



REVIEW

Intraocular pressure and glaucoma: Is physical exercise beneficial or a risk?



CrossMark

Charles William McMonnies*

School of Optometry and Vision Science, University of New South Wales, Kensington 2052, Australia

Received 11 July 2015; accepted 7 December 2015

Available online 12 January 2016

KEYWORDS

Intraocular pressure;
Glaucoma;
Physical exercise;
Frailty

Abstract Intraocular pressure may become elevated with muscle exertion, changes in body position and increased respiratory volumes, especially when Valsalva manoeuvre mechanisms are involved. All of these factors may be present during physical exercise, especially if hydration levels are increased. This review examines the evidence for intraocular pressure changes during and after physical exercise. Intraocular pressure elevation may result in a reduction in ocular perfusion pressure with the associated possibility of mechanical and/or ischaemic damage to the optic nerve head. A key consideration is the possibility that, rather than being beneficial for patients who are susceptible to glaucomatous pathology, any intraocular pressure elevation could be detrimental. Lower intraocular pressure after exercise may result from its elevation causing accelerated aqueous outflow during exercise. Also examined is the possibility that people who have lower frailty are more likely to exercise as well as less likely to have or develop glaucoma. Consequently, lower prevalence of glaucoma would be expected among people who exercise. The evidence base for this topic is deficient and would be greatly improved by the availability of tonometry assessment during dynamic exercise, more studies which control for hydration levels, and methods for assessing the potential general health benefits of exercise against any possibility of exacerbated glaucomatous pathology for individual patients who are susceptible to such changes.

© 2016 Spanish General Council of Optometry. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

PALABRAS CLAVE

Presión intraocular;
Glaucoma;
Ejercicio físico;
Fragilidad

Presión intraocular y glaucoma: ¿es beneficioso el ejercicio físico, o entraña un riesgo?

Resumen La presión intraocular puede elevarse con el exceso de trabajo muscular, los cambios de la posición del cuerpo y el incremento de los volúmenes respiratorios, especialmente cuando

* Correspondence to: 77 Cliff Avenue, Northbridge, Sydney, New South Wales 2063, Australia. Tel.: +61 2 9958 3046; fax: +61 2 9958 3012; mobile: +61 0409 038 799.

E-mail address: c.mcmonnies@unsw.edu.au

<http://dx.doi.org/10.1016/j.optom.2015.12.001>

1888-4296/© 2016 Spanish General Council of Optometry. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

los mecanismos de la maniobra de Valsalva se ven implicados. Todos estos factores pueden presentarse durante el ejercicio físico, especialmente cuando se incrementan los niveles de hidratación. Esta revisión examina la evidencia de los cambios en la presión intraocular con anterioridad y posterioridad al ejercicio físico. El incremento de la presión intraocular puede derivar en una reducción de la perfusión ocular, con la posibilidad asociada de daño mecánico y/o isquémico de la cabeza del nervio óptico. Una consideración clave es la posibilidad de que, en lugar de ser beneficioso para los pacientes susceptibles de patología glaucomatosa, cualquier incremento de la presión intraocular podría resultar perjudicial. La disminución de la presión intraocular tras el ejercicio puede ser resultado de su elevación, originando la aceleración de una descarga del acuoso durante el ejercicio. También se ha estudiado la posibilidad de que las personas con menor debilidad sean propensas a realizar ejercicio, y tengan menor probabilidad de padecer o desarrollar glaucoma. Por tanto, cabría esperar una menor prevalencia del glaucoma en aquellas personas que realizan ejercicio. La base de la evidencia para esta cuestión es deficiente, y mejoraría si se dispusiera de pruebas de tonometría realizadas durante el ejercicio dinámico, un mayor número de estudios que controlen los niveles de hidratación, y métodos de evaluación de los beneficios potenciales y generales para la salud al realizar ejercicio, en contraposición a cualquier posibilidad de empeoramiento de la patología glaucomatosa en las personas susceptibles de dichos cambios.

© 2016 Spanish General Council of Optometry. Publicado por Elsevier España, S.L.U. Este es un artículo Open Access bajo la licencia CC BY-NC-ND (<http://creativecommons.org/licencias/by-nc-nd/4.0/>).

Elevated intraocular pressure (IOP) is a key risk factor for the development and progression of glaucoma.^{1,2} A number of conditions such as congenital, angle-closure and secondary glaucoma clearly show that increased IOP is sufficient to lead to glaucomatous optic neuropathy.³ Treatment to reduce IOP has been demonstrated to decrease glaucoma progression.⁴ Fluctuation in IOP, either over the 24 h diurnal period or across visits, may also contribute to glaucomatous pathology.^{5,6} There are a range of physiological IOP fluctuations as well as those due to sporadic and routine day-to-day activities which are associated with both small and large IOP elevations and fluctuations⁷⁻¹⁰ which may also be associated with glaucoma progression.⁷ Fluctuations in IOP in sitting position during office hours were concluded to not be an independent risk factor for glaucoma.¹¹ However, in-office sitting position measurements cannot capture the wide range of IOP fluctuation, often involving non-sitting postures, which occur during various activities performed outside an office and which are known to elevate IOP in any 24 h period. Significant IOP fluctuations were detected by assessment every 2 h for 24 h in patients with obstructive sleep apnea, especially in patients receiving continuous positive airway pressure therapy.¹² The high prevalence of glaucoma in obstructive sleep apnea patients may be explained by these fluctuations which were paralleled by a decrease in ocular perfusion pressure (OPP)¹² with the associated possibility of both mechanical and ischaemic damage to the optic nerve head (ONH).

Preventing large fluctuations in diurnal IOP may be as important as attaining target IOP in the prevention of glaucoma progression.¹³ The Collaborative Initial Glaucoma Treatment Study¹⁴ and the Advanced Glaucoma Intervention Study¹⁵ both found that fluctuation in IOP is predictive of greater visual field loss. The goal of detecting and reducing abnormal 24-h IOP fluctuation is warranted in all newly

diagnosed glaucomatous patients as well as in patients who continue to progress despite treatment which lowers pressure.¹⁶ Avoiding or reducing exposure to elevated IOP (fluctuation episodes) may improve the prognosis for some glaucoma suspects and patients with glaucoma.⁷⁻¹⁰ The key determinants of any pathological significance associated with IOP elevation episodes may not only be the degree of elevation, its duration and frequency as well as the time over which it occurs, but also individual susceptibility to them.^{7,10} For example, the pathology associated with normal tension glaucoma (NTG) suggests that such eyes have a lower IOP threshold for neuropathic changes. Increasing glaucoma prevalence with age¹⁷ suggests that susceptibility to IOP fluctuation and elevation episodes could increase with age.

Histories of the frequency and intensity of participation in activities which are known to elevate IOP allow an estimate of individual exposure to IOP fluctuation.¹⁰ Most episodes of IOP elevation appear likely to remain undetected due to difficulties in monitoring IOP during many activities. Studies of IOP fluctuation with physical exercise have typically measured IOP with an insufficient sampling rate to truly measure IOP variability.¹ For example, head movement during dynamic exercise necessitates that tonometry be performed during a break in an exercise sequence or after the sequence has been completed.¹⁸ Static isometric phases which occur during weightlifting and which enable tonometry to be performed are exceptions.^{19,20} Ideally, 24 h continuous monitoring of IOP will capture a complete record of the degree, duration and frequency of episodic elevations and so the full extent of IOP fluctuation.^{8,9} This review examines the mechanisms for IOP elevation and fluctuation during and subsequent to physical exercise. A key consideration is the possibility that, rather than being beneficial,²¹ and that it may be reasonable to encourage dynamic exercise

in glaucoma patients,^{22–27} physical exercise-related changes could be detrimental for patients who have glaucoma or are glaucoma suspects. A PubMed Search for intraocular pressure and sport and intraocular pressure and physical exercise yielded 231 and 277 results, respectively. Papers providing evidence of IOP changes during and after exercise were used to examine the possibility that any IOP elevation episodes occurring during physical exercise could accelerate aqueous outflow and result in reduced IOP being measured after the completion of the exercise.

Valsalva manoeuvre mechanisms for IOP elevation

Important mechanisms for many IOP elevation-related activities include Valsalva manoeuvres (VMs), which occur with a closed glottis, and partial VMs associated with increased expiratory effort.²⁸ IOP recorded by tonography was found to increase with and be sustained for the duration of increased expiratory effort.²⁹ For example, deep respirations produce an IOP rise and fall of as much as 5 mmHg.³⁰ Respiration-related influences on IOP can be much greater according to the degree to which VM phenomena are involved. In association with a VM, IOP was found to elevate to over 40 mmHg for some subjects, especially for those who were myopic.³¹ Greater intrathoracic pressure was required to produce a given respiratory flow at lesser degrees of lung inflation, especially in emphysematous subjects.³² Reduced lung capacity with age may necessitate deeper respirations during exercise as well as restrict the range of exercise intensity performed.

Abdominal muscle effort during many VM-related activities also increases intrathoracic pressure.²⁸ For example, due to the increased expiratory and abdominal muscle effort associated with balloon inflation, a significant VM stress is produced in cycles of rapid rises and falls in both intra-abdominal and intrathoracic pressure.³³ Similar cycles occur according to the intensity of physical exercises and associated increased respiration volumes. Intra-abdominal pressure has been shown to increase consistently during both static and dynamic lifting tasks.³⁴ Breath control is a significant factor in the generation of intra-abdominal pressure magnitude during lifting tasks.³⁴ IOP increases significantly during a bench press exercise and to a greater extent with breath holding.³⁵

Expiratory effort and intracranial pressure elevation

Apart from IOP elevation, increased expiratory effort and increased intra-abdominal pressure also cause an increase in intracranial pressure.³⁶ Weightlifting activities have been reported in association with a wide range of adverse responses including stroke, cerebral haemorrhage, subarachnoid haemorrhage, conjunctival and retinal haemorrhage as well as retinal detachment.²⁰ It was suggested that elevation of intracranial pressure and IOP may contribute to these responses.²⁰ The risk of glaucomatous pathology could be increased when exercise-related increases in both intracranial and intraocular pressure result

in compressive lamina cribrosa damage.³⁷ Glaucomatous eyes can undergo pulsatile stretching due to greater ocular pulse amplitude and larger diurnal variation in IOP.³⁸ Increased IOP during weightlifting activities^{20,35} suggests that any associated fundus stretching could contribute to reported adverse responses such as retinal detachment or haemorrhage.³⁹ Risk for ocular pathology may be a contraindication for weightlifting especially for patients with myopic fundus pathology,³⁹ and for keratoconus patients with increased susceptibility to cone formation or progression in a thin area of cornea, when IOP is elevated.⁴⁰

Muscular effort mechanisms for IOP elevation

IOP rises during sustained muscular contraction²⁰ and on relaxation it falls.³⁵ IOP was measured towards the end of a short series of weightlifting-related efforts at 80% of individual maximum capacity.³⁵ It was found that IOP was more elevated when a VM was simulated (mean 4.3 mmHg, 23.1% higher than pre-exercise baseline) than when a VM was avoided (mean 2.2 mmHg, 11.8% higher than baseline).³⁵ Subjects were examined while making a weightlifting effort at the level of their maximum capacity with no attempt to control for the use of a VM.²⁰ Mean IOP elevations of 115.4% above baseline were measured.²⁰ One subject's IOP reached 46 mmHg during maximal isometric muscle contraction effort.²⁰ Upper body muscle exertion is not essential for this effect on IOP. Isometric exercise involving a sustained squat posture, with thigh and lower leg maintained at right angles, caused a significant (37%) mean increase in IOP.⁴¹

Changes in body position mechanisms for IOP elevation

A mean IOP elevation of 4.4 mmHg (approximately 25% of baseline) was measured in changing from a sitting to a supine position⁴² which may be significant during exercises such as sit-ups and weightlifting bench presses. Older age is usually associated with reduced participation in exercise⁴³ although, as discussed below, there are significant numbers of older people who do exercise. However, age appears likely to limit opportunities for some body positions and higher levels of exertion to be significant factors during physical exercise. However, there can be exceptions. A 68 year old athletic physician developed glaucoma over a period during which he performed inverted head stand exercises for up to 5 min each day.¹⁹ With medication his IOPs were stabilized at 10 mmHg but increases to over 40 mmHg when he assumed his inverted head stand position.¹⁹ In addition, his habit of assuming a strenuous flexed isometric muscle posture while sitting, and which involved a considerable amount of facial congestion, resulted in elevations above baseline IOP ranging from 10 mmHg to 25 mmHg.¹⁹ Apart from muscular effort, increased facial muscle tension has been shown to be associated with IOP elevation.⁴⁴ His glaucomatous field losses stabilized after he took advice to desist from these exercises.¹⁹

Non-competitive lap swimming can be a favoured form of exercise for older patients. IOP could be elevated when swimming according to the influence of deep respiration and the degree of muscle effort expended. A mean sitting

position IOP elevation while wearing different styles of swimming goggles was 4.5 mmHg.⁴⁵ For two of the five styles of goggle evaluated, mean elevations were 10.1 mmHg and 13.4 mmHg but again these were sitting position findings.⁴⁵ Apart from the type of goggles worn when swimming, IOP elevation may be significantly higher than found for a sitting position according to the extent and influence of increased respiration volumes, a horizontal body position and the degree of muscular effort expended. In addition, a prone body position has been shown to elevate IOP to a higher level than a supine position.⁴⁶ With the exception of backstroke, all other swimming strokes involve prone positions in addition to muscular effort, increased respiration rate and possibly the use of goggles. It appears possible that IOP is elevated by any or all of these several mechanisms during swimming as it has been found to be elevated during static weightlifting.

Other influences on IOP

Any exercise-related IOP elevation may also be influenced by other factors such as pre-exercise medications,⁴⁷ caffeine intake in coffee, tea, cola, energy drink or milk chocolate,⁴⁸ and cigarette smoking,⁴⁹ all of which are known to have the potential to elevate IOP. Elevations due to water ingestion are innocuous in normal patients but in glaucomatous patients, especially those with abnormally low aqueous outflow capacity, they may be pathological.⁵⁰ For example, a water drinking test administered to NTG patients found a significantly higher peak IOP compared to normal controls.⁵¹ IOP was found to be progressively reduced during exercise causing dehydration, but remained relatively stable when hydration was maintained.⁵² Hydration levels may also influence IOP depending on exercise intensity and duration, ambient temperature and humidity as well as opportunities to ingest water before, during and after exercise. Open mouth heavy breathing dries the mouth, especially under low humidity conditions. Having ready access to water uptake before, during and after exercise can lead to more frequent water ingestion and over-hydration in association with exercise.

Lower IOP found immediately on completion of dynamic forms of physical exercise

IOP was lower than baseline for healthy subjects after performing a set of chest press exercises but then reduced further after completing second and third sets of the same exercise.²² Exercise bike for 9 min was found to result in lowering of IOP.⁵³ The biomicroscope and Goldmann tonometer were set up in front of the exercise bike with measurements recorded with the subject sitting on the bike (not pedalling) with their head position stabilized by the instrument headrest (Saarela V, written communication, 25th May 2015).

Except for an immediate rise in IOP due to the contraction of abdominal and thoracic muscles and/or the performance of a VM, a fall in IOP after hard and/or prolonged forms of exercise has consistently been reported.⁵⁴ Walking, for example, was found to be associated with a significant fall in IOP.⁵⁴ After running a 42 km marathon, IOP was found to

have dropped by a mean of 2.25 mmHg with greater reduction occurring for subjects with higher baseline readings.⁵⁵ After completion of a 110 km march carrying a 20 kg backpack, the mean reduction in IOP was 4.1 mmHg (26.5%).⁵⁶ Reductions in IOP were found to increase with the duration of walking, jogging and running.⁵⁷ IOP measured after both isometric and isokinetic exercise was found to be lower than pre-exercise IOP.⁵⁸ The degree of lowering was directly proportional to the intensity of the exercise and significantly greater for isokinetic exercise.⁵⁸ Recovery of IOP to pre-exercise levels after exercise has been reported to take 10 min,⁵⁹ 15 min⁵⁵ and up to an hour.²¹

Possible reasons for IOP reduction found after exercise

Although the finding that lowered IOP is measured after exercise is widely reported in the literature, the mechanism for it is poorly understood.²⁶ Three theories of its aetiology involve decreased blood pH, elevated blood plasma osmolarity, and elevated blood lactate.²⁶ Another mechanism which has been suggested involves autonomic changes which might reduce IOP.⁶⁰ To the extent that respiratory, muscular effort and non-erect body positions for example, could contribute to IOP elevation during exercise, the finding of reduced IOP elevation after exercise might be explained by a number of other factors.

Exercise greatly increases the metabolic demands of muscle tissue and several respiratory and cardiovascular adjustments are made to meet this requirement.⁶¹ For example, an increase in heart rate, stroke volume and cardiac output, increased blood flow to muscles and skin, reduced blood flow to other organs, increased blood cell concentration and blood-oxygen exchange as well as a decrease in blood plasma volume are all changes occurring during exercise.⁶¹ Exercise resulted in significant increases in mean arterial blood pressure (56%) and pulse rate (84%)⁴¹ and IOP is transiently elevated according to the level of blood pressure elevation.²⁹ Increased blood pressure and an associated increase in passive production of aqueous humour would elevate IOP.⁶²

Other explanations for lower IOP after exercise

Dehydration may contribute to reduced aqueous production and lower IOP.⁵³ Significant dehydration appears to be unlikely during lap swimming and also, depending on ambient temperature, during light to moderate exercise which may usually be performed by older people. However, the mechanisms which appear to explain IOP elevation during static weightlifting^{19,20,35} which can include, deeper respirations, VMs, non-upright body positions, and muscular effort, may also elevate IOP during dynamic forms of exercise.^{28,29,33} These possibilities give rise to the following hypothesis to help explain IOP reduction recorded after dynamic exercise²¹ as well as after both static and dynamic weightlifting.³⁵ Honan Balloon,⁶³ ocular massage⁶⁴ and tonographic assessments⁶⁵ illustrate how elevated IOP can reduce IOP over time by accelerating aqueous outflow from the eye. According to this hypothesis the facility

of aqueous outflow and the level of any IOP elevation during exercise would help determine post-exercise IOP. This mechanism could explain reduced IOP being measured immediately after exercise.

Ocular perfusion pressure, exercise and glaucoma risk

The vascular theory of glaucoma considers related optic neuropathy to be due to OPP reduction in response to factors such as increased IOP³ and/or a decrease in blood pressure.⁶² Deficient autoregulation of the ONH results in unstable ocular perfusion, ischaemia and reperfusion oxidative stress damage.³ A case series of elevated IOP in association with anterior ischaemic optic neuropathy led to the finding that elevated IOP may be a risk factor for ischaemic ONH pathology.⁶⁶ A possible consequence of disturbed ocular blood flow is ONH damage associated with its increased sensitivity to IOP.^{3,67,68} For example, in low tension glaucoma volunteers, low ocular perfusion amplitude changes varied with the degree to which subjects were vasospastic.⁶⁹

Ordinarily, blood pressure increases could compensate for IOP elevation during exercise activity so that OPP variation might be limited. Three cases of young glaucoma patients experiencing visual loss during exercise have been reported.⁷⁰ It was hypothesized that these changes could be due to reduced ocular perfusion pressure because of blood being diverted to other organs during exercise.⁷⁰ Any IOP increase during exercise in combination with failure of autoregulation of blood flow to the ONH may also contribute to these changes. Increased susceptibility to reduced OPP may also be a consequence of low blood pressure. For example, several studies have reported associations between low diastolic perfusion pressure with higher prevalence or incidence of glaucoma⁷¹ so that antihypertensive treatment could increase the risk of glaucoma.⁶² Decreases in blood pressure and ocular perfusion pressure have been associated with glaucoma.⁷² The vast majority of published studies dealing with blood flow report reduced ocular perfusion in glaucoma patients compared with normal subjects.³

Glaucoma, frailty, exercise

The concept of frailty is a non-specific state of vulnerability which is associated with a reduced functional reserve and a consequent decrease in adaptation (resilience) to any sort of stressors.⁷³ Frailty results in a higher risk for accelerated physical and cognitive decline, disability and death.⁷³ The causes of frailty are complex and multidimensional based on the interplay of genetic, biological, physical, psychological, social and environmental factors.⁷³ Assessment of frailty depends on health deficit accumulation⁷⁴ and the frailty index employed in the National Population Health Survey of Canada was determined according to a score based on 36 deficits.⁷⁵ Health deficits included medical conditions, functional impairments, symptoms and poor health attitudes.⁷⁵ For example, having deficits such as high blood pressure, diabetes, cataracts, glaucoma, no regular exercise habits as well as having limited habits of other forms of physical

exercise, were part of the information collected to identify and quantify an individual frailty index.⁷⁵

As is the case for frailty, the level of physical exercise in which individuals are engaged at any point in their lifespan reflects a complex interaction of biological, psychological, and sociological factors.⁷⁶ A longitudinal study found that those who were relatively fitter at baseline tended to remain healthy and use fewer health care services.⁷⁴ Conversely, those with a higher frailty index value were more likely to require greater use of health care services and to have less involvement in physical exercise.⁷⁴ These findings suggest that the accumulation of frailty-related deficits is a fact of ageing, not age, and that the antecedents of frailty in late life manifest at least by middle age.⁷⁴

Prevalence of glaucoma in habitual runners

Low frailty is consistent with greater resilience and absence of frailty indicators such as glaucoma and cataract as well as having a greater involvement in physical exercise.⁷⁴ For example, in a sample of 49,005 men and women both moderate (walking) exercise (mean age 61 ± 11 years) and vigorous (running) exercise (mean age 48 ± 11 years) groups were similarly associated with reduced risk of cataract.⁷⁷ This association may be a function of exercise habits delaying cataract formation but may also be a function of people who exercise being more likely to have low frailty which, in turn, is associated with reduced cataract prevalence.⁷⁵ People with high frailty who are more likely to have or to develop glaucoma and cataract may not feel well enough to exercise.⁷⁸

However, a study was performed involving 29,854 male runners and participants in foot-races (mean age 43.3 ± 10.7 years (range 18–73) without diabetes who were followed for 7.7 years.²⁷ Two hundred incident glaucoma cases (0.67%) (mean age $\pm$ SE: 53.6 ± 0.73) were reported by questionnaire response during follow-up.²⁷ The two hundred men reporting physician-diagnosed glaucoma did not include 115 who had been diagnosed with glaucoma before or in the year of the their survey baseline.²⁷ At end of the survey 315 subjects ($200 + 115 = 1.05\%$) had reported being diagnosed with glaucoma.²⁷ Newly diagnosed diabetes mellitus and high level of blood glucose are associated with elevated IOP and high-tension glaucoma, and⁷⁹ consequently, the number of glaucoma cases at the end of the survey period might have been greater than 1.05% if any diabetics who also developed glaucoma during the survey period had been included.²⁷ Given that 57.1% of glaucoma cases were found to be undiagnosed among 5000 Greek people over 59 years of age,⁸⁰ there may also have been a significant number of undiagnosed cases of glaucoma at the end of the survey so that the prevalence might have been even greater. When glaucoma and participation in exercise are two of the factors used as an indication of frailty,⁷⁵ a sample of runners and participants in foot-races, with presumed low frailty, appear likely to have low prevalence of glaucoma. However, rather than being lower compared to people who did not exercise, the prevalence of glaucoma in this vigorous exercise sample could be no different to or even greater than a non-exercise group with mean age $\pm$ SE of 53.6 ± 0.73 years. Such an interpretation of the study findings would suggest

that physical exercise may not be beneficial in regard to glaucoma pathology and could even be detrimental.

Old age and participation in exercise

A survey in the United States found rapid declines in levels of physical exercise activity during adolescence which often continued into young adulthood.⁸¹ The period from middle adulthood up to retirement at 65 years often revealed relatively stable activity patterns.⁸¹ Post-retirement, the prevalence of participation in regular vigorous activities such as running, jogging and swimming increased.⁸¹ The results of three National surveys indicated that of those over 75 years of age, 26.5% and 15.5% performed at least moderate and vigorous levels of exercise, respectively.⁸² These participation rates⁸² may be a function of people in retirement having more time available to exercise as well as greater awareness of the associated health benefits. Other motivations may be relevant. For example, a study of Australian Masters Games athletes aged 60–89 years found that motivation to train for and participate in competition was related to the value placed on winning and achievement.⁸³

An alternative to the conclusion that exercise reduces the risk of having or developing glaucoma²¹ and that it may be reasonable to encourage dynamic exercise in glaucoma patients^{22–27} could be that less-frail people are more likely to exercise regularly as well as being less likely to have or develop glaucoma because they are less frail. However, any IOP elevation during exercise may negate that expectation and lead to an increase in glaucoma cases among people who exercise regularly?

Discussion

Reviews of the literature found that, with the exception of pigment dispersion syndrome, Uthoff's symptom, and some cases of advanced glaucoma, regular dynamic exercise is not harmful to eyes²¹ and that it may be reasonable to encourage dynamic exercise in glaucoma patients.^{22–27} The findings of this review raises question about these recommendations. The study reported by Williams²⁷ is open to the interpretation that glaucoma prevalence is not less than would be expected in higher performance athletes but could even be higher than normal for a group who are more likely to have low frailty. Such an interpretation suggests that IOP could be elevated to levels which are pathological in less frail but glaucoma-susceptible individuals. The possibility of that group being less frail is suggested by their subscription to a running magazine, their participation in footrace events and that 80% of them were capable of running in 10 kilometres events. Measurements of IOP elevation during static weightlifting led to the conclusion that weight lifting could be a risk factor for the development or progression of glaucoma.³⁵ The possibility of dynamic exercise also being associated with elevated IOP and increased glaucoma risk is suggested by the evidence that increased respiratory volumes, VMs and muscular effort as well as non-erect body positions can all elevate IOP.

The hypothesis that exercise may be detrimental to people with glaucoma would be supported by IOP elevation during dynamic exercise being confirmed. Increased aqueous

production due to hydration before and re-hydration during exercise sessions as well as elevated blood pressure may all exacerbate mechanisms for IOP elevation. Elevated IOP increases aqueous outflow,⁸⁴ and any IOP elevation during dynamic exercise would help explain lower than baseline readings being recorded when the exercise has ceased. The cyclic nature of increased respiration volumes during exercise may contribute to rises and falls in IOP and a "pumping" influence which increases aqueous outflow. Increased aqueous outflow could be supplementary to any other mechanisms for lowering IOP. For example, depending on ambient temperature and humidity as well as exercise intensity and duration, IOP could be lowered by dehydration during exercise. Nevertheless, any IOP elevation during exercise, which would have no adverse consequences for most people, could contribute to new or additional glaucomatous pathological change in susceptible individuals. Patients with ocular hypertension can have larger IOP elevations for changes in body position.⁸⁵ A water drinking test administered to NTG patients found a significantly higher peak IOP compared to normal controls.⁵¹ Glaucoma, patients who often have elevated IOP and/or reduced outflow facility,^{31,35,86} may be at risk for higher levels of IOP elevation than the levels found for healthy subjects. Increased glaucoma susceptibility in older people may increase the significance of IOP elevations during less vigorous exercise.¹⁷

Elevated IOP remains an important risk factor in glaucoma and IOP reduction is still the only treatment of proven benefit⁸⁷ having been demonstrated to decrease glaucoma incidence.¹² Moderation or avoidance of IOP elevation episodes may be a useful adjunct to other forms of glaucoma management.^{7–10,88} Inverted body positions and strenuous isometric muscle effort,¹⁹ playing high wind resistant instruments,⁸⁹ and eye rubbing⁹⁰ have all been associated with glaucoma. All of those activities are known to elevate IOP.¹⁰ Healthy participation in physical exercise is known to be beneficial to health with associated reduced risk for hypertension, cardiovascular disease and diabetes for example.^{24,60} It is clear that healthy physical exercise should be encouraged.⁹¹ However, with the exception of weightlifting, it is not clear as to whether dynamic forms of exercise activity can be potentially harmful for glaucoma suspects or people with glaucoma.

It is possible that, if IOP is elevated during dynamic exercise, then periods of lower than baseline post-exercise IOP might allow recovery from any associated glaucomatous damage which could occur during the exercise. The evidence-base for this topic is deficient and would be greatly improved by tonometry assessment during dynamic exercise, more studies which control for hydration levels, and means for assessing the potential general health benefits of exercise against any possibility of exacerbated glaucomatous pathology for individual glaucoma patients and glaucoma suspects. The identification of risk factors associated with undiagnosed glaucoma may be important to better achieve higher case ascertainment yields in the community.⁸⁰ This review suggests that IOP elevation during participation in dynamic physical exercise could contribute to the onset or progression of glaucoma in susceptible individuals and questions the recommendations that participation in physical exercise is either harmless or is actually beneficial,²¹ and that it may be reasonable to encourage

dynamic exercise in glaucoma patients^{22–27} The beneficial effect of any intervention which successfully reduces IOP in a glaucoma patient will be reduced by any IOP elevation associated with muscle exertion, changes in body position and increased respiratory volumes, especially when Valsalva manoeuvre mechanisms are involved. All of these factors may be present during physical exercise associated with vocational and recreational activities including sport, especially if hydration levels are increased.

Conflicts of interest

There is no research commercial, funding, proprietary or conflicting interests to acknowledge in relation to this review.

References

- Gardiner SK, Fortune B, Wang L, Downs JC, Burgoyne CF. Intraocular pressure magnitude and variability as predictors or rates of structural change in non-human primate experimental glaucoma. *Exp Eye Res.* 2012;103:1–8.
- Awadalla MS, Fingert JH, Roos BE, et al. Copy number variations of TBK1 in Australian patients with primary open-angle glaucoma. *Am J Ophthalmol.* 2015;159:124–130.
- Flammer J, Orgul S, Costa VP, et al. The impact of ocular blood flow in glaucoma. *Prog Ret Eye Res.* 2002;21:359–393.
- Detry-Morel M. Currents on target intraocular pressure and intraocular pressure fluctuations in glaucoma management. *Bull Soc Belge Ophthalmol.* 2008;308:35–43.
- Varma R, Hwang L-J, Grunden JW, Bean GW. Inter-visit IOP range: an alternative parameter for assessing intraocular pressure control in clinical trials. *Am J Ophthalmol.* 2008;145:336–342.
- Asrani S, Zeimer R, Wilensky J, Gieser D, Vitale S, Lindenmuth K. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. *J Glaucoma.* 2000;9:134–142.
- Krist D, Cursiefen C, Junemann A. Transitory intrathoracic and abdominal pressure elevation in the history of 64 patients with normal pressure glaucoma. *Klin Monbl Augenheilkd.* 2001;218:209–213.
- Downs JC, Roberts MD, Burgoyne CF. The mechanical environment of the optic nerve head in glaucoma. *Optom Vis Sci.* 2008;85:425–435.
- 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;95:627–629.
- McMonnies CW. An examination of the hypothesis that intraocular pressure elevation episodes can have prognostic significance in glaucoma suspects. *J Optom.* 2015;8:223–231, <http://dx.doi.org/10.1016/j.optom.2014.07.008>.
- Bengtsson B, Heijl A. Diurnal IOP fluctuations: not an independent risk factor for glaucomatous visual field loss in high-risk ocular hypertension. *Graefes Arch Clin Exp Ophthalmol.* 2005;243:513–518.
- Kiekens S, De Groot V, Coeckelbergh T, et al. Continuous positive airway pressure therapy is associated with an increase in intraocular pressure in obstructive sleep apnea. *Invest Ophthalmol Vis Sci.* 2008;49:934–940.
- The AGIS investigators. The Advanced Glaucoma Intervention Study: 7 The relationship between control of intraocular pressure and visual field deterioration. *Am J Ophthalmol.* 2000;130:429–440.
- Musch DC, Gillespie BW, Lichter PR, Niziol LM, Janz NK, CIGTS Study Investigators. Visual field progression in the Collaborative Initial Treatment Study. *Ophthalmology.* 2009;116:200–207.
- Nouri-Mahdavi K, Hoffman D, Coleman AL, Liu G, et al. Predictive factors for glaucomatous visual field progression in the Advanced Glaucoma Intervention Study. *Ophthalmology.* 2004;111:1627–1635.
- Kass MA, Heurer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller P, for the Ocular Hypertension Treatment Study Group. The Ocular hypertension treatment study. A randomized trial determines that topical ocular hypotensive medication, delays or prevents the onset of primary open-angle glaucoma. *Arch Ophthalmol.* 2002;120:701–703.
- Caprioli J, Coleman AL. Intraocular pressure fluctuation a risk factor for visual field progression at low intraocular pressures in the advanced glaucoma intervention study. *Ophthalmology.* 2008;115:1123–1129.
- Krejci RC, Gordon RB, Moran CT, Sargent RG, Magun JC. Changes in intraocular pressure during acute exercise. *Am J Optom Physiol Opt.* 1981;58:144–148.
- O'Connor PS, Poirier RH. Ocular effects of gravity inversion. *JAMA.* 1985;254:756.
- 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;21:243–246.
- Gale J, Wells AP, Wilson G. Effects of exercise on ocular physiology and disease. *Surv Ophthalmol.* 2009;54:349–355.
- Chromiak JA, Abadie BR, Braswell RA, Koh YS, Chiles DR. Resistance training exercises acutely reduce intraocular pressure in physically active men and women. *J Strength Cond Res.* 2003;17:715–720.
- Era P, Parssinen O, Kallinen M, Suominen H. Effect of bicycle ergometer test on intraocular pressure in elderly athletes and controls. *Acta Ophthalmol.* 1993;71:301–307.
- Passo MS, Elliot DL, Goldberg L. Long-term effects of exercise conditioning on intraocular pressure in glaucoma suspects. *J Glaucoma.* 1992;1:39–41.
- Natsis K, Asouhidou I, Noutsos G, Chatzibalas T, Vlasik K, Karabatakis V. Aerobic exercise and intraocular pressure in normotensive and glaucoma patients. *BMC Ophthalmol.* 2009;9:1–7.
- Risner D, Ehrlich R, Kheradiya NS, Siesky B, McCranor L, Harris A. Effects of exercise on intraocular pressure and blood flow: a review. *J Glaucoma.* 2009;18:429–436.
- Williams PT. Relationship of incident glaucoma versus physical activity and fitness in male runners. *Med Sci Sports Exerc.* 2009;41:1566–1572.
- Porth CJ, Bamrah VS, Tristani FE, Smith JJ. The Valsalva maneuver: mechanisms and clinical implications. *Heart Lung.* 1984;13:507–518.
- Rosen DA, Johnston VC. Ocular pressure patterns in the Valsalva maneuver. *Arch Ophthalmol.* 1959;62:810–816.
- Adler FH. *Physiology of the eye.* St. Louis: The C.V. Mosby Company; 1959:137.
- 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 bulb in myopia. *Graefes Arch Clin Exp Ophthalmol.* 1982;218:51–54.
- Fry DL, Ebert RV, Stead WW, Brown CC. The mechanics of pulmonary ventilation in normal subjects and in patients with emphysema. *Am J Med.* 1954;16:80–97.
- Georgiou T, Pearce IA, Taylor RH. Valsalva retinopathy associated with blowing balloons. *Eye.* 1999;13:686–687.
- Hagins M, Pietrek M, Sheikhzadeh A, Nordin M, Axen K. The effects of breath control on intra-abdominal pressure during lifting tasks. *Spine.* 2004;29:464–469.

35. Vieira GM, Oliveira HB, Tavares de Andrade D, Bottaro M, Ritch R. Intraocular pressure variations during weight lifting. *Arch Ophthalmol*. 2006;124:1251–1254.
36. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugarman HJ. A proposed relationship between increased intra-abdominal, intrathoracic, and intracranial pressure. *Crit Care Med*. 1997;25:496–503.
37. McMonnies CW. The interaction between intracranial pressure, intraocular pressure and lamina cribrosa compression in glaucoma. *Clin Exp Optom*. 2015 [in press].
38. Sigal IA, Ethier CR. Biomechanics of the optic nerve head. *Exp Eye Res*. 2009;88:799–807.
39. McMonnies CW. An examination of the relation between intraocular pressure, fundus stretching and myopic pathology. *Clin Exp Optom*. 2015 [in press].
40. McMonnies CW. The possible significance of the baropathic nature of keratectasia. *Clin Exp Optom*. 2013;96:197–200.
41. Kiss B, Dallinger S, Polak K, Findl O, Eichler H-G, Schmetterer L. Ocular hemodynamics during isometric exercise. *Microvasc Res*. 2001;61:1–7.
42. 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;75:652–655.
43. Malina RM. Tracking of physical activity and physical fitness across the lifespan. *Res Quart Exerc Sport*. 1996;67(S3):S48–S57.
44. Suyapa E, Silvia M, Raczynski JM, Kleinstein RN. Self-regulated facial muscle tension effects on intraocular pressure. *Psychophysiol*. 1984;21:79–82.
45. Morgan WH, Cunneen TS, Balaratnasingam C, Dao-Yi Y. Wearing swimming goggles can elevate intraocular pressure. *Br J Ophthalmol*. 2008;92:1218–1221.
46. Lam AKC, Douthwaite MA. Does the change of anterior chamber depth or/and episcleral venous pressure cause intraocular pressure change in postural variation. *Optom Vis Sci*. 1997;74:664–667.
47. Rath EZ. Drug-induced glaucoma (glaucoma secondary to systemic medications). In: Rumelt S, ed. *Glaucoma – basic and clinical concepts*. Rijeka, Croatia: InTech; 2011:547–554 [chapter 28].
48. Avisar R, Weinberger D. Effect of coffee consumption on intraocular pressure. *Ann Pharmacother*. 2002;36:992–995.
49. Mehra KS, Roy PN, Khare BB. Tobacco smoking and glaucoma. *Ann Ophthalmol*. 1976;8:462–464.
50. Leyhdecker W. Evaluation of the water-drinking test. *Br J Ophthalmol*. 1954;38:290–294.
51. Susanna R, Vessani RM, Sakata L, Zacarias LC, Hatanaka M. The relation between intraocular pressure peak in the water drinking test and visual field progression in glaucoma. *Br J Ophthalmol*. 2005;89:1298–1301.
52. Hunt AP, Feigl B, Stewart IB. The intraocular pressure response to dehydration: a pilot study. *Eur J Appl Physiol*. 2012;112:1963–1966.
53. Saarela V, Ahvenvaara E, Tuulonen A. Variability of Heidelberg retina tomograph parameters during exercise. *Acta Ophthalmologica*. 2013;91:32–36.
54. Leighton DA, Phillips CI. Effect of moderate exercise on the ocular tension. *Brit J Ophthalmol*. 1970;54:599–602.
55. Myers KJ. The effect of aerobic exercise on intraocular pressure. *Invest Ophthalmol Vis Sci*. 1974;13:74–77.
56. Ashkenazi I, Melamed S, Blumenthal M. The effect of continuous strenuous exercise on intraocular pressure. *Invest Ophthalmol Vis Sci*. 1992;33:2874–2877.
57. Qureshi IA. Effects of mild, moderate and severe exercise on intraocular pressure of sedentary subjects. *Ann Hum Biol*. 1995;22:545–553.
58. Avunduk AM, Yilmaz B, Sahin M, Kapicioglu Z, Dayanir V. The comparison of intraocular pressure reductions after isometric and isokinetic exercises in normal individuals. *Ophthalmologica*. 1999;213:290–294.
59. Rufer F, Schiller J, Klettner A, Lanzi I, Roeder J, Weisse B. Comparison of the influence of aerobic and resistance exercise of the upper and lower limb on intraocular pressure. *Acta Ophthalmol*. 2014;92:249–252.
60. Passo MS, Goldberg L, Elliot DL, Van Buskirk M. Exercise training reduces intraocular pressure among subjects suspected of having glaucoma. *Arch Ophthalmol*. 1991;109:1096–1098.
61. Astrand P-O, Rodahl K. *Textbook of work physiology*. New York: McGraw-Hill Book Company; 1970:3–7.
62. Leske MC. The epidemiology of open-angle glaucoma: a review. *Am J Epidemiol*. 1983;118:166–186.
63. McDonnell PJ, Quigley HA, Maumenee AE, Stark WJ, Hutchins GM. The Honan intraocular pressure reducer. *Arch Ophthalmol*. 1985;103:422–425.
64. Ernest JT, Goldstick TK, Stein MA, Zheutlin JD. Ocular massage before cataract surgery. *Trans Am Ophthalmol Soc*. 1985;83:205–217.
65. Hetland-Erikson J, Odberg T. Experimental tonography on enucleated human eyes. II. The loss of intraocular fluid caused by tonography. *Invest Ophthalmol Vis Sci*. 1975;14:944–947.
66. Tomsak RL, Remier BF. Anterior ischemic optic neuropathy and increased intraocular pressure. *J Clin Neuro-Ophthalmol*. 1989;9:116–120.
67. Grieshaber M, Flammer J. Blood flow in glaucoma. *Curr Opin Ophthalmol*. 2005;16:79–83.
68. Kuehn MH, Fingert JH, Kwon YH. Retinal ganglion cell death in glaucoma: mechanisms and neuroprotective strategies. *Ophthalmol Clin N Am*. 2005;18:383–395.
69. Schmidt K-G, Mittag TW, Pavlovic S, Hessemer V. Influence of physical exercise and nifedepine on ocular pulse amplitude. *Graefes Arch Clin Exp Ophthalmol*. 1996;234:527–532.
70. Shah P, Whittaker KW, Wells AP, Khaw PT. Exercise-induced visual loss associated with advanced glaucoma in young adults. *Eye*. 2001;15:616–620.
71. Topouzis F, Coleman AL, Harris A, et al. Association of blood pressure status with the optic disk structure in non-glaucoma subjects: the Thessaloniki Eye Study. *Am J Ophthalmol*. 2006;142:60–67.
72. Caprioli J, Coleman AL. Blood Flow in Glaucoma Discussion. Blood pressure, perfusion pressure, and glaucoma. *Am J Ophthalmol*. 2010;149:704–712.
73. Fulop T, Larbi A, Witkowski JM, et al. Aging, frailty and age-related diseases. *Biogerontology*. 2010;11:547–563.
74. Rockwood K, Song X, Mitnitski A. Changes in relative fitness and frailty across the adult lifespan: evidence from the Canadian National Population Health Survey. *Can Med Ass J*. 2011;183:E487–E494.
75. Song X, Mitnitski A, Rockwood K. Prevalence of 10 year outcomes of frailty in older adults in relation to deficit accumulation. *J Am Geriatr Soc*. 2010;58:681–687.
76. Sallis JF, Hovell MF. Determinants of exercise behaviour. *Exerc Sport Sci Rev*. 1990;18:307–330.
77. Williams PT. Walking and running are associated with similar reductions in cataract risk. *Med Sci Sports Exerc*. 2013. <http://dx.doi.org/10.1249/MSS.0b013e31828121d0>.
78. King AC, Castro C, Wilcox S, Eyler AA, Sallis JF, Brownson RC. Personal and environmental factors associated with physical inactivity among different racial-ethnic groups of U.S. middle-aged and older women. *Health Psychol*. 2000;19:354–364.
79. Dielemans I, de Jong PTVM, Stolk R, Vingerling JR, Grobbee DE, Hofman A. Primary open-angle glaucoma, intraocular pressure, and diabetes mellitus in the general elderly population. *Ophthalmology*. 1996;103:1271–1275.

80. Topouzis F, Coleman AL, Harris A, et al. Factors associated with undiagnosed open-angle glaucoma: the Thessaloniki Eye Study. *Am J Ophthalmol*. 2008;145:327–335.
81. 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;32:1601–1609.
82. 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:S526–S535.
83. Dionigi R. A leisure pursuit that 'goes against the grain': older people and competitive sport. *Ann Leisure Res*. 2005;8:1–18.
84. Fatt I. *Physiology of the eye*. Boston: Butterworths; 1978:67–68.
85. Leonard TJK, Muir MGK, Kirkby GR, Hitchings RA. Ocular hypertension and posture. *Br J Ophthalmol*. 1983;67:362–366.
86. Niyadurupola N, Broadway DC. Pigment dispersion syndrome and pigmentary glaucoma-a major review. *Clin Exp Ophthalmol*. 2009;36:868–882.
87. Hughes E, Spry P, Diamond J. 24-hour monitoring of intraocular pressure in glaucoma management: a retrospective review. *J Glaucoma*. 2003;12:232–236.
88. Kang MH, Morgan WH, Balaratnasingam C, Anastas C, Yu D-YY. Case of normal tension glaucoma induced or exacerbated by wearing swimming goggles. *Clin Exp Ophthalmol*. 2010;38:428–429.
89. Schuman JS, Massicotte EC, Connolly S, Hertzmark E, Mukherji B, Kunen MZ. Increased intraocular pressure and visual field defects in high resistance wind instrument players. *Ophthalmology*. 2000;107:127–133.
90. Pecora L, Carrasco MA. Eye-rubbing optic neuropathy. *Am J Ophthalmol*. 2002;134:460–462.
91. Shephard RJ. Aging and exercise. In: Fahey TD, ed. *Encyclopedia of sports medicine and science*. Internet Society for Sport Science. Available at: <http://sportsoci.org>. Accessed March 7, 1998.