Original Article

The evaluation of intraocular pressure fluctuation in glaucoma subjects during submaximal exercise using an ocular telemetry sensor

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Purpose: To evaluate the effect of acute submaximal exercise on intraocular pressure (IOP) fluctuations in open-angle glaucoma (OAG) subjects using an ocular telemetry sensor (OTS, Sensimed TriggerFish®). **Methods:** Twelve OAG subjects aged 45–65 years with no medical limitation for exercise were included in this prospective study. A submaximal exercise test was performed using a cycle ergometer for 20 min during which OTS voltages and metabolic parameters were recorded continuously. IOP voltages taken before, during, and after exercise were compared using the Friedman test and correlations with the metabolic parameters were evaluated using the Spearman analysis. **Results:** In two subjects, the OTS stopped functioning after a few hours. Median OTS measurements were 37.60 mVeq 10 min before exercise [interquartile range (IQR) 137.27], 51.75 (IQR 121.2), 62.35 (IQR 123.72), 54.6 (IQR 141.3), and 59.7 mVeq (IQR 196.7) during exercise (4 time points, 5 min apart), and 50.7 (IQR 147.35) and 64.2 mVeq (IQR 103.25) 10 and 30 min after exercise and the change was statistically non-significant ($P = 0.66$). No correlations were found between OTS and metabolic parameters measured at the same time points ($P > 0.05$). Nocturnal acrophase pattern was detected in five subjects (50%), diurnal acrophase in two patients, and double-hump in two patients. Median IOP voltages in the morning, afternoon/evening, and night were 335.84, 149.15, and 341.38 mVeq, respectively ($P < 0.001$). **Conclusion:** Continuous IOP monitoring did not reveal a remarkable voltage change in OAG patients during or immediately after exercise, but nocturnal IOP peaks in half of the patients.

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The clinical management of glaucoma still relies on one intraocular pressure (IOP) measurement usually taken during office hours. However, IOP has a dynamic nature with physiologic variations, and even multiple IOP measurements within office hours in sitting position cannot reveal nocturnal IOP peaks and fluctuations in supine position.[1–5] Sporadic and routine daily activities, such as increased amount of water intake, deep respirations, changing body position from sitting to supine, weight lifting, Valsalva maneuver, and exercise may result in small and large IOP fluctuations, which most of the time remain undetected due to difficulties in monitoring IOP during many activities.[6–12]

Continuous 24-h IOP monitoring with current tonometry devices requires hospitalization, limitation of daily activities, disruption of sleep cycles, positioning of the patients (sitting/supine), and topical anesthetic use in case of contact use.[13,14] The Sensimed TriggerFish® (Sensimed AG, Lausanne, Switzerland) is a minimally invasive ocular telemetry sensor (OTS) that provides continuous 24-h monitoring of ocular dimensional changes at the cornea-scleral junction related to IOP fluctuations without disturbing the patient's daily routines or sleep cycle.[15–18] The system comprises a disposable contact lens (CL) with an embedded microelectromechanical systems sensor, an antenna, and a telemetry application-specific integrated circuit. Recent studies showed that OTS was reproducible, safe, and well tolerated in glaucoma subjects.[15–18] The Sensimed TriggerFish® can detect IOP spikes missed by single measurements taken during office hours, allowing the treatment schedule to be tailored according to an individual's daily IOP peaks and troughs.

There are many studies in the literature evaluating the effect of aerobic exercise (walking, running/jogging, and bicycling) on IOP, most of which showed an IOP lowering effect in short term.[12,19–31] In these studies, IOP was measured with an insufficient sampling rate either at specific time points before, during, and after exercise, which necessitated interruption of the exercise or at time points before and after exercise, giving no information about IOP change during exercise.

Maximal exercise testing is considered as the gold standard to determine the subjects' maximal aerobic exercise capacity.[32] In these exercises, maximal oxygen consumption (VO_2 max), the largest amount of oxygen that an individual can utilize during strenuous exercise to complete exhaustion, is measured.

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VO_2 max is commonly used to assess cardio-respiratory endurance, physical exercise, health, and longevity. However, the effectiveness of such testing is often reduced due to musculoskeletal impairments or pain/fatigue rather than cardiac exertion. Because of higher risk for cardiovascular complications, they require advanced expertise, equipment, and medical supervision. In the last decade, many submaximal tests have been developed to overcome the limitations of maximal exercise testing and they became the method of choice for the majority of individuals who are frequently limited by cardiopulmonary, musculoskeletal, and neuromuscular impairments.

In this study, we used continuous IOP monitoring for the first time during exercise to assess IOP fluctuations in open-angle glaucoma (OAG) subjects. We also evaluated the correlations between IOP voltages and heart rate (HR), systolic/diastolic blood pressure, oxygen consumption (VO_2), and carbon dioxide production (VCO_2) taken at the same time points during exercise.

Methods

This prospective, non-randomized, single-center pilot study was conducted in accordance with the tenets of the Declaration of Helsinki. The Ethics Committee of Selcuk University approved the study protocol and written informed consent was obtained from the participants.

Twenty OAG patients between 45- and 65-year-old who had no medical limitation for exercise such as heart disease, uncontrolled systemic hypertension, rheumatologic disorder, or physical limitation for cycling were included in the study.

All subjects underwent complete ophthalmic examination including visual acuity, the Goldmann Applanation tonometry, gonioscopy, pachymetry, corneal topography (Sirius, CSO, Italy), anterior segment optical coherence tomography (AS-OCT, Spectralis, Heidelberg Engineering), and visual field testing with the Humphrey perimetry (Carl Zeiss Meditec). Although, we tried to include women in the study, most of them did not accept to participate after explaining the exercise program or because of systemic diseases such as heart disease and physical limitations. OTS was applied only to one eye of the patient. Eyes with previous ocular surgery, laser trabeculoplasty or iridotomy, uveitic glaucoma, ocular trauma, or severe dry eye were excluded. If both the eyes were eligible for the study, the eye with more severe glaucoma was fitted with the OTS. If the perimetric stages of the eyes were similar, the right eye was selected.

Metabolic measurements and calculation of maximal oxygen consumption

The Astrand–Ryming protocol was used to determine VO_2 max of the participants.[33] Measurements in testing protocol were explained in detail to the subjects before the application of the test. Polar 810i (Polar Electro Oy, Finland) monitors were attached to measure resting and exercise HR. The patients were connected to a metabolic analyzer (Quark b2, Cosmed, Italy) by means of a mask, and VO_2 and VCO_2 during exercise at each inspiration and expiration (breath by breath) were recorded using a computer software. Participants started cycling (Ergoline 900; SensorMedics Corp, USA) with a predefined resistance level that was specifically determined for each patient and was increased until the HR of the participant reached 130–170/min. Estimated VO_2 max was calculated through combining average HR and VO_2 values attained between 5–6 min in the Astrand–Ryhming nomogram which is corrected for age.[34] Out of 20 subjects, 12 men who completed the exercise program successfully were found to be eligible for the study. Two women could not complete the initial submaximal exercise test to determine their estimated VO_2 max value.

Application of the ocular telemetry sensor

One week after the submaximal exercise test, an OTS with a base curve of 8.7 mm and a diameter of 14.1 mm was placed to the selected eye of the subject. Then a periorbital self-adhesive patch, which contains an antenna and transmits data from the CL to the recorder via data cable, was applied. The OTS records dimensional changes in the corneoscleral area for 30 s of every 5 min over a 24-h period; each recording "burst" represents 300 measurements, and approximately 80,000 data points are recorded in 24 h. The median value from these reading intervals is recorded and transmitted from the sensor to the antenna and then to the recorder. Computer-generated graphs show the IOP fluctuations in milliVolt equivalents (mVeq) on the vertical axis. The fitting was then evaluated at the slit-lamp for lens centration, movement, and air bubbles.

Exercise program

About 1 h after placement of the OTS, the subjects were connected to a metabolic analyzer with a mask [Fig. 1]. Based on their predetermined 55–60% VO_2 max values, subjects were exercised on the cycle ergometer for 20 min during which metabolic parameters (HR, systolic/diastolic blood pressure, VO_2, and VCO_2) were recorded continuously. The water intake before and immediately after exercise was limited to two glasses, which was similar among the subjects. Subjects were instructed to instill IOP lowering drugs according to their usual treatment schedule and register their daily activities and sleeping time in an activity logbook.

After 24 h, the recorder shut off automatically. The patients were examined for the position of the lens, conjunctival hyperemia, and corneal edema. The OTS was removed and the data were transferred to computer to visualize the 24-h IOP pattern. Central corneal thickness (CCT) measurements were taken with AS-OCT.

Analysis of charts

The initial reading taken when the CL is inserted is recorded as 0 mVeq. If the distension of corneoscleral region is less than the initial fitting, it is recorded as negative reading, while if the distension is more, the reading is positive, which means a high mVeq corresponds to a high IOP. Sleep periods were determined based on blink cessation (identified as short- and high-amplitude spikes that are displayed by the software) and were confirmed using sleep times reported by the subjects. Patients were then classified into pattern groups as follows: diurnal acrophase (peak during the diurnal/wakefulness period), nocturnal acrophase (peak during the nocturnal/sleep period), double-hump acrophase (peaks during both periods), and no significant acrophase.

Data points within the exercise period (four measurements, 5 min apart), 1 data point 10 min before the exercise (baseline) and 2 data points after the exercise (after 10 and 30 min) were analyzed to evaluate the effect of exercise on IOP fluctuation.

For 24-h IOP fluctuation, smoothed 1-h data points extrapolated from the chart using the mean of three measurements were used for analysis. The charts were also analyzed according to the morning (6:00–11:00 am), afternoon/evening (12:00–11:00 pm), and night (12:00–5:00 am) subperiods.

Statistical analysis

Repeated measurements were compared using non-parametric Friedman test, whereas the correlations between OTS and metabolic parameters taken at the same time points during exercise were evaluated using the Spearman correlation analysis and linear regression analysis.

Results

In two subjects, OTS stopped recording after a few hours. Mean age of the remaining 10 men (8 POAG and 2 exfoliation glaucoma) was 55.2 ± 5.05 years (45–65 years). Mean cup-to-disc (C/D) ratio was 0.76 ± 0.08. Mean peripapillary retinal nerve fiber layer thickness was 73 ± 20.56 µm. Visual field indices mean deviation (MD) and pattern standard deviation (PSD) were 8.87 and 5.09, respectively. Mean antiglaucoma medications were 2.4 ± 0.8 and six patients were using prostaglandin analogues. Mean IOP before and after OTS application was 14.90 ± 2.8 mmHg and 15.6 ± 2.6 mmHg. Mean height and weight of the subjects were 171.6 ± 8.3 cm and 89.3 ± 17.8 kg. Mean body fat ratio and HR were 30.8% ±6.4 and 78.7 ± 8.9, respectively. VO_2 max and 60% load were calculated as 26.4 ± 3.2 mL/kg/min and 71.4% ±18.9 W, respectively.

Five subjects reported blurred vision during lens wear. Conjunctival hyperemia was mild-moderate in seven patients and severe in three patients. None of the subjects had clinically significant cornea edema. Six subjects had superficial punctuate keratitis and two eyes showed a CL edge pressure mark. CCT values after OTS removal were statistically higher compared to baseline measurements (510 ± 27.7 µm and 494.2 ± 23.8 µm, respectively) (Wilcoxon signed-ranks test, $P = 0.007$).

Ocular telemetry sensor analysis

Median OTS measurements were 37.6 mVeq 10 min before exercise (IQR 137.27), 51.7 (IQR 121.2), 62.3 (IQR 123.72), 54.6 (IQR 141.3), and 59.7 mVeq (IQR 196.7) during exercise (4 time points, 5 min apart), and 50.7 (IQR 147.35) and 64.2 mVeq (IQR 103.25) 10 and 30 min after exercise. The change in OTS measurements during exercise was non-significant (Friedman test, $P = 0.66$) [Table 1]. During exercise, voltage values decreased in four patients, increased in three patients, and no remarkable change was detected in three patients [Fig. 2].

The physiological parameters measured during exercise were given in Table 1. Using non-parametric Spearman correlation analysis, no correlations were found between OTS voltages and HR, systolic BP, diastolic BP, VO_2, and VCO_2 taken at the same time points ($P > 0.05$) [Table 2]. Linear regression analysis showed no relationship between OTS and five physiological parameters at baseline ($P = 0.79$), 1st time point ($P = 0.53$), 2nd time point ($P = 0.62$), 3rd time point ($P = 0.95$), and 4th time point ($P = 0.42$).

The mean amplitude of the 24-h curves was 251.5 ± 128.3 mVeq (median 215.4 mVeq). The change in 24-h measurements was statistically significant (Friedman test, $P < 0.005$). Nocturnal acrophase pattern was detected in five subjects (50%); diurnal acrophase in two patients, and double-hump in two patients, while no acrophase was detected in one patient.

Median IOP voltages in the morning, afternoon/evening, and night were 335.84, 149.15, and 341.38 mVeq, respectively ($P < 0.001$) [Fig. 3]. Significant differences were observed for

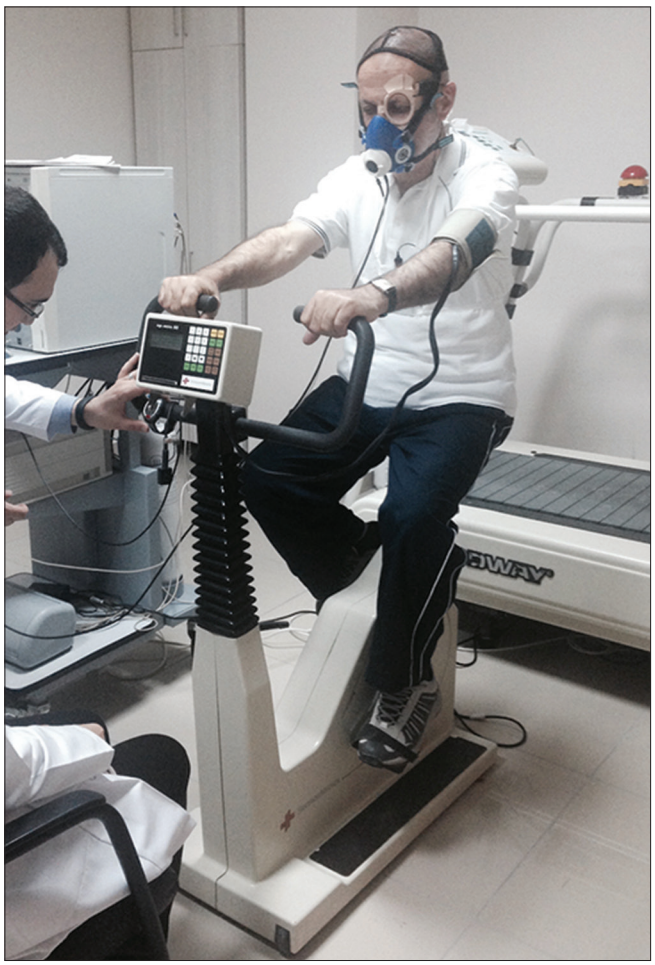

Figure 1: After placement of the Sensimed Triggerfish®, the participant got on a cycle ergometer and was connected to a metabolic analyzer with a mask

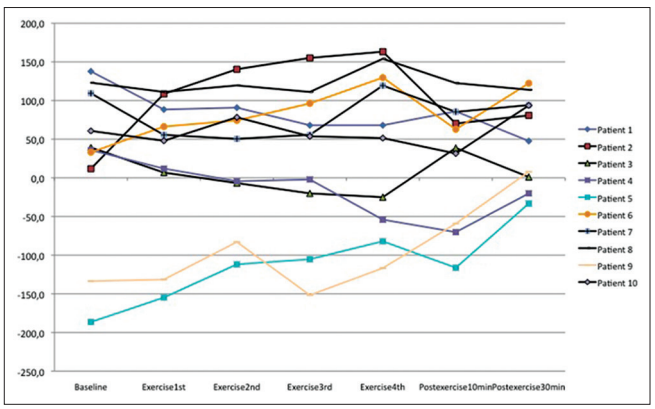

Figure 2: Ocular telemetry sensor measurements of the subjects during exercise

Table 1: Ocular telemetry sensor values (median and interquartile range) and physiological parameters (mean±standard deviation) before, during, and after exercise

	Baseline	Exercise 1st	Exercise 2nd	Exercise 3rd	Exercise 4th	Post-exercise 10 min	Post-exercise 30 min
OTS values mVeq	37.60-137.27	51.75-121.2	62.35-123.72	54.6-141.3	59.7-196.7	50.7-147.35	64.2-103.25
HR (beats/min)	89.2±12.4	117.6±13.6	123±15.6	125±16.8	126.7±17.6		
SBP (mmHg)	130.0±20.5	167.6±29.3	167.8±25.8	161.9±26.6	165.0±28.0		
DBP (mmHg)	86.7±11.1	85.5±8	84.7±9.9	85.6±9.2	83.5±8.5		
VO_2 (mL/min)	348.3±75.8	536.6±107.2	548.5±97.8	554.4±93.2	560.4±92.4		
VCO_2 (mL/min)	734.5±125.9	1387.7±204.3	1403.5±214.4	1386.6±216.7	1385±204.7		

Data points 10 min before (baseline), within the exercise period (4 measurements, 5 min apart), 10 and 30 min after the exercise. BP: Blood pressure, VO_2: Oxygen consumption, VCO_2: Carbon dioxide production, OTS: Ocular telemetry sensor, SBP: Systolic BP, DBP: Diastolic BP, HR: Heart rate

Table 2: Correlation analysis between ocular telemetry sensor values and corresponding physiological parameters before and during submaximal exercise

	HR	SBP	DBP	VO_2	VCO_2
Baseline OTS	−0.42; *P* 0.23	0.11; *P* 0.75	0.27; *P* 0.44	0.31; *P* 0.38	0.21; *P* 0.56
Exercise 1st	−0.53; *P* 0.12	0.036; *P* 0.92	−0.48; *P* 0.16	0.2; *P* 0.58	0.38; *P* 0.28
Exercise 2nd	−0.02; *P* 0.96	0.19; *P* 0.6	0.11; *P* 0.76	0.18; *P* 0.63	0.41; *P* 0.24
Exercise 3rd	−0.18; *P* 0.63	0.18; *P* 0.63	0.05; *P* 0.88	0.28; *P* 0.42	0.26; *P* 0.47
Exercise 4th	−0.25; *P* 0.49	0.07; *P* 0.85	−0.16; *P* 0.65	0.35; *P* 0.33	0.38; *P* 0.28

Spearman rho correlation coefficient and *P* values. BP: Blood pressure, SBP: Systolic BP, DBP: Diastolic BP, HR: Heart rate, VO_2: Oxygen consumption, VCO_2: Carbon dioxide production, OTS: Ocular telemetry sensor

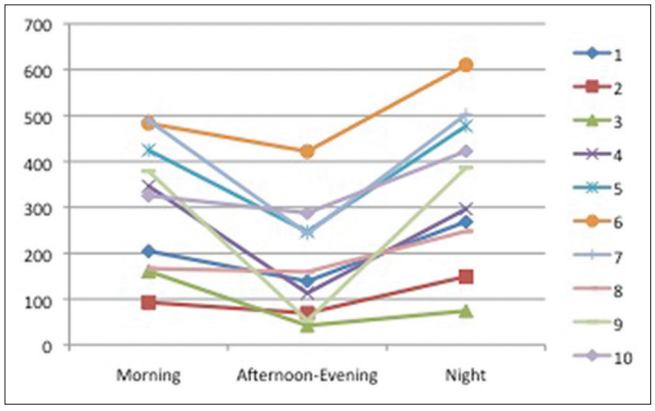

Figure 3: Ocular telemetry sensor measurements of the subjects according to the morning, afternoon/evening, and night subperiods

morning versus afternoon/evening ($P < 0.001$) and night versus afternoon/evening ($P < 0.001$); there was no difference between morning and night subperiods (Wilcoxon signed-rank test, $P = 0.093$).

Discussion

Aerobic exercise involves repeated rhythmic movements of the large muscles and includes any type of exercise, typically those performed at moderate levels of intensity for extended periods of time, which maintains an increased HR, such as walking, running/jogging, swimming, cycling, and dancing. Except for an immediate rise in IOP due to the contraction of abdominal and thoracic muscles and Valsalva maneuver, aerobic exercise has consistently been shown to lower IOP in the acute post-exercise period.[12,19–31] The degree of IOP reduction was shown to be directly proportional to the intensity of the exercise and recovery of IOP to baseline levels after exercise has been reported to take up to an hour.[21,23,27]

Qureshi[19] evaluated the effects of sitting, walking, jogging, and running fast until exhaustion on IOP in 15 healthy sedentary men at an interval of 4 days and found that during sitting, walking, and jogging IOP decreased by 1.20 ± 0.66, 3.20 ± 1.19, and 5.07 ± 1.76 mmHg, respectively, whereas the mean decrease after running was 5.7 ± 1.09 mmHg. Qureshi[20] further compared the effects of walking, jogging, and running fast until exhaustion on IOP in seven normal and seven OAG subjects and showed that glaucoma patients had a greater drop in IOP measurements (7.72, 10.86, and 12.86 mmHg, respectively) as compared to normal subjects (2.43, 3.85, and 4 mmHg, respectively). Avunduk *et al*.[21] measured IOP before and 10 min after exercise and showed that both isometric and isokinetic exercises lowered IOP with direct relationship to exercise intensity and total energy consumption. In a study by Price *et al*.,[22] 18 subjects cycled for 4 min at a constant rate of 80 rpm on a bicycle ergometer. Pneumotonometry measurements taken before, immediately after cessation of exercise, and at 5, 10, 20, and 30 min thereafter revealed that mean IOP fell by 5.5 mmHg after exercise and recovered gradually toward baseline over a period of 30 min. In a study on 21 healthy subjects, Rüfer *et al*.[23] reported that IOP decreased about 2 mmHg after cycling with an HR of 170 beats/min and returned to baseline level after a rest period of 10 min. Read and Collins[24] reported that a 10-min period of low-impact, moderate-intensity exercise with a bicycle ergometer reduced IOP by 1.71 ± 1.24 mmHg immediately after exercise, 1.26 ± 1.19 mmHg at 5 min, and 0.56 ± 1.03 mmHg at 10 min after exercise in young adult subjects. In a study by Karabatakis *et al*.[25] on 29 healthy subjects, IOP decreased significantly after 20 min of jogging. Krejci *et al*.[26] designed an

individualized four-stage workload with a bicycle ergometer using the Astrand–Ryhming protocol to bring each subject to 85% of a predicted maximum HR and found a substantial decrease in IOP (using a non-contact tonometer) at the end of the fourth stage and after 1, 5, and 10 min of recovery. In the study of Saarela *et al*.[27] the biomicroscope and Goldmann Tonometer were set up in front of the exercise bike and the measurements were recorded with the subject sitting on the bike, but not pedaling, with their head position stabilized by the instrument headrest. The authors found that exercise bike for 9 min lowered IOP, statistically. Natsis *et al*.[30] measured the IOPs of 145 healthy and glaucoma individuals with GAT 3 min before and 5 min after exercise on a bicycle ergometer (about 10 min of duration at 60–80 W, based on individual preference). Aerobic exercise reduced IOP both in healthy eyes where a β-blocker, prostaglandin analogue, or α-agonist was previously instilled and in glaucomatous eyes under treatment. Instillation of the β-blocker did not counterbalance the effect of exercise on IOP. A meta-analysis of 10 studies evaluating the effect of acute aerobic exercise on IOP has shown that aerobic exercise clearly reduces IOP, more markedly in sedentary subjects (4.2 mmHg) compared to physically active subjects (2.3 mmHg).[31]

In his review, McMonnies suggested that dynamic exercise and cyclic nature of increased respiration volumes may lead to IOP elevations during exercise, which increase aqueous outflow by a pumping effect and result in lower IOP levels after exercise compared to pre-exercise levels.[12] He questioned whether fluctuations in IOP is related to muscle exertion; changes in body position; and increased respiratory volumes during physical exercise may contribute to progression of glaucoma in susceptible individuals.

Although most of the papers in the literature reported a decrease in IOP after exercise, some papers found no effect on IOP.[35–36] Era *et al*.[35] evaluated the effect of maximal bicycle ergometer test on IOP in elderly athletes and controls and found that IOP decreased > 2 mmHg in 34% of the subjects, did not changed in 57% and increased in 9%. Moura *et al*.[36] evaluated the effect of water intake on IOP of six healthy men with a mean age of 24.0 ± 3.5 years. The subjects exercised until exhaustion on a cycle ergometer at a 60% VO_2 peak and IOP was measured before and after exercise (0, 15, 30, and 45 min) using the Applanation tonometry. IOP increased after water ingestion both under exercising and resting conditions ($P < 0.05$), but did not differ between resting and exercising situations.

The main problem in most of the studies in the literature is the lack of standardization and continuous tonometry during dynamic exercise. The Sensimed Triggerfish® OTS is a wireless silicon CL sensor, which can be used for recording an IOP-related pattern over a 24-h period. This is the first study in which continuous IOP monitoring was used to detect IOP fluctuations during cycling in OAG subjects, which is impossible with the current tonometry systems. In our study, voltage values decreased in four patients, increased in three patients, and did not change in three patients during exercise ($P = 0.66$).

The major limitations of this study were the small sample size and the lack of control group, because of limited grant and proper patient selection for such an exercise. However, it is a pilot study that evaluates the uninterrupted, continuous behavior of IOP during exercise in glaucoma subjects, which might clarify the relation between dynamic exercises, increase in respiration volumes, and increase in IOP. The OTS actually records IOP-related cornea-scleral curvature changes plotted against time, and theoretically, a change of 1 mmHg in IOP is assumed to cause a 3 μm difference in corneal radius.[37] However, it should be kept in mind that the relationship between IOP and corneal curvature is a complicated function that depends on the thickness and elasticity of cornea and sclera, and therefore deviations from standard ocular biomechanical parameters might result in false measurements with the OTS. Another problem is that the OTS data are in electrical units, referenced against a starting value of zero at each recording session, not in millimeters of mercury. Faschinger and Mossböck,[38] Sunaric-Megevand *et al*.,[39] and Holló *et al*.[40] reported that OTS values did not correlate with tonometric measurements. In the study of Holló *et al*.[40], although the 24-h GAT IOP decreased from 22.91 ± 5.11 to 18.24 ± 2.49 mmHg after treatment with travoprost ($P < 0.001$), two OTS curves before the treatment and one OTS curve after the treatment were similar (152.94, 142.35, and 132.98 au, respectively, $P = 0.27$) and temporary changes in IOP were not always picked up on the OTS. In a recent study by Vitish-Sharma *et al*.,[41] Sensimed Triggerfish® CLS data output showed only a weak correlation ($r = 0.291$) (95%) with IOP measurements taken by Tono-pen XL® Applanation tonometer during laparoscopic colorectal surgery. Therefore, based on the results of these studies, it seems that Sensimed Triggerfish® cannot be relied upon to reflect the changes in IOP during exercise.

In this study, we found no correlations between OTS voltages, HR, systolic/diastolic blood pressure, oxygen consumption, and CO_2 production measured at the same time points during exercise, similar with previous studies.[25,26,42]

Conclusion

Although aerobic exercise has been known to have an IOP lowering effect in the acute post-exercise period, we could not find a remarkable change in OTS measurements among glaucoma patients during or after submaximal exercise, but nocturnal IOP peaks in half of the patients. Further studies with a larger group of glaucoma and healthy subjects in comparison with IOP measurements using other tonometries before and after exercise are needed to demonstrate the reliability and the reproducibility of OTS technology.

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Conflicts of interest

There are no conflicts of interest.

References

1. David R, Zangwill L, Briscoe D, Dagan M, Yagev R, Yassur Y, *et al*. Diurnal intraocular pressure variations: An analysis of 690 diurnal curves. Br J Ophthalmol 1992;76:280-3.
2. Saccà SC, Rolando M, Marletta A, Macrí A, Cerqueti P, Ciurlo G. Fluctuations of intraocular pressure during the day in open-angle glaucoma, normal-tension glaucoma and normal subjects. Ophthalmologica 1998;212:115-9.
3. Hughes E, Spry P, Diamond J. 24-hour monitoring of intraocular pressure in glaucoma management: A retrospective review. J Glaucoma 2003;12:232-6.

4. Barkana Y, Anis S, Liebmann J, Tello C, Ritch R. Clinical utility of intraocular pressure monitoring outside of normal office hours in patients with glaucoma. Arch Ophthalmol 2006;124:793-7.
5. Liu JH, Zhang X, Kripke DF, Weinreb RN. Twenty-four-hour intraocular pressure pattern associated with early glaucomatous changes. Invest Ophthalmol Vis Sci 2003;44:1586-90.
6. Vieira GM, Oliveira HB, de Andrade DT, Bottaro M, Ritch R. Intraocular pressure variation during weight lifting. Arch Ophthalmol 2006;124:1251-4.
7. Kerr NM, Danesh-Meyer HV. Understanding the mechanism of the water drinking test: The role of fluid challenge volume in patients with medically controlled primary open angle glaucoma. Clin Exp Ophthalmol 2010;38:4-9.
8. Yamabayashi S, Aguilar RN, Hosoda M, Tsukahara S. Postural change of intraocular and blood pressures in ocular hypertension and low tension glaucoma. Br J Ophthalmol 1991;75:652-5.
9. Krist D, Cursiefen C, Jünemann A. Transitory intrathoracic and -abdominal pressure elevation in the history of 64 patients with normal pressure glaucoma. Klin Monbl Augenheilkd 2001;218:209-13.
10. Porth CJ, Bamrah VS, Tristani FE, Smith JJ. The valsalva maneuver: Mechanisms and clinical implications. Heart Lung 1984;13:507-18.
11. Rosen DA, Johnston VC. Ocular pressure patterns in the valsalva maneuver. Arch Ophthalmol 1959;62:810-6.
12. McMonnies CW. Intraocular pressure and glaucoma: Is physical exercise beneficial or a risk? J Optom 2016;9:139-47.
13. Liu JH, Weinreb RN. Monitoring intraocular pressure for 24 h. Br J Ophthalmol 2011;95:599-600.
14. Sit AJ. Continuous monitoring of intraocular pressure: Rationale and progress toward a clinical device. J Glaucoma 2009;18:272-9.
15. Mansouri K, Shaarawy T. Continuous intraocular pressure monitoring with a wireless ocular telemetry sensor: Initial clinical experience in patients with open angle glaucoma. Br J Ophthalmol 2011;95:627-9.
16. Mansouri K, Liu JH, Weinreb RN, Tafreshi A, Medeiros FA. Analysis of continuous 24-hour intraocular pressure patterns in glaucoma. Invest Ophthalmol Vis Sci 2012;53:8050-6.
17. Lorenz K, Korb C, Herzog N, Vetter JM, Elflein H, Keilani MM, *et al.* Tolerability of 24-hour intraocular pressure monitoring of a pressure-sensitive contact lens. J Glaucoma 2013;22:311-6.
18. Agnifili L, Mastropasqua R, Frezzotti P, Fasanella V, Motolese I, Pedrotti E, *et al.* Circadian intraocular pressure patterns in healthy subjects, primary open angle and normal tension glaucoma patients with a contact lens sensor. Acta Ophthalmol 2015;93:e14-21.
19. Qureshi IA. Effects of mild, moderate and severe exercise on intraocular pressure of sedentary subjects. Ann Hum Biol 1995;22:545-53.
20. Qureshi IA. The effects of mild, moderate, and severe exercise on intraocular pressure in glaucoma patients. Jpn J Physiol 1995;45:561-9.
21. Avunduk AM, Yilmaz B, Sahin N, Kapicioglu Z, Dayanir V. The comparison of intraocular pressure reductions after isometric and isokinetic exercises in normal individuals. Ophthalmologica 1999;213:290-4.
22. Price EL, Gray LS, Humphries L, Zweig C, Button NF. Effect of exercise on intraocular pressure and pulsatile ocular blood flow in a young normal population. Optom Vis Sci 2003;80:460-6.
23. Rüfer F, Schiller J, Klettner A, Lanzl I, Roider J, Weisser B. Comparison of the influence of aerobic and resistance exercise of the upper and lower limb on intraocular pressure. Acta Ophthalmol 2014;92:249-52.
24. Read SA, Collins MJ. The short-term influence of exercise on axial length and intraocular pressure. Eye (Lond) 2011;25:767-74.
25. Karabatakis VE, Natsis KI, Chatzibalis TE, Lake SL, Bisbas IT, Kallinderis KA, *et al.* Correlating intraocular pressure, blood pressure, and heart rate changes after jogging. Eur J Ophthalmol 2004;14:117-22.
26. Krejci RC, Gordon RB, Moran CT, Sargent RG, Magun JC. Changes in intraocular pressure during acute exercise. Am J Optom Physiol Opt 1981;58:144-8.
27. Saarela V, Ahvenvaara E, Tuulonen A. Variability of heidelberg retina tomograph parameters during exercise. Acta Ophthalmol 2013;91:32-6.
28. Leighton DA, Phillips CI. Effect of moderate exercise on the ocular tension. Br J Ophthalmol 1970;54:599-605.
29. Myers KJ. The effect of aerobic exercise on intraocular pressure. Invest Ophthalmol 1974;13:74-6.
30. Natsis K, Asouhidou I, Nousios G, Chatzibalis T, Vlasis K, Karabatakis V, *et al.* Aerobic exercise and intraocular pressure in normotensive and glaucoma patients. BMC Ophthalmol 2009;9:6.
31. Roddy G, Curnier D, Ellemberg D. Reductions in intraocular pressure after acute aerobic exercise: A meta-analysis. Clin J Sport Med 2014;24:364-72.
32. Noonan V, Dean E. Submaximal exercise testing: Clinical application and interpretation. Phys Ther 2000;80:782-807.
33. Astrand P, Rodahl K, Dahl HA, Stromme SB. Textbook of Work Physiology. Physiological Bases of Exercise. Champaign, IL: Human Kinetics Publishers; 2003.
34. Cooper CB, Storer TW. Exercise Testing and Interpretation: A Practical Approach. New York, NY: Cambridge University Press; 2001. p. 51-92.
35. Era P, Pärssinen O, Kallinen M, Suominen H. Effect of bicycle ergometer test on intraocular pressure in elderly athletes and controls. Acta Ophthalmol (Copenh) 1993;71:301-7.
36. Moura MA, Rodrigues LO, Waisberg Y, De Almeida HG, Silami-Garcia E. Effects of submaximal exercise with water ingestion on intraocular pressure in healthy human males. Braz J Med Biol Res 2002;35:121-5.
37. Leonardi M, Pitchon EM, Bertsch A, Renaud P, Mermoud A. Wireless contact lens sensor for intraocular pressure monitoring: Assessment on enucleated pig eyes. Acta Ophthalmol 2009;87:433-7.
38. Faschinger C, Mossböck G. Validity of the results of a contact lens sensor? JAMA Ophthalmol 2013;131:696-7.
39. Sunaric-Megevand G, Leuenberger P, Preußner PR. Assessment of the triggerfish contact lens sensor for measurement of intraocular pressure variations. Acta Ophthalmol 2014;92:e414-5.
40. Holló G, Kóthy P, Vargha P. Evaluation of continuous 24-hour intraocular pressure monitoring for assessment of prostaglandin-induced pressure reduction in glaucoma. J Glaucoma 2014;23:e6-12.
41. Vitish-Sharma P, Acheson AG, Stead R, Sharp J, Abbas A, Hovan M, *et al.* Can the SENSIMED triggerfish® lens data be used as an accurate measure of intraocular pressure? Acta Ophthalmol 2018;96:e242-6.
42. Risner D, Ehrlich R, Kheradiya NS, Siesky B, McCranor L, Harris A. Effects of exercise on intraocular pressure and ocular blood flow: A review. J Glaucoma 2009;18:429-36.