International Journal of Medical and Allied Health Sciences

ISSN: 2583-1879

Review Article

The Impacts of Physical Exercises, Dynamic Activities and Isometric Exercises on Intraocular Pressure of Eye: A Review

Sayma Ilyas[1], Areeb Rehmani[2], Tara Rani[3], Labishetty Sai Charan[4], Dr. Kamal Pant[5] Salal Khan[6]*

[1] Optometry Scholar, Department of Optometry, Galgotias University, Greater Noida, U.P.
[2,6] M. Optometry Scholar, Department of Optometry, Galgotias University, Greater Noida, U.P.
[3]Assistant Professor, Department of Optometry, Galgotias University, Greater Noida, U.P.
[4]Assistant Professor, Department of Optometry, Chandigarh University, Punjab.
[5]Associate Professor & Head, Department of Optometry, Uttar Pradesh University of Medical Sciences, Saifai, (U.P)

Article Information: Received on 25-03-2022 Accepted 16-5 -2022 Available online 30 -05-2022

Abstract

To review the effects of exercise and other physical activities on intraocular pressure, which can lead to glaucomatous damage to the optic nerve. This can help in the prognosis in the early stages of glaucoma. Intraocular pressure reduction is currently the only treatment available for decreasing the risk of glaucoma progression so it is important to find additional ways to control it. This was the Perspective study after a literature review and Analysis of pertinent publications in the peer-reviewed literature. In this study, various findings have shown that intraocular pressure can be decreased or raised depending on the workouts and activities performed. Although we have not compared the amount of IOP drop or rise in different workouts, a general concept of which exercise/activity is beneficial or risky for glaucoma patients may be derived. When compared to isometric workouts, dynamic exercise has a greater effect on lowering IOP. The relative intensity of the exercise is the metric that most closely corresponds to the IOP-lowering impact. Activities including bungee jumping, yoga in a head-down posture, swimming goggles, and coffee ingestion soon before exercise, on the other hand, have been demonstrated to elevate IOP readings. Valsalva Maneuver performed soon after a hard workout can offset the activity's IOP-reducing effect. To avoid additional glaucomatous damage, glaucoma patients should avoid these activities that raise IOP.

Keywords: Intraocular pressure, Exercise, Activities, Eye Health, Circadian cycle, Optometry

1. Introduction

Physical work has been shown to improve health in a variety of systemic disorders, including progressive visual neuropathy marked by rapid death of retinal ganglion cells (RGCs). The two basic theories for glaucoma development and progression are raised intraocular pressure (IOP) and less ocular perfusion (OP). Numerous researches have proven the benefits of exercise in these two areas. According to a 2009 analysis of these two theories, exercise causes a transitory reduction in IOP but an inconsistent increase in ocular perfusion [1].

* **Salal Khan,** Department of Optometry, Galgotias University, Greater Noida, U.P (India).
E-mail: salalmohammad786@gmail.com
ORCID ID: https://orcid.org/0000-0002-1269-3428
doi: https://doi.org/10.54618/IJMAHS.2022212

IOP fluctuations are significantly linked to the development of glaucoma and Ocular hypertension, and IOP reduction and stabilization is a critical element in preventing ocular damage [2]. Circadian variations [3], physical exercise [4,5], cognitive processing [6], the Valsalva maneuver [7], and daily activities [8] have all been demonstrated to produce changes in IOP. To maintain ocular health, it is necessary to understand IOP behavior as a result of all of these factors. Risk factors of glaucoma are; Intraocular pressure (IOP), heredity, race, age, smoking and vascular variables [9]. Hypotensive treatments have been proven to decrease the progression of illness such as visual field defects and acuity changes [9-14].

A vascular factor has become increasingly implicated as contributing to the chronic illnesses [15-18] process in patients who continue to demonstrate glaucomatous development despite well-controlled intraocular pressure [19,15]. According to Siesky et al.

[18] evidence for vascular etiologies in the literature, which states that vascular risk factors for glaucoma are aging, systemic blood pressure, nocturnal hypotension, migraine, ocular perfusion pressure (OPP), disc hemorrhage, and loss of ocular blood flow (OBF).

In some studies, it was shown that following doing the squat and military press exercises, OPP showed a considerable reduction, indicating a trend toward a reduction after strength training [20]. These findings complement prior data that extremely challenging resistance training workouts affect IOP and OPP responses, but these changes appear to be influenced by the kind of exercise and participant characteristics. Furthermore, both isometric and dynamic activities have been demonstrated to increase OPP while overall ocular perfusion stays steady, demonstrating auto regulation [21-25]. Dynamic exercise causes a transient rise in OBF before returning to baseline [26], and pulsatile flow also increases slightly [27,28]. Auto regulation is successful up to 70% above baseline OPP levels, after which it begins to fail.

The link between exercise and mental status has been extensively researched, and increased physical activity has been linked to lower levels of anxiety and depression [29,30]. Depression was linked to inadequate physical activity in two recent population-based studies published in 2016 and 2017, encompassing 204186 and 178867 persons from more than 30 low-income countries, respectively. Depression was linked to inadequate physical activity in 30 low- and middle-income nations, according to research published in 2016 and 2017[31,32].

The average human life has increased dramatically in recent years as a result of tremendous advancements in modern medicine. However, aging is unavoidable, and it is marked by a variety of degenerative phenotypes in different organs, particularly the eye. Deterioration of the retina and loss of lens accommodation are two examples of age-related ocular alterations. Exercise can help elderly people with this process because it reduces the susceptibility of the old optic nerve to negative stimuli [33-35]. Several eye illnesses, such as glaucoma and AMD, are thought to be exacerbated by oxidative stress, which plays a role in the fragility of aging retinas. Treadmill training has been shown in mice to be advantageous for aging retinas by reducing oxidative injury [35,36].

In both humans and experimental animals, exercise has been demonstrated to boost neural flexibility and increase resistance to degeneration in the cardiovascular system and brain [37-43]. Up regulating neurotropic expression, improving mitochondrial function, and lowering inflammation are some of the ways exercise produces neuroprotection.

2. Physical exercise (dynamic and isometric) and IOP

Dynamic and isometric exercises are two different types of physical activity. Dynamic exercise, such as jogging and cycling, requires a change in muscle length, whereas isometric exercise, such as weightlifting and hand gripping, is done in a static position.

According to Risner D et al. [4] IOP decreased following dynamic exercise, while IOP after isometric exercise was more controversial. Although the underlying mechanism of IOP reduction remains unknown, intraocular pressure has regularly been found to decrease during vigorous exercise. Due to the varying intensity and duration of the different types of exercise, the level of IOP decrease has been observed to be diverse. Experiments on humans revealed that the rise in IOP during isometric exercise was related to changes in blood pressure and was regulated in part by carbon dioxide partial pressure (measured by the transcutaneous route) [44].

Table-1: Effect of different types of dynamic exercises and change in IOP

Type of dynamic exercise	Reported change in IOP	Effect on OPP and OBF	Study
Treadmill	Decrease in IOP	No significant change was observed in OPP and OBF	[48]
Bicycle ergometer	Decrease in IOP	Increase in OPP Increase in OBF in the ophthalmic artery No change in the central retinal artery	[49]
Stair climbing	No significant change in intraocular pressure	No significant change in ocular blood flow	[50]
Brisk Walk	Decrease in IOP	No significant change in OPP and OBF	[51]
Submaximal exercises	Decreased as well as increased	Decreased as well as increased	[52]

[this table shows various types of dynamic exercise and their effect on IOP, OPP, and OBF; IOP=Intraocular pressure, OPP =Ocular perfusion pressure, OBF= Ocular blood flow]

The Valsalva manoeuvre has been linked to the IOP increase caused by isometric exercise. When individuals were forced to hold their breath, their IOP increased significantly while bench pressing, reaching as high as 10 mmHg in some cases. When the Valsalva procedure was stopped, the IOP rise was partially eased [5]. Low-intensity activity has been linked to a decrease or no change in intraocular pressure [4,45-47]. In contrast, high-intensity physical activity causes a significant increase in IOP [45].

Although some clinical investigations have found that dynamic exercise can help reduce IOP in the short term [53,54], the evidence on the long-term benefits of exercise on IOP is inconsistent. One clinical trial found a reduction in IOP [55], while after 6 months of exercise, another clinical investigation found no reduction in IOP [56].

Furthermore, no population-based research has been done on the long-term link between regular exercise and IOP. After controlling for known risk variables, research in a general Japanese population found that increasing exercise frequency and time were substantially related to lower IOP levels [57]. Reitman et al. [58] looked at six newly diagnosed diabetes patients and discovered that aerobic exercise in daily routine helped decreased fasting plasma glucose levels and enhance oral glucose tolerance, allowing maintenance of glucose homeostasis. Another small investigation into human subjects found that a 45-minute bout of cycling exercise might elicit an immediate glucose-lowering impact [59].

3. Exercise, OBF, and OPP

The OPP refers to the pressure gradient indicating the flow of blood to the eye, which represents the relation between BP and IOP. The OPP is estimated as 2/3rd of the mean arterial pressure (MAP) [60]. The EPIC-Norfolk eye research, which looked at 5650 adults, found that a lower OPP was linked to less physical activity [61]. An epidemiological study reveals that, individuals with impaired OPP were more likely to develop glaucoma and experience disease progression [62].

In general, auto regulation keeps ocular blood perfusion steady and appropriate even when perfusion pressure fluctuates as a result of exercise [63-65]. Blood flow to the retina [66], choroid [21,64], and optic nerve head [23,67] are controlled to keep it consistent during activity. At high levels of effort, OPP may rise by 40–60%, at which time regulatory limitations are surpassed and blood flow has been shown to increase linearly [68]. In some people, such as those with diabetic autonomic neuropathy, who have less retinal vasoconstriction and blood flow control during exercise than those without neuropathy or diabetes, this typically protective process may be deleterious [69-70].

OBF is controlled by the interaction of vasodilators (such nitric oxide) and vasoconstrictors (like endothelin). Nitric oxide (NO) and endothelin-1 (ET-1) were found to be involved in the modulation of exercise-induced OBF but not angiotensin II, even if the precise underlying mechanism is unknown. After six minutes of the Master's double two-step test, no metabolites rose, this was accompanied by an increase in ocular blood flow [24].

Nitric oxide may be implicated in retinal autoregulation during isometric exercise, as evidenced by the fact that the response of the retinal venous diameter to 6-min hand gripping was less evident after the injection of a NO inhibitor [71]. Low diastolic perfusion pressure was identified as a significant risk factor for POAG in the Egna-Neumarkt Study [72]. Epidemiological studies have shown that individuals with impaired OPP were more likely to develop glaucoma and experience disease progression [62,50]. Although several studies have been done on the long-term effects of exercise on OBF, despite the fact that many studies have concentrated on the acute effects of exercise on OBF and OPP. A higher prevalence of glaucoma in men has been associated to vigorous daily exercise in population-based studies in South Korea [33].

Clinicians should therefore give patients with glaucoma clear instructions and suggest individualized exercise programmes with the proper intensity and frequency. If you want to know if starting an exercise programme will benefit from a chronic condition, it's critical to consider the long-term effects of fitness and activity on IOP. Numerous researches in this area have looked at IOP in both active and inactive individuals. However, the standards for athletes are sometimes ill-defined and varied among studies.

One study found that there is no statistically significant difference in the extent of the deterioration between athletes and sedentary people [32]. Another study found that when IOP was evaluated 30 minutes and 2 hours after exercise, it rapidly elevated to a substantial degree before falling in athletes. IOP dropped promptly among sedentariness and stayed low for 30 minutes and 2 hours after activity. Another study that looked at individual sexes found that male athletes had higher IOP than sedentary men, but that IOP did not vary significantly in sedentary guys. Female athletes' IOP did not alter significantly, but sedentary females' IOP decreased significantly [73].

Table 2: Effect of different types of isometric exercises and change in IOP

Type of isometric exercise	Effect on IOP	Effect on OPP and OBF	Study
Squatting	Increase in IOP from baseline	Increase in OPP and OBF	[44]
Yoga (Head down posture)	Increase in IOP	No significant change in OPP and OBF	[74]
Hand gripping	Increase in IOP	Increase in OPP and OBF	[44]
Martin's vigorimeter	No significant change in IOP	Increase in OPP and OBF	[75]

[This table shows the effects of several forms of isometric exercise on IOP. Isometric workouts have a smaller IOP lowering effect than dynamic activities, according to most studies.]

Isometric exercise has been shown to enhance OPP while simultaneously having a vasoconstrictive impact, narrowing the retinal venous and artery diameters [76]. In contrast to the differences in IOP responses to dynamic and isometric exercise, ocular perfusion responses to both forms of exercise were identical (despite the measuring techniques being different). Both dynamic and isometric exercise increased the OPP, albeit the rise was restricted due to autoregulatory response [1].

4. Psychological Health and Glaucoma

Glaucoma, being a chronic condition, can have a negative impact on patients' mental health in addition to vision impairment. Glaucoma progresses slowly, and patients are generally worried about keeping their IOP and visual field steady. Long-term stress, like other chronic diseases like cardiac disorders and hypertension [77-78], can arouse patients' fears of losing their vision, potentially leading to mental disorders like neurosis, anxiety, and depression [79-81], which can have a significant impact on patients' quality of life.

Due to the subjective nature of emotional stress, persons may have very different perspectives on the same event [82]. Numerous additional factors, like as age and personality types, were found to have an impact on people's responses to stress and IOP changes. Subjects were only allowed to describe circumstances that they subjectively considered as difficult, based on their personality and emotional threshold, because the stressful stimuli were self-reported real-life experiences.

5. Strength and resistance exercises

Numerous chronic diseases, such as diabetes, osteoporosis, hypertension, and cancer, can be benefited by regular physical activity, especially resistance training [83]. In light of this research, the benefits of physical activity are apparent, and the positive effects are significantly greater in clinical groups [84].

A customized exercise prescription is required to maximize the benefits of exercise in the therapy of the clinical condition, as standard physical activity guidelines are inadequate for the majority of medical diseases [85]. A linear and direct link exists between the resistance imposed and IOP values [88], with the size of the resistance and the participant's fitness level acting as significant modulators of the IOP changes generated by maximal cycling sprints [86] and resistance training exercises [87].

In addition, participants with higher levels of fitness showed more stable IOP behaviour compared to participants with lower levels of fitness, despite lifting the same relative weights. Prior to and after doing various resistance training configurations, researchers have examined the acute impact of strength training activities on IOP values [5,88]. There hasn't been any prior research that examined IOP behaviour during a typical resistance training session that is performed to muscle failure, despite the fact that some researchers have looked at the IOP alterations caused by a single continuous contraction [45,89].

Increased effort or the number of cumulative repetitions, during weight training exercises produces an instantaneous and progressive increase in IOP in healthy young men and women. These IOP increases showed a positive linear connection with cumulative repetitions during resistance training, leading to muscle failure, and were substantially reliant on the kind of exercise (squat > military press > biceps curl > calf raise) [18]. OPP significantly decreased after performing the squat and military press exercises, demonstrating a propensity for a reduction after strength training.

These results support earlier research showing that intense resistance training sessions alter IOP and OPP responses, however these alterations seem to be regulated by the kind of exercise and the individual [18].

Vieira et al. investigated the effects of four repetitions of the bench press exercise at 80% of one-repetition maximum (RM) with and without holding their breath, finding significant IOP increases when participants followed the bench press protocol, and even greater increases when they held their breath [45].

Rüfer et al. [5] discovered that upper limb physical anaerobic effort (20 repetitions at 65% RM on the butterfly machine) resulted in a significant increase in IOP, whereas the leg curl exercise resulted in no significant change in IOP after 20 and 10 repetitions at 65% RM, respectively. IOP rises when strength training exercises are performed in a high-intensity manner.

The extent of increases in IOP is affected by both the intensity and the kind of activity. The rise in IOP is proportional to the increase in load, and the increase in IOP is greater during the bench press throw than during the jump squat for the same relative load (% RM) [88].

Aqueous humour production was later discovered to be suppressed by endurance exercise via the release of norepinephrine produced by DA2 and DA3 receptor activation. Endurance exercise has previously been related to the systemic release of dopamine [90]. Increasing exercise duration and frequency were crucial independent variables in reducing IOP, to put it briefly. Regular exercise and IOP showed no statistically significant relationship in the subgroup analysis. Regular exercise may therefore be crucial in decreasing cholesterol [57].

6. Old-Age and Participation in Exercise

Exercise is neuroprotective for glaucoma, light-induced retinal damage, ARMD and genetically-induced photoreceptor mortality, among other retinal degenerative illnesses. Inhibiting oxidative stress, boosting BDNF expression, and lowering glutamatergic excitotoxicity are some of the putative mechanisms driving its benefits. Increased synthesis of adipokines, modulation of microglia and mitochondrial function, and influence on autophagy are all possible pathways for exercise-induced neuroprotection in conditions other than eye diseases [91].

In a study conducted in the United States (US), it was found that levels of physical activity declined rapidly during adolescence and typically persisted into early adulthood. From middle life through retirement at 65 years of age, activity habits were frequently very consistent. The incidence of frequent strenuous activities such as running, jogging, and swimming rose after retirement [92].

According to the findings of three national studies, 26.5% and 15.5% of people over 75 years old exercised at least moderately and vigorously, respectively. These higher involvement rates might be because retirees have more time available to exercise and are more aware of the health advantages [93-94].

Less vulnerable individuals are more likely to exercise frequently and are less likely to have or acquire glaucoma, which leads to the conclusions that exercise lowers the risk of having or developing glaucoma [68] and that it may be reasonable to encourage dynamic exercise in glaucoma patients [4,95-97]. On the other contrary, any increase in IOP during exercise may challenge this theory and raise the incidence of glaucoma in habitually exercising individuals.

6.1 Valsalva Manoeuvre

Various IOP-related actions depend on the Valsalva manoeuvres (VMs), which take place while the glottis is closed. Partial VMs that are accompanied by increased expiratory effort are also important [98]. IOP was found to rise along with and remain stable during increasing expiratory effort as determined by tomography [99].

For example, deep breathing can result in an IOP rise and decrease of up to 5 mmHg [100]. The effects of respiration on IOP can be much bigger depending on how much VM phenomena are active. When given a VM, some participants' IOP was reported to increase, notably in myopic participants, to above 40 mmHg [101]. Particularly in emphysematous patients, higher intrathoracic pressure was needed to obtain a given respiratory flow at lower lung inflation levels [102]. With aging comes a reduction in lung capacity, which may require deeper breathing during exercise and limit the spectrum of exercise intensity.

When doing many VM-related tasks, abdominal muscle activity increases intrathoracic pressure [98]. For example, because to the increased abdominal and expiratory muscle activity brought on by balloon inflation, significant VM stress is produced in cycles of rapid increases and falls in both intra-abdominal and intrathoracic pressure [103].

Similar cycles occur as a result of increasing respiratory volumes and the intensity of physical activity. During both static and dynamic lifting exercises, intra-abdominal pressure has been demonstrated to increase regularly [104]. During lifting jobs, breath management plays a key role in generating intra-abdominal pressure magnitude [74]. IOP rises considerably during a bench press workout and much more so when you hold your breath [45].

Dickerman described a substantial increase in IOP in power athletes after performing the Valsalva maneuver, which reached 46 mmHg in certain cases. During an isometric contraction, the mean IOP climbed from 13 mmHg at rest to 28 mmHg, and these substantial pressure fluctuations lasted 3 to 8 seconds [74,89,90,108]. According to the research, OPA (ocular pulse amplitude) measures in healthy young boys are unaffected by VM. However, the majority of these people (83.6%) had a substantial increase in IOP, whereas nine people (16.4%) had a decrease in IOP from baseline [7].

7. Effect of caffeine consumption on IOP

Although Vera et al. investigated the combined effects of low-intensity endurance exercise and caffeine consumption on IOP [105], there is a growing body of data associating caffeine usage to an increase in IOP [105,106]. According to studies conducted on caffeine's effects on physical performance [107,108], caffeine's effects on IOP during low-intensity aerobic activity are unrelated to the heart rate response. However, ocular perfusion pressure, which is controlled by blood pressure and IOP levels, plays a significant role in the development of glaucoma [109].

Future studies should examine the impact of caffeine consumption before exercise on ocular perfusion because there is evidence that acute caffeine intake increases blood pressure. Hunt et al. [110] found a correlation between the degree of dehydration and the drop in IOP brought on by low-intensity aerobic activity. Contrary to popular belief, caffeine does not cause dehydration when consumed before to exercise, according to empirical study [111,112].

By taking a caffeine pill (4 mg/kg) 30 minutes before cycling, the IOP-lowering reaction to low-intensity endurance exercise was prevented. During the activity, IOP levels were reduced in the placebo group, but they remained steady following coffee administration. Caffeine consumption before a low-intensity endurance exercise should be avoided when physical activity is advised to lower IOP levels, according to these data (i.e., glaucoma patients or those at risk) [105].

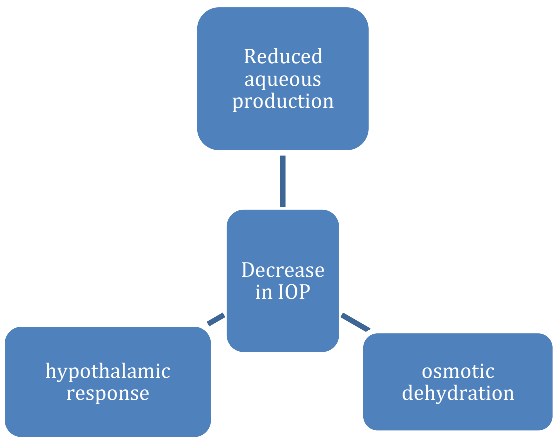

Fig 1: Three mechanisms for reduction of IOP are depicted in the figure i.e., a) Osmotic dehydration b) Reduced aqueous production and c) Hypothalamic response

8. Physical Activities and IOP

8.1 Swimming

Swimming is one of the most widely practiced sports. Swimming goggles, on the other hand, might significantly raise IOP. After putting on the swimming goggles, a tiny but substantial IOP rise of 2.3 mmHg was detected, which remained high until the goggles were removed [113]. According to Morgan et al. research [114], wearing goggles led to a 4.5 mmHg rise in IOP, which was associated with a smaller goggle/face area.

The short IOP increase did not affect the retinal nerve fibre layer in healthy participants [113], but it could worsen RGC loss in an already compromised optic nerve, such as in glaucoma. According to the research, it appears that swimming goggles temporarily increase IOP in healthy individuals but significantly in glaucoma patients, leading to irreparable harm. Glaucoma patients should receive advice regarding wearing swimming goggles indiscriminately.

8.2 Yoga

Yoga is the most extensively done exercise in India with some believing it to be a viable alternative to traditional medical care [115]. Some yoga poses can be considered isometric workouts and may include body inversions, which have been shown to have a significant effect on IOP [103,116]. In four popular head-down yoga postures, Halasana, Adho Mukha Svanasana, and Viparita Karani; Jessica et al. noticed changes in IOP in both glaucoma patients and healthy individuals. He found that both POAG patients and normal subjects have IOP elevations ranging from 16 to 70 percent in all four positions, with no statistically significant difference between the two groups [116]. There is proof that the transient increase in IOP in Sirsasana causes the onset of glaucoma. A 46-year-old patient with NTG [117] and a 47-year-old woman with a history of congenital glaucoma both suffered progressive optic neuropathy and visual field loss after years of consistent practise with Sirsasana.

IOP and EVP are related, as was already mentioned. Both animal [118-119] and human [120] studies have validated the rise in EVP during the head-down position. A rise in cerebral cerebrospinal fluid pressure, which indirectly influences the choroid veins that drain into the superior ophthalmic vein and then into the intracranial cavernous sinus, may also cause the choroidal thickness to grow in head-down posture in addition to EVP [116,121]. As a result, these head-down postures may increase the risk of glaucoma development and should be avoided in those who have or are at high risk of glaucoma.

Table 3: Physical activities and change in IOP (Intraocular pressure) and OPP (Ocular perfusion pressure)

Activities	Effect on IOP	Effect On OPP	Other Findings	Study
Dynamic and aerobic exercises	Short-term decrease in IOP	Increase in OPP	The Effect is more in sedentary population than active population. Increases blood flow in retinal and choroidal vessels.	[133]
Isometric exercises	Increase in IOP	Increase in OPP	This change may be related to changes in BP and the Valsalva Manoeuvre. There was no change in IOP as long as Valsalva manoeuvre is avoided.	[45]
Valsalva manoeuvre	Increase in IOP	No significant change in OPP	More in myopic patients	[5]
Caffeine consumption	Increase in IOP	No significant change in OPP	Caffeine was taken 30 minutes before exercise	[134]
Swimming goggles	Increase in IOP	No significant change in OPP	It was linked to smaller goggles/ face area	[114]
Yoga (Head down posture)	Increase in IOP	No significant change in OPP	The effect was due to head-down positions, an increase in episcleral venous pressure, and an increase in choroidal thickness.	[74]
Bungee jumping	Increase in IOP	No significant change in OPP	The impact is more in people with shallow anterior chamber angle. This increase may be due to head down position during bungee jumping.	[123]
Alcohol	Temporary drop in IOP	No significant change in OPP	No other findings	[130]

(This table depicts how various activities, like workouts and other activities, affect IOP levels in both sedentary and sporty individuals.)

9. Bungee jumping

In order to avoid hitting the ground, bungee jumping involves jumping and diving from a height while wearing a long elastic string around the body. The body quickly slows down as the cord reaches its maximum length during free fall, but inertia causes the blood in the arteries to keep moving toward the top of the head, increasing the hydrostatic pressure in the blood vessels of the eye [122].

The head-down position and the force of the impact could both increase IOP, even if increases have not been observed during bungee jumping, especially in people with shallow anterior chambers. However, bungee jumping has been linked to a variety of ocular complications, including subconjunctival hemorrhage [123-124], pre-retinal and retinal hemorrhage [124-127], macular edema [124,127], and bungee as a result, people with glaucoma should be advised about the hazards of bungee jumping.

10 Alcohol

Alcohol use is generally believed to be hazardous for those with glaucoma. There hasn't been much research on the direct impact of dietary ethanol on IOP, and it's possible that alcohol's neurotoxic effects play a role in the association between drinking and the advancement of glaucoma [128–129]. In a single case study, drinking Champagne caused a systematic transient reduction in IOP in a 66-year-old glaucoma patient [130].

This could be explained by alcohol's biphasic effect, which causes a decrease in blood pressure for up to 12 hours before increasing [131], which could be mirrored by IOP, as Takashi et al. suggested in a large cross-sectional study of 1569 subjects showing a positive relationship between systolic blood pressure and IOP [132].

The aforementioned research indicates that the majority of routine daily activities have a considerable impact on IOP, which may be particularly crucial for glaucoma patients. This highlights the need of lifestyle advice for glaucoma patients. This may support the use of telemetry for continuous monitoring of IOP variations in borderline glaucoma cases to help physicians weigh the advantages of early surgical intervention against the patient's "normal" daily activities [133-137]. However, it would be necessary to ascertain how surgical treatments might affect IOP changes away from the office.

11. Oxidative stress, mitochondrial function, and PDG

Variations in the gene coding for mitochondrial protein are frequently observed in POAG patients, according to a recent epidemiological research of 3108 healthy people and 3430 patients with POAG. This shows that the pathophysiology of POAG may be significantly influenced by mitochondria [138]. According to Lenaz et al. [139], the principal source of intracellular reactive oxygen species (ROS), which causes RGC apoptosis [139,140], is the mitochondrial electron transport chain. Reactive oxygen species (ROS) harm mitochondria, causing cell death, starting with the opening of the membrane permeability transition pore (MPTP) [141].

Exercise has been shown to support mitochondrial biogenesis and assist reverse mitochondrial dysfunction. By boosting membrane potential and matrix oxidant load under hypoxic settings, HIT and moderate-intensity continuous training have both been demonstrated to improve lymphocyte mitochondrial dysfunction in humans [142]. Four weeks of exercise preconditioning on the free-running wheel were found to reduce hyperglycemia in a mouse model of type I diabetes by enhancing skeletal muscle mitochondrial activity [143].

Exercise has been demonstrated to improve MPTP resistance, mitochondrial physiology, reduce the production of ROS, and start the production of new mitochondria in the brain [144–145]. The positive impact of exercise on mitochondria may one day be used to treat Alzheimer's disease, a neurodegenerative condition of the brain [146]. Retinal ganglion cells (RGCs) are more vulnerable to mitochondrial dysfunction because they have long axons and a high density of mitochondria, which provides energy to transport proteins to distant axons [147]. Since glaucomatous damage can lead to RGC loss, the exercise-induced mitochondrial increase may be helpful in preventing this loss.

Oxidative stress, which is known to contribute to the vulnerability of aged retinas, is thought to exacerbate a number of eye diseases, including glaucoma and AMD. It has been demonstrated in mice that treadmill training benefits ageing retinas by lowering oxidative damage [37]. The central nervous system (CNS) ageing process and a number of neurodegenerative diseases, including Alzheimer's and Parkinson's disease, have long been linked to oxidative stress [148–149]. Exercise over an extended period of time has been demonstrated to lower oxidative stress and increase antioxidant capacity in the elderly. Additionally, retinal neuropathology is expected to contain a large amount of oxidative stress [150].

According to Kim et al., treadmill training is protective for aged retinas since it reduces oxidative stress in mice by down-regulating the expressions of carboxymethyl lysine (CML), 8-hydroxy-2-deoxyguanosine (8-OHdG), and nitrotyrosine, as seen by decreased immunological reactivities [35].

In the process of developing exercise-induced pigment dispersion, individuals with the pigmentary subtype of glaucoma demonstrated an increase in IOP [151]. Exercise would be of minimal value to these people, and so it should not be suggested. More research is needed to determine the criteria for encouraging glaucoma sufferers to exercise regularly. During exercise, young individuals with advanced glaucoma have been observed to undergo a "vascular steal," which can cause temporary vision loss [152]. Exercising causes a rise in IOP in individuals with the pigmentary subtype of glaucoma, which has been recognized for a long time [151,153-155]. Exercise should not be advocated for these people since it may be hazardous.

Conclusion

Dynamic exercise can lower IOP in glaucoma patients as well as healthy or myopic people (except in those with PDG or PDS). In healthy individuals, swimming with goggles is typically viewed as safe, although it has the potential to increase IOP to risky levels, which could result in disease development. Due to the risks, extreme sports like bungee jumping should be avoided. Despite the fact that exercise increases OPP, autoregulation in healthy individuals lessens the impact on ocular circulation. The potential impact of physical activity on lowering OBF and IOP in glaucoma patients need further investigation.

In addition to IOP and OBF, the effects of exercise on neuroprotection and mental health in glaucoma patients were explored. Increasing BDNF and enhancing mitochondrial function may aid in preventing vision loss brought on by RGC mortality brought on by glaucoma. Last but not least, physical activity can help glaucoma sufferers feel less anxious and depressed, which will improve their quality of life. Anxiety and depression are common in people with glaucoma.

In conclusion, this study's general public population found that increasing exercise frequency and duration were significant independent variables in reducing IOP. Regular exercise and IOP showed no statistically significant relationship in the subgroup analysis. As a result, regular exercise may be crucial in reducing IOP. IOP decreases following acute exercise in both healthy and glaucomatous subjects, and exercise training lowers IOP levels at rest.

Vascular auto regulation in healthy individuals results in stable OBF during exercise despite increases in OPP, systemic blood pressure, and heart rate. Since elevated IOP and vascular abnormalities are known to be risk factors for the onset of glaucoma, these issues must be taken into account when providing care for glaucoma patients. Although there is a wealth of outstanding research and articles in the existing literature, many of them do not address the same issues since they fall outside the purview of that particular study.

Exercise is neuroprotective for glaucoma, AMD, light-induced retinal damage, and genetically-induced photoreceptor mortality, among other retinal degenerative illnesses. Inhibiting oxidative stress, boosting BDNF, and lowering glutamatergic excitotoxicity are some of the putative mechanisms driving its benefits. Other mechanisms that might explain exercise's neuroprotective effects on illnesses other than eye diseases include increased adipokine production and modulation of microglia and mitochondrial function.

Funding- NIL

Conflicts of interest- NIL

References

[1]. Zhu MM, Lai JSM, Choy BNK, Shum JWH, Lo ACY, Ng ALK, et al. Physical exercise and glaucoma: a review on the roles of physical exercise on intraocular pressure control, ocular blood flow regulation, neuroprotection and glaucoma-related mental health. Acta Ophthalmol. 2018;96(6):e676–91.

[2]. Zhao D, Kim MH, Pastor-Barriuso R, Chang Y, Ryu S, Zhang Y, et al. A longitudinal study of association between adiposity markers and intraocular pressure: The kangbuk samsung health study. PLoS One. 2016;11(1).

[3]. L. A, et al. Circadian intraocular pressure patterns in healthy subjects, primary open angle and normal tension glaucoma patients with a contact lens sensor. Acta Ophthalmol [Internet]. 2015;93(1):e14–21.

[4]. 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(6):429–36.

[5]. 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(3):249–52.

[6]. Vera J, Jiménez R, García JA, Cárdenas D. Intraocular pressure is sensitive to cumulative and instantaneous mental workload. Appl Ergon. 2017;60:313–9.

[7]. Aykan U, Erdurmus M, Yilmaz B, Bilge AH. Intraocular pressure and ocular pulse amplitude variations during the Valsalva maneuver. Graefe's Arch Clin Exp Ophthalmol. 2010;248(8):1183–6.

[8]. Vera J, Diaz-Piedra C, Jiménez R, Morales JM, Catena A, Cardenas D, et al. Driving time modulates accommodative response and intraocular pressure. Physiol Behav. 2016;164:47–53.

[9]. Lichter PR, Musch DC, Gillespie BW, Guire KE, Janz NK, Wren PA, et al. Interim clinical outcomes in the Collaborative Initial Glaucoma Treatment Study comparing initial treatment randomized to medications or surgery. Ophthalmology. 2001;108(11):1943–53.

[10]. Schulzer M, Drance SM, Douglas GR. A Comparison of Treated and Untreated Glaucoma Suspects. Ophthalmology. 1991;98(3):301–7.

[11]. Migdal C, Hitchings R. Control of chronic simple glaucoma with primary medical, surgical and laser treatment. Trans Ophthalmol Soc U K. 1986;105(6):653–6.

[12]. Migdal C, Hitchings R. Primary therapy for chronic simple glaucoma. The role of argon laser trabeculoplasty. Trans Ophthalmol Soc U K. 1985;104(1):62–6.

[13]. Smith RJ. Medical versus surgical therapy in glaucoma simplex. Br J Ophthalmol. 1972;56(3):277–83.

[14]. R S. A comparison between medical and surgical treatment of glaucoma simplex--results of a prospective study. Trans Ophthalmol Soc Aust. 1968;27:17–29.

[15]. Arend O, Plange N, Sponsel WE, Remky A. Pathogenetic aspects of the glaucomatous optic

neuropathy: Fluorescein angiographic findings in patients with primary open angle glaucoma. Brain Res Bull. 2004;62(6):517–24.

[16]. Flammer J, Orgül S, Costa VP, Orzalesi N, Krieglstein GK, Serra LM, et al. The impact of ocular blood flow in glaucoma. Prog Retin Eye Res. 2002;21(4):359–93.

[17]. B. Siesky et al; The clinical significance of vascular risk factors in glaucoma; Journal of Current Glaucoma Practice, Sept.-Dec. 2007;1(2):12-17

[18]. Vera J, Jiménez R, Redondo B, Torrejón A, De Moraes CG, García-Ramos A. Effect of the level of effort during resistance training on intraocular pressure. Eur J Sport Sci. 2019;19(3):394–401.

[19]. Anderson DR. Introductory comments on blood flow autoregulation in the optic nerve head and vascular risk factors in glaucoma. Surv Ophthalmol. 1999;43(6 SUPPL.):S5.

[20]. Németh J, Knézy K, Tapasztó B, Kovás R, Harkányi Z. Different autoregulation response to dynamic exercise in ophthalmic and central retinal arteries: A color Dopper study in healthy subjects. Graefe's Arch Clin Exp Ophthalmol. 2002;240(10):835–40.

[21]. Lovasik J V., Kergoat H, Riva CE, Petrig BL, Geiser M. Choroidal blood flow during exercise-induced changes in the ocular perfusion pressure. Investig Ophthalmol Vis Sci. 2003;44(5):2126–32.

[22]. Morgan AJ, Hosking SL. Non-invasive vascular impedance measures demonstrate ocular vasoconstriction during isometric exercise. Br J Ophthalmol. 2007;91(3):385–90.

[23]. Movaffaghy A, Chamot SR, Petrig BL, Riva CE. Blood flow in the human optic nerve head during isometric exercise. Exp Eye Res. 1998;67(5):561–8.

[24]. Okuno T, Sugiyama T, Kohyama M, Kojima S, Oku H, Ikeda T. Ocular blood flow changes after dynamic exercise in humans. Eye. 2006;20(7):796–800.

[25]. 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(6):460–6.

[26]. Lovasik J V., Kergoat H. Consequences of an increase in the ocular perfusion pressure on the pulsatile ocular blood flow. Optom Vis Sci. 2004;81(9):692–8.

[27]. Kiss B, Dallinger S, Polak K, Findl O, Eichler HG, Schmetterer L. Ocular hemodynamics during isometric exercise. Microvasc Res. 2001;61(1):1–13.

[28]. Riva CE, Titze P, Hero M, Movaffaghy A, Petrig BL. Choroidal blood flow during isometric exercises. Investig Ophthalmol Vis Sci. 1997;38(11):2338–43.

[29]. M.H.M. DM, et al. Regular exercise, anxiety, depression and personality: A population-based study. Prev Med (Baltim) [Internet]. 2006;42(4):273–9.

[30]. Stubbs B, Koyanagi A, Schuch FB, Firth J, Rosenbaum S, Veronese N, et al. Physical activity and depression: a large cross-sectional, population-based study across 36 low- and middle-income countries. Acta Psychiatr Scand. 2016;134(6):546–56.

[31]. B. S, et al. Physical activity levels and psychosis: A mediation analysis of factors influencing physical activity target achievement among 204 186 people across 46 low- and middle-income countries. Schizophr Bull [Internet]. 2017;43(3):536–45.

[32]. Ozmerdivenli R, Simsek E, Bulut S, Karacabey K, Saygin O. Comparison of the effects of acute and regular exercise on intraocular pressure in Turkish athlete and sedentarians. Int J Neurosci. 2006;116(3):351–60.

[33]. Lin JB, Tsubota K, Apte RS. A glimpse at the aging eye. *NPJ Aging Mech Dis*. 2016;2:16003. Published 2016 Mar 10. doi:10.1038/npjamd.2016.3

[34]. Crowston JG, Fahy ET, Fry L, Trounce IA, Van Wijngaarden P, Petrou S, et al. Targeting retinal ganglion cell recovery. Eye. 2017;31(2):196–8.

[35]. Chrysostomou V, Kezic JM, Trounce IA, Crowston JG. Forced exercise protects the aged optic nerve against intraocular pressure injury. Neurobiol Aging. 2014;35(7):1722–5.

[36]. Chrysostomou V, Galic S, van Wijngaarden P, Trounce IA, Steinberg GR, Crowston JG. Exercise reverses age-related vulnerability of the retina to injury by preventing complement-mediated synapse elimination via a BDNF-dependent pathway. Aging Cell. 2016;15(6):1082–91.

[37]. Michelini LC, Stern JE. Exercise-induced neuronal plasticity in central autonomic networks: Role in cardiovascular control. Exp Physiol. 2009;94(9):947–60.

[38]. Alvarez-Mejia L, Morales J, Cruz GJ, Olayo MG, Olayo R, Díaz-Ruíz A, et al. Functional recovery in spinal cord injured rats using polypyrrole/iodine implants and treadmill training. J Mater Sci Mater Med. 2015;26(7).

[39]. Sandrow-Feinberg HR, Houlé JD. Exercise after spinal cord injury as an agent for neuroprotection, regeneration and rehabilitation. Brain Res. 2015;1619:12–21.

[40]. Chali F, Desseille C, Houdebine L, Benoit E, Rouquet T, Bariohay B, et al. Long-term exercise-specific neuroprotection in spinal muscular atrophy-like mice. J Physiol. 2016;594(7):1931–52.

[41]. Hüttenrauch M, Braub A, Kurdakova A, Borgers H, Klinker F, Liebetanz D, et al. Physical activity delays hippocampal neurodegeneration and rescues memory deficits in an Alzheimer disease mouse model. Transl Psychiatry. 2016;6.

[42]. Otsuka S, Sakakima H, Sumizono M, Takada S, Terashi T, Yoshida Y. The neuroprotective effects of preconditioning exercise on brain damage and neurotrophic factors after focal brain ischemia in rats. Behav Brain Res. 2016;303:9–18.

[43]. Li L, Xu M, Shen B, Li M, Gao Q, Wei SG. Moderate exercise prevents neurodegeneration in D-galactose-induced aging mice. Neural Regen Res. 2016;11(5):807–15.

[44]. Castejon H, Chiquet C, Savy O, Baguet JP, Khayi H, Tamisier R, et al. Effect of acute increase in blood pressure on intraocular pressure in pigs and humans. Investig Ophthalmol Vis Sci. 2010;51(3):1599–605.

[45]. Shephard RJ. Intraocular Pressure Variation During Weight Lifting. Yearb Sport Med. 2007;2007:186–7.

[46]. Natsis K, Asouhidou I, Nousios G, Chatzibalis T, Vlasis K, Karabatakis V. Aerobic exercise and intraocular pressure in normotensive and glaucoma patients. BMC Ophthalmol. 2009;9(1).

[47]. Read SA, Collins MJ. The short-term influence of exercise on axial length and intraocular pressure. Eye. 2011;25(6):767–74.

[48]. Marcus DF, Krupin T, Podos SM, Becker B. The effect of exercise on intraocular pressure. I. Human beings. Invest Ophthalmol. 1970;9(10):749–52.

[49]. Kielar RA, Teraslinna P, Rowe DG, Jackson J. Standardized aerobic and anaerobic exercise: differential effects on intraocular tension, blood pH, and lactate. Invest Ophthalmol. 1975;14(10):782–5.

[50]. Leske MC, Heijl A, Hyman L, Bengtsson B, Dong LM, Yang Z. Predictors of Long-term Progression in the Early Manifest Glaucoma Trial. Ophthalmology. 2007;114(11):1965–72.

[51]. Leighton DA, Phillips CI. Effect of moderate exercise on the ocular tension. Br J Ophthalmol. 1970;54(9):599–605.
[52]. Dane Ş, Koçer I, Demirel H, Ucok K, Tan Ü. Effect of acute submaximal exercise on intraocular pressure in athletes and sedentary subjects. Int J Neurosci. 2006;116(10):1223–30.
[53]. Ashkenazi I, Melamed S, Blumenthal M. The effect of continuous strenuous exercise on intraocular pressure. Investig Ophthalmol Vis Sci. 1992;33(10):2874–7.
[54]. Harris A, Malinovsky V, Martin B. Correlates of acute exercise-induced ocular hypotension. Investig Ophthalmol Vis Sci. 1994;35(11):3852–7.
[55]. Qureshi IA, Xi XR, Wu XD, Zhang J, Shiarkar E. The effect of physical fitness on intraocular pressure in Chinese medical students. 1996;58(5):317–22.
[56]. Sargent RG, Blair SN, Magun JC, Krejci RC, Sacoco C, Langley TD, et al. Physical fitness and intraocular pressure. Optom Vis Sci. 1981;58(6):460–6.
[57]. Fujiwara K, Yasuda M, Hata J, Yoshida D, Kishimoto H, Hashimoto S, et al. Long-term regular exercise and intraocular pressure: the Hisayama Study. Graefe's Arch Clin Exp Ophthalmol. 2019;257(11):2461–9.
[58]. Reitman JS, Vasquez B, Klimes I, Nagulesparan M. Improvement of glucose homeostasis after exercise training in non-insulin-dependent diabetes. Diabetes Care. 1984;7(5):434–41.
[59]. Zinman B, Zuniga-Guajardo S, Kelly D. Comparison of the acute and long-term effects of exercise on glucose control in type I diabetes. Diabetes Care. 1984;7(6):515–9.
[60]. Riva CE, Grunwald JE, Sinclair SH, Petrig BL. Blood velocity and volumetric flow rate in human retinal vessels. Investig Ophthalmol Vis Sci. 1985;26(8):1124–32.
[61]. Zhang Y, Emeterio Nateras OS, Peng Q, Rosende CA, Duong TQ. Blood flow MRI of the human retina/choroid during rest and isometric exercise. Investig Ophthalmol Vis Sci. 2012;53(7):4299–305.
[62]. Quigley HA, West SK, Rodriguez J, Munoz B, Klein R, Snyder R. The prevalence of glaucoma in a population-based study of Hispanic subjects: Proyecto VER. Arch Ophthalmol. 2001;119(12):1819–26.
[63]. Boltz A, Told R, Napora KJ, Palkovits S, Werkmeister RM, Schmidl D, et al. Optic nerve head blood flow autoregulation during changes in arterial blood pressure in healthy young subjects. PLoS One. 2013;8(12).
[64]. Riva CE, Hero M, Titze P, Petrig B. Autoregulation of human optic nerve head blood flow in response to acute changes in ocular perfusion pressure. Graefe's Arch Clin Exp Ophthalmol. 1997;235(10):618–26.
[65]. Iester M, Torre PG, Bricola G, Bagnis A, Calabria G. Retinal blood flow autoregulation after dynamic exercise in healthy young subjects. Ophthalmologica. 2007;221(3):180–5.
[66]. Robinson F, Riva CE, Grunwald JE, Petrig BL, Sinclair SH. Retinal blood flow autoregulation in response to an acute increase in blood pressure. Investig Ophthalmol Vis Sci. 1986;27(5):722–6.
[67]. Pournaras CJ, Riva CE, Bresson-Dumont H, De Gottrau P, Bechetoille A. Regulation of optic nerve head blood flow in normal tension glaucoma patients. Eur J Ophthalmol. 2004;14(3):226–35.
[68]. J. G,et al. Effects of Exercise on Ocular Physiology and Disease. Surv Ophthalmol [Internet]. 2009;54(3):349–55
[69]. Lanigan LP, Clark C V., Hill DW. Retinal circulation responses to systemic autonomic nerve stimulation. Eye. 1988;2(4):412–7.
[70]. Lanigan LP, Clark C V., Allawi J, Hill DW, Keen H. Responses of the retinal circulation to systemic autonomic stimulation in diabetes mellitus. Eye. 1989;3(1):39–47.
[71]. Lasta M, Polak K, Luksch A, Garhofer G, Schmetterer L. Effect of NO synthase inhibition on retinal vessel reaction to isometric exercise in healthy humans. Acta Ophthalmol. 2012;90(4):362–8.
[72]. L B, et al. Vascular risk factors for primary open angle glaucoma: the Egna-Neumarkt Study. Ophthalmology. 2000;107(7):1287–93.
[73]. Dane Ş, Koçer I, Demirel H, Üçok K, Tan Ü. Long-term effects of mild exercise on intraocular pressure in athletes and sedentary subjects. Int J Neurosci. 2006;116(10):1207–14.
[74]. Baskaran M, Raman K, Ramani KK, Roy J, Vijaya L, Badrinath SS. Intraocular Pressure Changes and Ocular Biometry during Sirsasana (Headstand Posture) in Yoga Practitioners. Ophthalmology. 2006;113(8):1327–32.
[75]. Portmann N, Gugleta K, Kochkorov A, Polunina A, Flammer J, Orgul S. Choroidal blood flow response to isometric exercise in glaucoma patients and patients with ocular hypertension. Investig Ophthalmol Vis Sci. 2011;52(10):7068–73.
[76]. Luksch A, Wimpissinger B, Polak K, Jandrasits K, Schmetterer L. ETa-receptor blockade, but not ACE inhibition, blunts retinal vessel response during isometric exercise. Am J Physiol - Hear Circ Physiol. 2006;290(4).
[77]. Majani G, Pierobon A, Giardini A, Callegari S, Opasich C, Cobelli F, et al. Relationship between psychological profile and cardiological variables in chronic heart failure. The role of patient subjectivity. Eur Heart J. 1999;20(21):1579–86.
[78]. Härter MC, Conway KP, Merikangas KR. Associations between anxiety disorders and physical illness. Eur Arch Psychiatry Clin Neurosci. 2003;253(6):313–20.
[79]. Mabuchi F, Yoshimura K, Kashiwagi K, Shioe K, Yamagata Z, Kanba S, et al. High prevalence of anxiety and depression in patients with primary open-angle glaucoma. J Glaucoma. 2008;17(7):552–7.
[80]. Warrian KJ, Spaeth GL, Lankaranian D, Lopes JF, Steinmann WC. The effect of personality on measures of quality of life related to vision in glaucoma patients. Br J Ophthalmol. 2009;93(3):310–5.
[81]. Çakmak H, Altinyazar V, Yilmaz SG, Ömürlü IK, Kocatürk T, Yazici A, et al. The temperament and character personality profile of the glaucoma patient Glaucoma. BMC Ophthalmol. 2015;15(1).
[82]. Terracciano A, Scuteri A, Strait J, Sutin AR, Meirelles O, Marongiu M, Orru M, Pilia MG, Ferrucci L, Cucca F, Schlessinger D, Lakatta E. Are personality traits associated with white-coat and masked hypertension? J Hypertens. 2014 Oct;32(10):1987-92; discussion 1992.
[83]. Warburton DE, Bredin SS. Reflections on Physical Activity and Health: What Should We Recommend? Can J Cardiol. 2016;32(4):495–504.
[84]. Jones M. Therapeutic Exercise: Foundations and techniques. Physiotherapy. 1991;77(2):96.
[85]. Pedersen B. K. SB. Evidence for prescribing exercise as therapy in chronic disease. Scand J Med Sport. 2015;3–63.
[86]. Vera J, Jiménez R, Redondo B, Cárdenas D, De Moraes CG, Ramos AG. Intraocular pressure responses to maximal cycling sprints against different resistances: The influence of fitness level. J Glaucoma. 2017;26(10):881–7.

[87]. J. V, et al. Fitness Level Modulates Intraocular Pressure Responses to Strength Exercises.; Curr Eye Res;2018;43(6):740–6.
[88]. Vera J, García-Ramos A, Jiménez R, Cárdenas D. The acute effect of strength exercises at different intensities on intraocular pressure. Graefe's Arch Clin Exp Ophthalmol. 2017;255(11):2211–7.
[89]. Dickerman RD, Smith GH, Langham-Roof L, McConathy WJ, East JW, Smith AB. Intra-ocular pressure changes during maximal isometric contraction: Does this reflect intra-cranial pressure or retinal venous pressure? Neurol Res. 1999;21(3):243–6.
[90]. Mrówczyński W. Health benefits of endurance training: Implications of the brain-derived neurotrophic factor-a systematic review. Neural Plast. 2019;2019.
[91]. Li HY, Rong SS, Hong X, Guo R, Yang FZ, Liang YY, et al. Exercise and retinal health. Restor Neurol Neurosci. 2019;37(6):571–81.
[92]. Caspersen CJ, Pereira MA, Curran KM. Changes in physical activity patterns in the United States, by sex and cross-sectional age. Med Sci Sports Exerc. 2000;32(9):1601–9.
[93]. Pratt M, Macera CA, Blanton C. Levels of physical activity and inactivity in children and adults in the United States: Current evidence and research issues. Med Sci Sports Exerc. 1999;31(11 SUPPL.).
[94]. Dionigi R. A Leisure Pursuit that 'Goes Against the Grain': Older People and Competitive Sport. Ann Leis Res. 2005;8(1):1–22.
[95]. Chromiak JA, Abadie BR, Braswell RA, Koh YS, Chilek DR. Resistance Training Exercises Acutely Reduce Intraocular Pressure in Physically Active Men and Women. J Strength Cond Res. 2003;17(4):715–20.
[96]. Passo MS, Elliot DL, Goldberg L. Long-term effects of exercise conditioning on intraocular pressure in glaucoma suspects. J Glaucoma. 1992;1(1):39–41.
[97]. Williams PT. Relationship of incident glaucoma versus physical activity and fitness in male runners. Med Sci Sports Exerc. 2009;41(8):1566–72.
[98]. Porth CJM, Bamrah VS, Tristani FE, Smith JJ. The valsalva maneuver: Mechanisms and clinical implications. Hear Lung J Acute Crit Care. 1984;13(5):507–18.
[99]. Rosen DA, Johnston VC. Ocular Pressure Patterns in the Valsalva Maneuver. AMA Arch Ophthalmol. 1959;62(5):810–6.
[100]. Hugh Davson; Physiology of the Eye. Physiol Eye. 1990; 5th edition.
[101]. Oggel K, Sommer G, Neuhann T, Hinz J; Variations of intraocular pressure during valsalva's maneuver in relation to body position and length of the bulbus in myopia (author's transl)]. Graefes Arch Clin Exp Ophthalmol. 1982;218(1):51–4.
[102]. Fry DL, Ebert R V., Stead WW, Brown CC. The mechanics of pulmonary ventilation in normal subjects and in patients with emphysema. Am J Med. 1954;16(1):80–97.
[103]. Georgiou T, Pearce IA, Taylor RH. Valsalva retinopathy associated with blowing balloons [14]. Eye. 1999;13(5):686–7.
[104]. Hagins M, Pietrek M, Sheikhzadeh A, Nordin M, Axen K. The Effects of Breath Control on Intra-Abdominal Pressure during Lifting Tasks. Spine (Phila Pa 1976). 2004;29(4):464–9.
[105]. Vera J, Redondo B, Molina R, Bermúdez J, Jiménez R. Effects of caffeine on intraocular pressure are subject to tolerance: a comparative study between low and high caffeine consumers. Psychopharmacology (Berl). 2019;236(2):811–9.
[106]. Hogervorst E, Bandelow S, Schmitt J, Jentjens R, Oliveira M, Allgrove J, et al. Caffeine improves physical and cognitive performance during exhaustive exercise. Med Sci Sports Exerc. 2008;40(10):1841–51.
[107]. Hodgson AB, Randell RK, Jeukendrup AE. The Metabolic and Performance Effects of Caffeine Compared to Coffee during Endurance Exercise. PLoS One. 2013;8(4).
[108]. McNaughton LR, Lovell RJ, Siegler J, Midgley AW, Moore L, Bentley DJ. The effects of caffeine ingestion on time trial cycling performance. Int J Sports Physiol Perform. 2008;3(2):157–63.
[109]. Costa VP, Harris A, Anderson D, Stodtmeister R, Cremasco F, Kergoat H, et al. Ocular perfusion pressure in glaucoma. Acta Ophthalmol. 2014;92(4).
[110]. Hunt AP, Feigl B, Stewart IB. The intraocular pressure response to dehydration: a pilot study. Eur J Appl Physiol. 2012 May;112(5):1963-6.
[111]. Magkos F, Kavouras SA. Caffeine and exercise: metabolism, endurance and performance. Sports Med. 2004;34(13):871–89.
[112]. Armstrong LE, Casa DJ, Maresh CM, Ganio MS. Caffeine, fluid-electrolyte balance, temperature regulation, and exercise-heat tolerance. Exerc Sport Sci Rev. 2007;35(3):135–40.
[113]. Ma KT, Chung WS, Seo KY, Seong GJ, Kim CY. The effect of swimming goggles on intraocular pressure and blood flow within the optic nerve head. Yonsei Med J. 2007;48(5):807–9.
[114]. Morgan WH, Cunneen TS, Balaratnasingam C, Yu DY. Wearing swimming goggles can elevate intraocular pressure. Br J Ophthalmol. 2008;92(9):1218–21.
[115]. Nayak NN, Shankar K. Yoga: A therapeutic approach. Phys Med Rehabil Clin N Am. 2004;15(4):783–98.
[116]. Jasien J V., Jonas JB, Gustavo De Moraes C, Ritch R. Intraocular pressure rise in subjects with and without glaucoma during four common yoga positions. PLoS One. 2015;10(12).
[117]. Gallardo MJ, Aggarwal N, Cavanagh HD, Whitson JT. Progression of glaucoma associated with the sirsasana (headstand) yoga posture. Adv Ther. 2006;23(6):921–5.
[118]. Lavery WJ, Kiel JW. Effects of head down tilt on episcleral venous pressure in a rabbit model. Exp Eye Res. 2013;111:88–94.
[119]. Abe T, Mizobuchi Y, Sako W, Irahara S, Otomi Y, Obama Y, et al. Clinical significance of discrepancy between arterial spin labeling images and contrast-enhanced images in the diagnosis of brain tumors. Magn Reson Med Sci. 2015;14(4):313–9.
[120]. Friberg TR, Sanborn G, Weinreb RN. Intraocular and episcleral venous pressure increase during inverted posture. Am J Ophthalmol. 1987;103(4):523–6.
[121]. Jost B Jonas et al; Subfoveal choroidal thickness and cerebrospinal fluid pressure: Investigative Ophthalmology & Visual Science March 2014, Vol.55, 1292-1298.
[122]. Simons R, Krol J. Visual loss from bungee Jumping. Lancet. 1994;343(8901):853.
[123]. Chan J. Ophthalmic complications after bungee jumping. Br J Ophthalmol. 1994;78(3):239.
[124]. David DB, Mears T, Quinlan MP. Ocular complications associated with bungee lumping. Br J Ophthalmol. 1994;78(3):234–5.
[125]. Capão Filipe JA, Pinto AM, Rosas V, Castro-Correia J. Retinal complications after bungee jumping. Int Ophthalmol. 1994;18(6):359–60.

[126]. Jain BK, Talbot EM. Bungee jumping and intraocular haemorrhage. Br J Ophthalmol. 1994;78(3):236–7.

[127]. Curtis EB, Collin HB. Ocular injury due to bungee jumping. Clin Exp Optom. 1999;82(5):193–5.

[128]. Rüfer F. Secondary Neuroprotection in Glaucoma from Change in Lifestyle. Klin Monbl Augenheilkd. 2020;237(2):158–62.

[129]. Song JE, Kim JM, Lee MY, Jang HJ, Park KH. Effects of Consumption of Alcohol on Intraocular Pressure: Korea National Health and Nutrition Examination Survey 2010 to 2011. *Nutrients*. 2020;12(8):2420. Published 2020 Aug 12.

[130]. Mansouri K, Medeiros FA, Weinreb RN. Intraocular pressure changes during sexual activity. Acta Ophthalmologica. 2013 Jun;91(4):e324-5.

[131]. Tasnim S, Tang C, Musini VM, Wright JM. Effect of alcohol on blood pressure. Cochrane Database Syst Rev. 2020;2020(7).

[132]. Takahashi S, Hara K, Sano I, Onoda K, Nagai A, Yamaguchi S, et al. Systemic factors associated with intraocular pressure among subjects in a health examination program in Japan. PLoS One. 2020;15(6).

[133]. Roddy G, Curnier D, Ellemberg D. Reductions in intraocular pressure after acute aerobic exercise: A meta-analysis. Clin J Sport Med. 2014;24(5):364–72.

[134]. Vera J, Redondo B, Bardón A, Pérez-Castilla A, García-Ramos A, Jiménez R. Effects of caffeine consumption on intraocular pressure during low-intensity endurance exercise: A placebo-controlled, double-blind, balanced crossover study. Clin Exp Ophthalmol. 2020;48(5):602–9.

[135]. Konstas AG, Kahook MY, Araie M, Katsanos A, Quaranta L, Rossetti L, et al. Diurnal and 24-h Intraocular Pressures in Glaucoma: Monitoring Strategies and Impact on Prognosis and Treatment. Adv Ther. 2018;35(11):1775–804.

[136]. Quaranta L, Biagioli E, Riva I, Tosoni C, Brusini P, Centofanti M, et al. Effect of trabeculectomy and canaloplasty on intra-ocular pressure modifications after postural changes in open-angle glaucoma. Acta Ophthalmol. 2014;92(6).

[137]. Konstas AGP, Topouzis F, Leliopoulou O, Pappas T, Georgiadis N, Jenkins JN, et al. 24-Hour Intraocular Pressure Control with Maximum Medical Therapy Compared with Surgery in Patients with Advanced Open-Angle Glaucoma. Ophthalmology. 2006;113(5):761-765.e1.

[138]. Khawaja AP, Cooke Bailey JN, Kang JH, Rand Allingham R, Hauser MA, Brilliant M, et al. Assessing the association of mitochondrial genetic variation with primary open-angle glaucoma using gene-set analyses. Investig Ophthalmol Vis Sci. 2016;57(11):5046–52.

[139]. Lenaz G. The mitochondrial production of reactive oxygen species: Mechanisms and implications in human pathology. IUBMB Life. 2001;52(3–5):159–64.

[140]. Wang Y, Xu K, Zhang H, et al. Retinal ganglion cell death is triggered by paraptosis via reactive oxygen species production: A brief literature review presenting a novel hypothesis in glaucoma pathology. Mol Med Rep. 2014;10(3):1179–83.

[141]. Kowaltowski AJ, Castilho RF, Vercesi AE. Opening of the mitochondrial permeability transition pore by uncoupling or inorganic phosphate in the presence of Ca2+ is dependent on mitochondrial-generated reactive oxygen species. FEBS Lett. 1996;378(2):150–2.

[142]. Tsai HH, Chang SC, Chou CH, Weng TP, Hsu CC, Wang JS. Exercise Training Alleviates Hypoxia-induced Mitochondrial Dysfunction in the Lymphocytes of Sedentary Males. Sci Rep. 2016;6.

[143]. de Carvalho AK, da Silva S, et al. Prior Exercise Training Prevent Hyperglycemia in STZ Mice by Increasing Hepatic Glycogen and Mitochondrial Function on Skeletal Muscle. J Cell Biochem. 2017;118(4):678–85.

[144]. Marques-Aleixo I, Santos-Alves E, Balça MM, Rizo-Roca D, Moreira PI, Oliveira PJ, et al. Physical exercise improves brain cortex and cerebellum mitochondrial bioenergetics and alters apoptotic, dynamic and auto(mito)phagy markers. Neuroscience. 2015;301:480–95.

[145]. Taghizadeh G, Pourahmad J, Mehdizadeh H, Foroumadi A, Torkaman-Boutorabi A, Hassani S, et al. Protective effects of physical exercise on MDMA-induced cognitive and mitochondrial impairment. Free Radic Biol Med. 2016;99:11–9.

[146]. Bernardo TC, Marques-Aleixo I, Beleza J, Oliveira PJ, Ascensão A, Magalhães J. Physical Exercise and Brain Mitochondrial Fitness: The Possible Role Against Alzheimer's Disease. Brain Pathol. 2016;26(5):648–63.

[147]. Barron MJ, Griffiths P, Turnbull DM, Bates D, Nichols P. The distributions of mitochondria and sodium channels reflect the specific energy requirements and conduction properties of the human optic nerve head. Br J Ophthalmol. 2004;88(2):286–90.

[148]. Simonian NA, Coyle JT. Oxidative stress in neurodegenerative diseases. Annu Rev Pharmacol Toxicol. 1996;36:83–106.

[149]. Zhang H, Davies KJA, Forman HJ. Oxidative stress response and Nrf2 signaling in aging. Free Radic Biol Med. 2015;88(Part B):314–36.

[150]. Rohowetz LJ, Kraus JG, Koulen P. Reactive oxygen species-mediated damage of retinal neurons: Drug development targets for therapies of chronic neurodegeneration of the retina. Int J Mol Sci. 2018;19(11).

[151]. Haynes WL, Johnson AT, Alward WLM. Effects of Jogging Exercise on Patients with the Pigmentary Dispersion Syndrome and Pigmentary Glaucoma. Ophthalmology. 1992;99(7):1096–103.

[152]. Shah P, Whittaker KW, Wells AP, Khaw PT. Exercise-induced visual loss associated with advanced glaucoma in young adults. Eye. 2001;15(5):616–20.

[153]. Schenker HI, Luntz MH, Kels B, Podos SM. Exercise-induced increase of intraocular pressure in the pigmentary dispersion syndrome. Am J Ophthalmol. 1980;89(4):598–600.

[154]. Gallenga PE, Mastropasqua L, Costagliola C, Ciancaglini M, Carpineto P. The use of a standardized exercise as a provocative test in pigmentary dispersion syndrome. Acta Ophthalmol Scand Suppl. 1997;75(224):26–7.

[155]. Smith DL, Kao SF, Rabbani R, Musch DC. The effects of exercise on intraocular pressure in pigmentary glaucoma patients. Ophthalmic Surg. 1989;20(8):561–7.