ophthalmodynamometry. Ocular compression was performed by pressing the lower eyelid at the temporal side, with the device held perpendicular to the eyeball as much as possible. Figure 3 shows the mounting of the Proview at a chin-rest while the subject maintained fixation on a distant object. A rebound tonometer was mounted in front of the right eye.

[Figure 1]

[Figure 2]

[Figure 3]

Monitoring of IOP

Baseline IOP was measured using a rebound tonometer (iCare TA01i, Tiolat, Helsinki, Finland) at 30-s intervals for 1 min. Each measurement set consisted of six consecutive readings. An average was generated automatically, with an error bar indicating the variation within the six readings. Whenever the six readings were significantly different, a new set was obtained to ensure accuracy. The baseline IOP (B-IOP) before ocular compression was the average of three measurements within 1 min. IOP during and after ocular compression was also monitored at 30-s intervals. For 2 min of ocular compression, there were five IOP measurements: C-0, C-30, C-60-, C-90, and C-120. During a 10-min recovery phase, 21 measurements were made: R-0 to R-600.

Statistical analysis

Test-retest repeatability (TRR) of rebound tonometry was calculated as 2.77 times within-subject standard deviation (S_W) from the three IOP measurements before ocular compression. The difference between B-IOP and C-0 was calculated. It should be greater than 2.77 S_W or the subject was excluded to ensure significant IOP elevation from ocular compression. The IOP change for the 2-min ocular compression was fitted using linear regression. IOP during the recovery phase (R-0 to R-600) was compared with B-IOP using parametric or non-parametric analysis whenever appropriate to determine the time point at which IOP returned to the baseline level. The IOP dynamics of the two refractive groups were compared. The data of high myopes was normal distributed, while data of low myopes was skewed. Therefore, for pairwise comparisons, repeated-measures analysis of variance (RMANOVA) and Friedman test with Bonferroni correction post hoc tests were used for high myopes and low myopes, respectively.

Results

The TRR of rebound tonometry at the baseline was 2.4mmHg. Thirty low myopes and 30 high myopes were recruited and all of them had IOP elevation at C-0 greater than 2.4mmHg when compared with the B-IOP. Table 1 shows the demographic characteristics of the two groups. There was no significant difference in gender distribution between the two groups (Chi-square, $p = 0.260$). The low and high myopic groups different axial lengths (24.4mm and 26.6mm, respectively, Mann-Whitney U test, $p < 0.001$), but had similar age (23.4 years and 24.3 years, respectively, Mann-Whitney U test, $p = 0.153$), and baseline IOP (14.9mmHg and 14.9mmHg, respectively, unpaired t-test, $p = 0.988$). The immediate IOP elevation (C-0 minus B-IOP) was similar between the low and high myopic groups (10.3mmHg and 10.2mmHg, unpaired t-test, $p = 0.960$). Figure 4 shows the IOP at each time point from the baseline, during ocular compression, and during the recovery phase. During ocular compression, low myopes demonstrated an IOP decline of -3.24mmHg/min (95% confidence interval, -3.84 to -2.64mmHg/min). High myopes had a slower IOP decline rate at -

2.58mmHg/min (95% confidence interval, -3.24 to -1.98mmHg/min). The difference in the slopes was close to significant (t=-2.407, p = 0.0528). It took 360 s (post-hoc test after Friedman test) for low myopes to have IOP sustainably returned to baseline levels after ocular compression, while high myopes spent 510 s (post hoc test after repeated measures analysis of variance).

[Table 1]

[Figure 4]

Discussion

This study found that IOP was not stable during ocular compression. It is incorrect to assume a stable IOP when performing OCT during ocular compression.[6,9] To better estimate the IOP increase during OCT, investigators should measure IOP again at the end of OCT because this IOP is lower. The average IOP values from these two measurements (initial and final) could provide better IOP estimation. For example, if IOP is measured as 50mmHg immediately after ocular compression, IOP is 40mmHg at the end of OCT. Using an average of these two values, that is 45mmHg, could better relate the compression effect on the LC. In this example, deformation of the LC could have already been achieved at 45mmHg. Relying solely on one IOP measurement at the initial stage (i.e. 50mmHg) would under-estimate the effect of IOP on LC deformation. There is one unknown factor which is recovery rate of the lamina cribrosa at different IOP levels. It may happen that greater LC deformation at 50mmHg also requires a longer recovery time than a deformation at 40mmHg.

During the course of this project, Chen et al.[10] reported a decline in IOP during a 4-min ocular compression. The dimensions of the Schlemm's canal were monitored using SD-OCT. They demonstrated collapse of the Schlemm's canal hence a rise in IOP. The Schlemm's canal cross-sectional area was reduced from 5440.0μm² at baseline to 3947.6μm² at 38.6mmHg during ocular compression. The collapse of the Schlemm's canal was positively associated with reduced aqueous outflow facilities. There was dilation of the Schlemm's canal hence a drop in IOP. There was no significant difference in the trabecular meshwork thickness. In the current study, a chin-rest was used to keep subject's head position steady. The modified Proview was mounted firmly to ensure a consistent compressive force applied through the lower eyelid. There were two beep tones at the pre-set and above the pre-set scales, respectively. Such modification could be better than previous studies just relied on the scale of the ophthalmodynamometer.

The current study found a faster IOP drop during ocular compression in low myopes than in high myopes. The differences approached statistical significance. It is postulated that the two groups had different dimensions of the Schlemm's canal and trabecular meshwork, hence, different aqueous outflow during ocular compression. Chen et al.[15] measured Schlemm's canal and trabecular meshwork of high myopes. They suggested that elongation of eyeball may change structures of the Schlemm's canal and alter its biomechanical properties. Qi et al.[16] also reported that highly myopic eyes had smaller Schlemm's canal diameter and area, smaller trabecular meshwork thickness and width. Aqueous outflow through the trabecular meshwork and the Schlemm's canal during ocular compression could be different in the two groups. Pulsatile aqueous outflow is the flow of aqueous from the Schlemm's canal to the episcleral veins.[17] Johnstone[18] suggested that IOP rise induced by ocular compression through ophthalmodynamometry or the water drinking test was regulated by pulsatile aqueous outflow. From the current study, it appears that low myopes have better regulation of aqueous outflow and IOP during ocular compression. Nevertheless, the postulation of different aqueous outflow of the two groups during ocular compression requires further studies to confirm.

When the compressive force was released, the IOP dropped below the baseline level and gradually returned to the baseline IOP. Chen et al.[10] monitored Schlemm's canal area and IOP during a 4-minute ocular compression using ophthalmodynamometry. At the second minute of ocular compression, Schlemm's canal area was still smaller than its original size, and IOP had not returned to the baseline level. At the 4th minute of ocular compression, both parameters returned to their baseline levels. In the current study, compressive force was released after two minutes. A sudden release of the compressive force resulted in expansion of the globe. This might create a negative pressure, hence, IOP was lower than the baseline level. Iwase et al.[19] had similar finding about dropping of IOP below the baseline level after release of ocular compression. Johnstone[18] suggested that pulsatile aqueous flow could stop when the IOP is reduced below its physiological point. It took some time to balance the aqueous humour dynamics. In the recovery phase, lower myopes had a faster recovery rate than high myopes, which may also indicate different aqueous humour dynamics of the two groups.

It would be interesting to observe different IOP profiles between the two myopic groups. A recent study demonstrated genetic influences between high myopia and POAG as well as the causal effect of myopia on POAG.[20] The study of aqueous humour dynamics would be useful to better understand the pathophysiology of POAG in patients with high myopia. If high myope has poor IOP regulation, it could be a risk factor for the development of glaucoma.

Can IOP variation during and after ocular compression be used as an indicator of aqueous outflow facilities? Aqueous outflow facilities are not routinely measured in clinical practice. It requires placing a weighted tonometer on the eye, such as the Schiötz tonometer. A pneumatonometer is also used and rate of IOP recovery is measured for several minutes while maintaining the pneumatonometer on the eye.[21] The aqueous outflow facility coefficient C can be determined using the following equation:

$$F = (P_{IOP} - P_{EPI}) \times C + F_{UVEO}$$

where F is the aqueous perfusion rate, P_{IOP} is the IOP, P_{EPI} is the episcleral venous pressure, and F_{UVEO} is the uveoscleral outflow. Aqueous perfusion rate demonstrated diurnal variation and also an age-related reduction.[22]

Clinically, the water drinking test may indicate outflow facility.[23] Fluorophotometry is a more common method for measuring outflow facilities. A high dose of fluorescein is applied topically at mid-night, followed by continuous fluorophotometric measurements the following morning.[24,25] This method is still used in research work nowadays but is difficult to conduct routinely in clinical practice.[26,27] A more invasive method for measuring outflow requires a manometric tonographic technique. Karyotakis et al.[28] found that the outflow coefficient decreased with a high IOP. The aqueous outflow facility coefficient at 20mmHg was almost four-fold more than that at 40mmHg. Further study should evaluate the usefulness of ocular compression and IOP changes in indicating aqueous outflow facilities.

This study has several limitations. Due to variations of lower eyelid morphology, in particular fatty tissues beneath the eyelid, it is hard to apply the modified Proview at a specific location to generate an identical compressive force for all subjects. Figure 3 shows how ocular compression was applied to a subject. Since the two groups had similar baseline IOP and could reach similar IOP rise immediately at ocular compression, similar forces should be provided through ocular compression in the two groups. The sample size was small and only healthy young adults were included. Older adults particularly POAG patients before and after treatments should be recruited in future study. Rebound tonometer was used in this study which is not gold standard in IOP measurement. Both rebound tonometry [10,14,19,29] and tonopen [6,30,31] have been used in ocular compression studies previously. We limited the IOP elevation within 40mmHg since rebound tonometry did not correlate

well with Goldmann tonometry at high IOP.[32] Measurement of the Schlemm's canal was not included and the change of Schlemm's canal dimension and IOP variation was based on the previous study by Chen et al.[10] Eyes with high myopia might also have different morphologic characteristics and there was no measurement of anterior segment dimensions such as anterior chamber depth, scleral thickness, etc.

To conclude, measuring IOP once immediately after ocular compression could under-estimate the effect of IOP elevation during ocular compression. Further studies are required regarding IOP changes from ocular compression in aqueous humour dynamics.

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Author contributions

F.X. and A.K.C.L designed the study, F.X. performed the experiment, F.X. and A.K.C.L analysed the data, F.X. and A.K.C.L wrote and edited the manuscript.

Data availability

Datasets used or analysed during the present study are available from the corresponding author on reasonable request.

Conflict of Interest

The authors have nothing to disclose.

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Table 1. Baseline information of the two refractive groups.

Parameters	Low myopes	High myopes	p-value
Age (years)	23.4 ± 3.0 Range: 20 to 30	23.9 ± 2.8 Range: 20 to 30	0.153*
Gender: female / male	19 / 11	23 / 7	0.260**
Spherical equivalent (diopter)	-1.74 ± 0.82 Range: -0.50 to -2.94	-7.89 ± 1.39 Range: -6.00 to -11.50	< 0.001*
Baseline IOP (mmHg)	14.9 ± 2.8 Range: 9.3 to 20.3	14.9 ± 2.8 Range: 9.0 to 21.3	0.988#
Compression IOP (mmHg)	21.7 ± 3.8 Range: 14.8 to 28.4	22.3 ± 4.2 Range: 16.6 to 35.8	0.877*
Axial length (mm)	24.4 ± 0.9 Range: 22.3 to 26.1	26.6 ± 1.1 Range: 24.8 to 29.3	< 0.001*

IOP: intraocular pressure

*Mann-Whitney test

** Chi-square test

#unpaired t-test

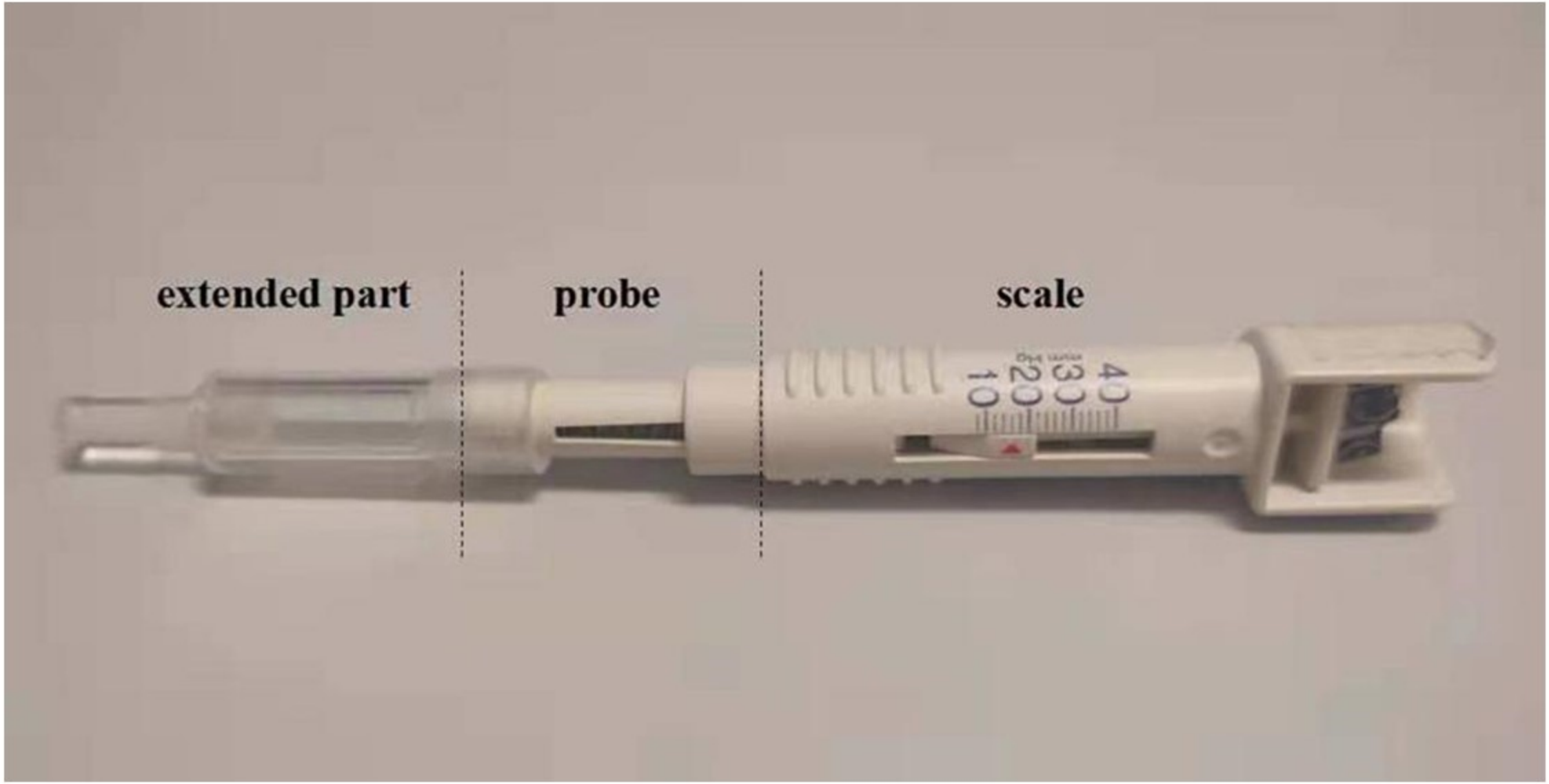
Figure captions

Figure 1. Modification of the Proview phosphene pressure tonometer.

Figure 2. Calibration of the Proview eye pressure monitor. Error bars indicate standard deviations.

Figure 3. The modified Proview is mounted at a chin-rest. A rebound tonometer is placed in front of the right eye for measurement of intraocular pressure.

Figure 4. Intraocular pressure profiles of the two refractive groups at baseline, during ocular compression and after ocular compression. The data were plotted as mean and standard error (error bar).
Low myopes: blue dots and line; high myopes: red dots and line; black horizontal dotted line: baseline IOP of 14.9mmHg; black vertical lines: different stages of IOP
B: baseline
C: compression
R: recovery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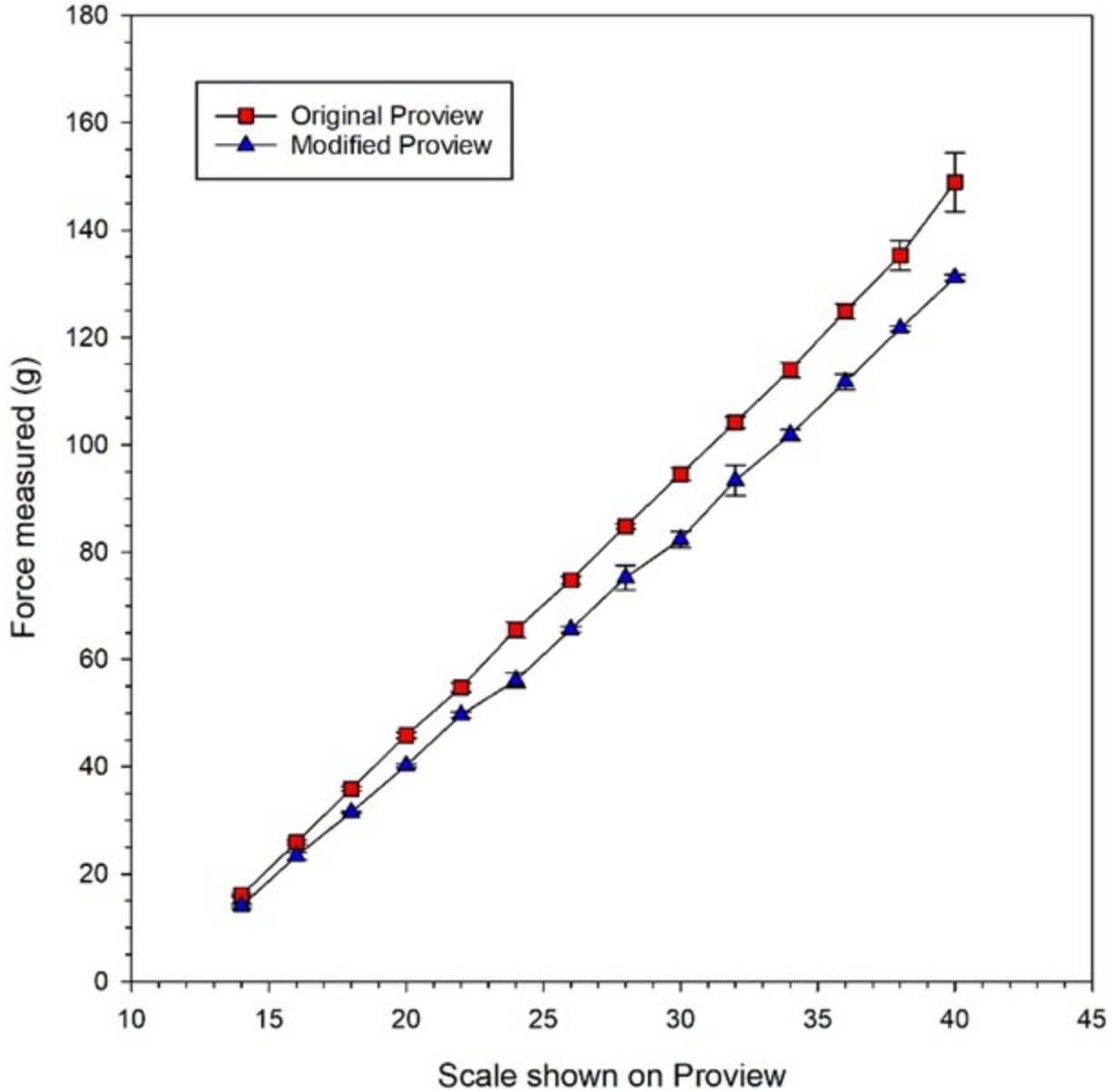
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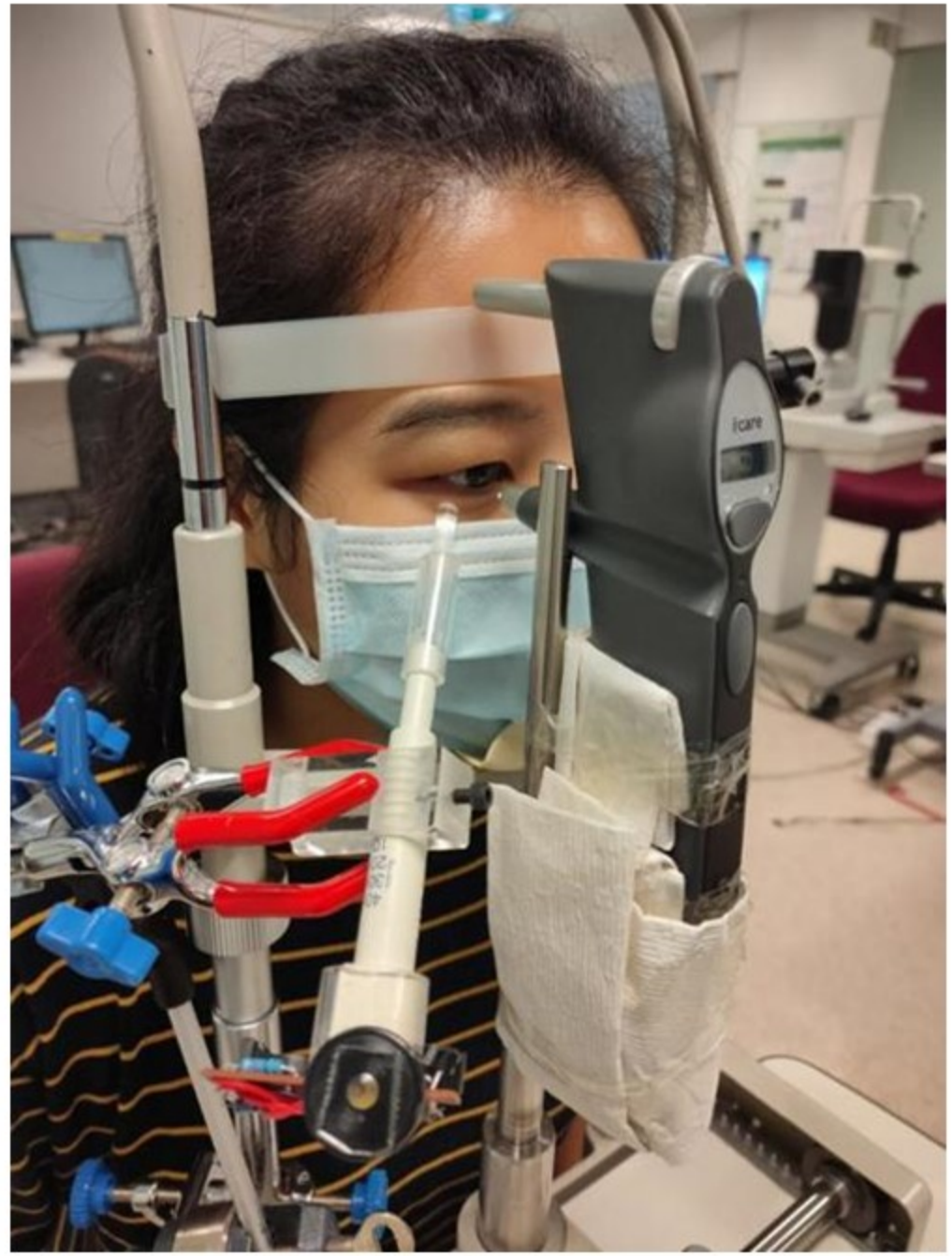
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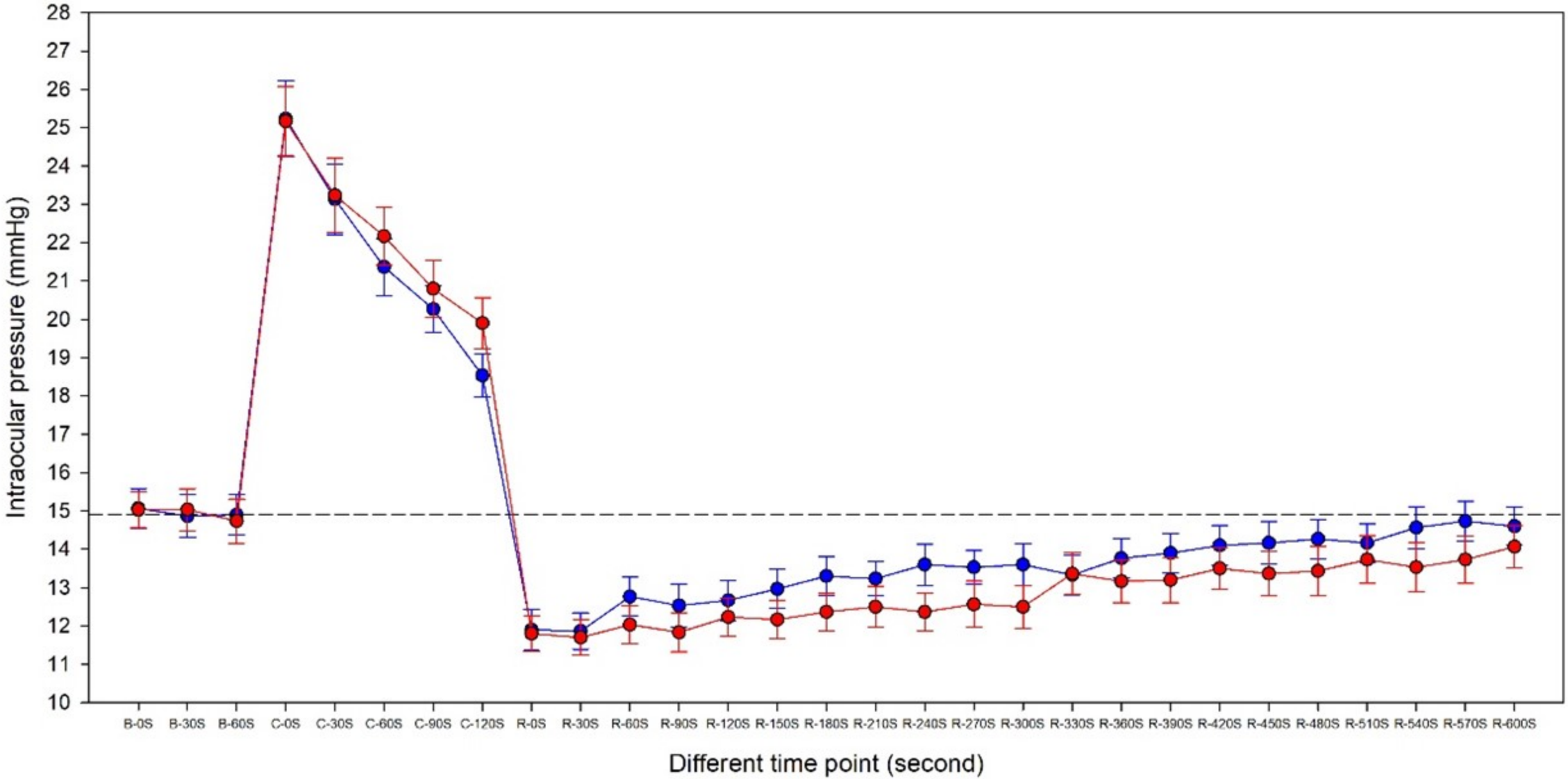
Intraocular pressure (mmHg)
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