

Exerc-eyes

Effects of exercise on ocular health

Emma Hilton examines the scientific evidence which weighs the pros and cons of regular exercise on ocular health, focusing, in particular, on intraocular pressure and ocular blood flow in specific eye diseases, and the adverse ocular effects of some extreme sports.

Exercise takes countless forms, however, in the scientific world specific terms are used to describe it – namely, isometric and isotonic. Isometric describes muscle contraction where, despite an increase in tension, muscles do not shorten in length; this type of exercise increases fitness and builds muscle. In contrast, isotonic describes muscle contraction during which the muscle changes length during a constant tension. During isotonic exercise, muscles can either shorten (concentric contraction), or lengthen (eccentric contraction).

Exercise is also referred to as static or dynamic. Static refers to isometric exercise without movement, i.e. pushing against a stationary object, whereas dynamic refers to isotonic exercise with movement, such as cycling or running. Finally, aerobic exercise refers to programmes which increase oxygen consumption, thereby benefiting the cardiovascular system and lungs. Anaerobic exercise does not require a substantial increase in oxygen delivery and involves short bursts of exertion followed by periods of rest.

What happens during exercise?

Exercise dramatically increases the metabolic demands of muscle tissue and a number of respiratory and cardiovascular adjustments are made to meet this requirement including (Figure 1):

- an increase in heart rate, stroke volume and, consequently, cardiac output
- an increase in ventilation to appropriately match cardiac output
- a change in the pattern of blood flow whereby blood flow to the kidneys, liver, stomach and intestines is reduced in favour of increased blood to the muscles and skin
- a number of biochemical alterations including an increase in blood-oxygen exchange, increase in red blood cell concentration, decrease in plasma volume, and an overall increase in acidity

Two primary responses occur in the eye as a result of exercise:

- intraocular pressure (IOP) decreases
- ocular blood flow alters

Intraocular pressure

In the eye, exercise is associated with a decrease in IOP and this has been demonstrated by a number of studies. In an investigation of 40 Chinese medical students, half of whom undertook a supervised exercise programme for 10 weeks, the exercising group showed a significantly reduced IOP compared to the control group after 10 weeks¹. In a separate study, a 110km march with a 20kg backpack, resulted in two significant drops in IOP – one immediately after the march and another 48 hours later. Both dips

corresponded with a significant peak in plasma osmolarity². In this regard, not only does exercise cause an acute pressure lowering effect, it also initiates a long-term effect.

The reasons why exercise results in reduced IOP in both the short and the long-term are yet to be fully elucidated, however, it seems possible that the properties of IOP reduction during isometric exercise differ to that of isotonic (dynamic) exercise. In a study which compared the ocular effects of the two types of exercise, both were found to result in a reduction in IOP with the latter (isotonic) exercise group showing the largest reduction. The authors proposed that a greater number of muscle fibrils were recruited in isotonic exercise due to a contraction of the muscle in the whole joint movement, which may have had a more profound effect on the rest of the body including the eyes³.

There is some evidence to suggest that isometric exercise-induced IOP reduction is mediated by an associated hypocapnia (decreased PCO₂) as prevention of the condition blocks the reduction in IOP⁴. Physiologically, large changes in PCO₂ are associated with parallel changes in IOP. In contrast, dynamic exercise appears to lower IOP through a separate mechanism unrelated to hypocapnia. The finding that the two types of exercise reduce IOP by independent mechanisms is not entirely surprising as the cardiorespiratory, neuromuscular, and autonomic neural responses to the two forms differ. There is evidence to suggest that dynamic exercise induces ocular hypotension in an exponential manner, i.e. when exercise is mild, IOP may not vary significantly,

Figure 1

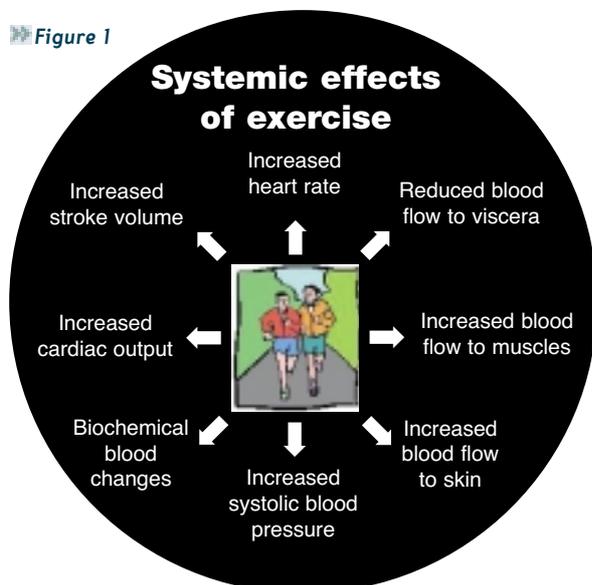
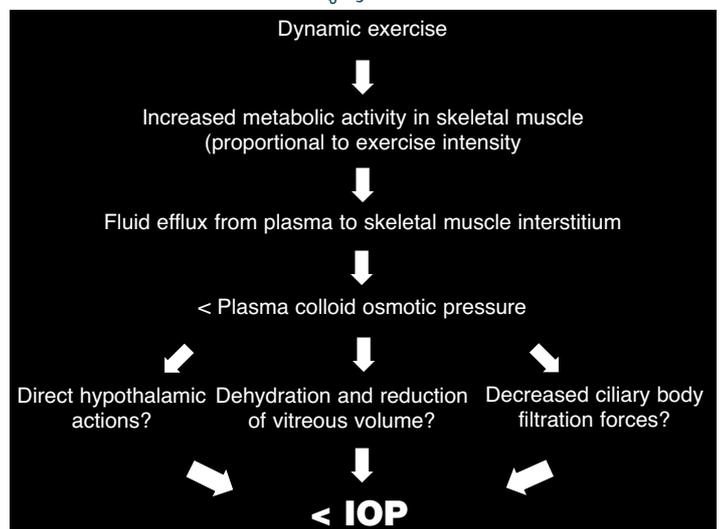


Figure 2

Mechanisms of IOP reduction during dynamic exercise



whereas when exercise is intense, IOP falls significantly⁵.

While IOP reduction during dynamic exercise is linked to intensity, the exercise duration seems less important. In 1999, Harris and colleagues delved deeply into the osmotic mechanistic properties of dynamic exercise-induced hypotension in order to establish the primary mechanism by which IOP becomes reduced. Investigations had already shown that neither plasma osmolality nor hypocapnia accounted for the IOP decline, and that selective and non-selective ocular β -adrenergic blockade was ineffective in altering exercise-induced IOP reduction. In Harris' studies, identical exercises were performed under conditions of varying hydration and osmotic fluid intake in order to best determine which factors were most closely correlated to IOP decline. Of those which were measured, only colloid osmotic pressure, which is linked to capillary ultrafiltration, was directly related to IOP changes during dynamic exercise; hematocrit, total plasma osmolality, and plasma protein concentration failed to show a correlation.

In terms of the eye, this correlation offers three explanations for reduced IOP (Figure 2):

1. Ocular dehydration may occur through osmotic changes in the retinal and uveal vasculature.
2. An increase in colloid osmotic pressure may reduce aqueous formation via reduced ultrafiltration and hence IOP.
3. Altered colloid osmotic pressure could act directly on the hypothalamus resulting in IOP reductions through an unspecified reflex response. The first hypothesis which could potentially result in dehydration and decreased vitreous volume is the most likely explanation⁶.

Blood flow and exercise

Several organs in the body, including the eye and the brain, are autoregulated in order to maintain a constant supply of blood in the face of metabolic stress or imbalance. Blood flow autoregulation refers to the ability of a vascular bed to maintain a relatively constant flow despite moderate alterations in perfusion pressure, or in terms of the eye, ocular perfusion pressure. In a normal healthy eye, ocular blood flow is autoregulated to remain constant when ocular perfusion pressure is increased. Exercise, by its very nature, leads to an increase in systolic blood

pressure and a decrease in IOP. These two components are strongly influenced by the autonomic nervous system, and the result is an increase in ocular perfusion pressure (Figure 3). While a normal healthy eye can very often cope with the stress of altered ocular perfusion pressure, a diseased eye, or an eye whose vasculature is compromised by a pre-existing systemic state, may not.

Autoregulation is facilitated by the adjustment of the peripheral vasculature to changes in perfusion pressure and/or nutritional flow. Pre-capillary sphincters are largely responsible for regulating capillary closure in many parts of the body, however, these regulatory gates do not exist in the eye. Instead, the retinal and choroidal circulations rely on physiological stimuli such as neurotransmitters, myogenic or metabolic factors, circulating hormones and endothelium-derived factors that affect the vascular tone and diameter of the entire arteriolar network.

Exercise-induced blood flow changes

The eye contains several vascular beds and a number of commercially available techniques have been developed and marketed to enable the measurement of blood flow variables at different levels – namely the retrobulbar vessels which supply the eye and the capillary beds of the choroid, optic nerve head and retina. Through the use of these techniques and simulated exercise in different groups of patients, the effect of sports on the eye has been studied.

Silver and co-workers⁷ measured the pulsatile component of the ophthalmic artery blood flow following acute exercise, and reported an increase which was accompanied by a decrease in IOP. In contrast, a separate study⁸, which used colour Doppler ultrasound imaging to measure blood flow velocities of the ophthalmic artery and central retinal artery in healthy volunteers before and after exercise, showed decreased blood flow in the ophthalmic artery, while blood flow in the central retinal artery remained stable. The authors concluded that efficient autoregulatory mechanisms existed for retinal blood flow, which was kept constant during the altered cardiac state, but not for the ophthalmic artery.

In an investigation of ocular pulse following physical exercise-related changes in pulse rate, systolic and diastolic blood pressure, and ocular perfusion pressure⁹, the authors concluded that components of the ocular vasculature elicited an autoregulatory effect which maintained constant ocular pulse amplitude in the presence of altered systemic variables.

While there is extensive evidence to suggest that retinal vessels autoregulate in order to maintain constant blood flow when ocular perfusion pressure is altered, the case for effective autoregulation in the

choroid is less convincing. Lusksch et al¹⁰ noted increased choroidal blood flow during isometric exercise as measured with a laser Doppler flowmeter and observed that the response was abolished with a nitric oxide inhibitor. This shows the importance of endothelium-derived nitric oxide in the vasodilatory response of the choroid.

Even though the human eye has autoregulatory capacity, certain components of the ocular vasculature are less efficient than others for maintaining a constant blood supply during increased metabolic demand. This finding, while not a problem in the healthy human eye, can be significant for diseased eyes, or eyes vascularly challenged by vasospasm, hypertension, hypotension and atherosclerosis.

Exercise and eye disease

In view of the IOP changes and ocular blood flow effects of exercise, it follows that exercise might have both negative and beneficial effects in eye disease.

Glaucoma

Over 50 years ago, Duke Elder described glaucoma as a "sick eye in a sick body". While this is an oversimplification of the condition, it is true that eyes with glaucoma are sensitive to damage from lack of nutrients and free radical formation, which are essentially determined from diet and lifestyle. Many years ago, a Washington University group demonstrated that people with normal tension glaucoma were more likely to suffer progressive damage if they were sedentary. Research has now shown that regular vigorous repeated exercise can reduce IOP by as much as 4mmHg; for some glaucoma patients, this reduction may be enough to protect the retinal ganglion cells from further damage. In addition to the acute-phase IOP lowering effect of exercise, consistent exercise programmes decrease ocular pressure in the long-term.

In one study which evaluated seven normal and seven glaucomatous sedentary individuals during various exercise programmes, the IOP reduction was most pronounced in the glaucomatous group¹¹. Furthermore, exercise has been shown to augment the IOP lowering effect of β -receptor blockers in glaucoma patients. As a result of this positive effect, some clinicians share the idea of adding an exercise programme to pharmacological intervention for glaucomatous patients.

While exercise can help reduce IOP which is directly beneficial to glaucoma, it also has other indirect positive effects. Just the very act of losing weight (for overweight people) has the beneficial effect of lowering IOP. Regular exercise also helps to control blood pressure which can help protect capillaries which would otherwise be predisposed to damage by raised blood pressure.

Figure 3
Calculation of ocular perfusion pressure

$$\text{Ocular Perfusion Pressure} = \left(\frac{2}{3} \text{MAP} \right) - \text{IOP}$$

Where:

$$\text{Mean Arterial Pressure (MAP)} = \frac{\left(\frac{2}{3} \text{Diastolic BP} \right) + \text{Systolic BP}}{3}$$

In order for exercise to be beneficial to patients with glaucomatous optic neuropathy, it has to be maintained at a certain level. Researchers in Oregon have shown that exercising for approximately 20 minutes, four times weekly, lowers IOP. Moreover, not all forms of sport are helpful – indeed, some are detrimental. In particular, patients with glaucoma should limit exercises where the legs are raised higher than the eyes (such as yoga head stands) for any length of time as this action raises IOP. Similarly, weight lifting, while good for developing muscle tone, is not known to have any beneficial effects for glaucoma and the straining associated with lifting heavy weights and accompanying breath holding may raise IOP. Instead, aerobic exercises such as cycling, swimming and walking are recommended for people with glaucoma.

While exercise may help many individuals, exercise that is so strenuous that it results in a reduction of blood flow to the optic nerve and free radical formation, may be detrimental. In 2001, the case reports of two glaucoma patients with exercise-related visual loss were presented¹². In each case, visual acuity, foveal function and visual fields were all reduced following exercise and the authors proposed a vascular steal phenomenon to explain the apparent visual function reduction. In this phenomenon, blood flow is thought to be redirected towards the heart and muscles to cope with increased metabolic demand at the expense of other vascular beds including the eye. In normal healthy eyes, an autoregulatory capacity is present to maintain a constant supply of blood even during times of metabolic stress. However, there is extensive evidence to suggest that the autoregulatory reserve of glaucomatous eyes is diminished.

While IOP normally falls in response to exercise in normal healthy individuals and patients with primary open angle glaucoma, several cases of anterior chamber pigment dispersion with accompanying IOP elevation have been reported in patients with pigment dispersion syndrome following jogging. This phenomenon, which may be attributable to the mechanical obstruction of trabecular outflow spaces by pigment, could contribute to, or predispose to optic nerve head damage and visual field loss. While not all patients with this form of glaucoma should be advised to avoid exercise, those who regularly partake in jogging or more strenuous, jarring exercises may benefit from an ophthalmic evaluation before and after the activity¹³. In the event that exercise-induced pigment dispersion does occur, pilocarpine therapy may be a preferential alternative to abstaining from exercise.

Diabetes

The effect of isometric exercise on choroidal blood flow in type I diabetes was

examined in some patients with diabetic retinopathy and some without. Exercise resulted in a substantive increase in ocular perfusion pressure in both groups. This was accompanied by a decrease in choroidal blood flow in the diabetic retinopathy group, while no change was apparent for the group without diabetic retinopathy. These findings indicate a lack of ocular vascular autoreactivity in the diabetic retinopathy group during isometric exercise¹⁴.

There is no doubt that exercise is beneficial for the management of type 2 diabetes, however, autonomic neuropathy affects between 20-40% of diabetics, most commonly type 2, and necessitates very careful evaluation and specific advice on how to exercise safely.

Uhthoff's sign

Uhthoff's sign, also referred to as Uhthoff's symptom or phenomenon, is a rare neuro-ophthalmological condition linked to multiple sclerosis and characterised by transient visual field loss or blurred vision precipitated by exercise, excess heat, fatigue or anxiety. It is thought that a metabolic by-product of exercise or increased body temperature causes a reversible conduction block in the demyelinated optic nerve culminating in a temporary loss or blurriness of vision. This sign is an indication of optic neuritis and a major risk factor in its recurrence.

Sports and ocular health

While all exercise has some bearing on ocular health some sports are more poignant than others. A review of all sports is outside the scope of this article, however, those sports where the eye is exposed to hyperbaric conditions, notably diving and mountain climbing, deserve special consideration.

Diving

Recreational SCUBA diving (Figure 4) is the most common form of hyperbaric exposure. Exposure to ambient pressures greater than sea level may result in various disorders and complications, some of which have ocular manifestations. Some eye disorders and post-operative conditions may be altered or exacerbated by the underwater environment or other hyperbaric exposure¹⁵. Air divers, those who breath a mixture of inert gas with oxygen, are generally limited to depths of less than 187 feet, which is the critical cut-off point for safe oxygen consumption. As depth increases, so does the partial pressure of oxygen and carbon dioxide in the blood, which may lead to a number of systemic and ocular complications. The prevalence of recreational, military, and commercial diving, as well as the medical use of hyperbaric oxygen therapy, necessitates a familiarity of ophthalmologists and optometrists with the effects of the hyperbaric environment on the normal and diseased eye.



Figure 4
SCUBA diver

(by courtesy of Dr S. Smith, Aston University)



Figure 5

Intraocular gas bubbles in a rat following severe hyperbaric stress

(by courtesy of Professor R. Vann, Duke University Medical Centre, Durham, USA)

Decompression sickness

Decompression sickness may occur following a rapid decrease in ambient pressure during descent where inert gas, which is normally dissolved in the body, tissues is released out of solution in the form of bubbles. A great many systemic complications may occur as a result of decompression sickness including limb pain, lymphoedema, skin discoloration and, in severe cases, neurological and cardiopulmonary manifestations.

Ocular involvement in decompression sickness was first described by Sir Robert Boyle in 1670, when he noted gas bubbles in the anterior chamber of a viper's eye exposed to increased pressure¹⁵. Figure 5 shows an example of a rat eye exposed to similar extreme stress. While bubbles within the eye are rarely seen in humans after diving, bubbles in the tear film outside the eye are fairly routine¹⁶. The incidence of ocular manifestations in patients with decompression sickness has been reported as between 7% and 12% (Table 1).

Decompression sickness is treated by recompression and hyperbaric oxygen therapy immediately after the diver has resurfaced. As treatment generally resolves most symptoms, including ophthalmic manifestations, divers are rarely referred to an optometrist or ophthalmologist. In a minority of cases, an incomplete response to treatment or recurrence of symptoms may mean that divers require medical ophthalmic assistance; however, this is generally of a non-urgent nature. Hyperbaric oxygen therapy, by its very nature, is not without complications, some of which are ocular. In particular, eyelid twitching, blurred vision and visual field constriction may occur. Where prolonged repeated treatments are required, myopic change and cataract formation have been reported.

Arterial gas embolism

Arterial gas embolism is a lesser known disorder where bubble formation occurs. In this case, pulmonary barotrauma occurs during ascent resulting in alveoli rupture and the entrance of gas bubbles to the systemic circulation. This disorder is characterised by the sudden loss of consciousness of the diver after they have reached the water surface and is associated with cerebral and cardiac dysfunction. Ophthalmic symptoms, including

hemianopias and cortical blindness as well as central retinal arterial occlusion, have been reported. As with decompression sickness, this disorder is treated by recompression and hyperbaric oxygen therapy.

Face mask barotraumas

During SCUBA diving, relative changes of the pressure in the diving mask compared to the environmental pressure are transmitted to the eye and the periorcular tissue – barotrauma occurs when there is a loss of pressure equilibrium¹⁷. In most cases, barotrauma occurs when the diver fails to exhale into the face mask during descent, thereby failing to equalise the pressure within the mask. As the pressure differential increases, the eyes and ocular adnexae are drawn towards the space within the mask. Marked lid oedema, bruising and sub-conjunctival haemorrhage may ensue as blood vessels are distorted by the distension.

Although alarming on first appearance, ocular barotrauma is mild and self-limited in the majority of cases resolving without sequelae. However, in more severe cases, perhaps where an unconscious diver sinks to great depths, more serious injury can occur. A rare case of an orbital haemorrhage following a face mask barotrauma was reported in a 41-year old

recreation diver¹⁸. In most cases, however, barotrauma can be avoided if divers are properly instructed in the technique of mask clearing.

Long-term changes

A number of studies have investigated frequent divers for ocular symptoms. In 1988, Polkinghorne and co-workers¹⁹ examined the ocular fundi of 84 divers and discovered areas of low retinal capillary density at the fovea, together with microaneurisms and small areas of capillary non-perfusion. The divers had significantly more abnormalities of the retinal pigment epithelium than non-divers, and the prevalence of the defects was significantly linked to the diving history. The authors observed that all of the diver-related changes appeared to be related to obstruction of the choroidal and retinal vasculature, possibly due to intravascular bubble formation during decompression, or to altered behaviour of blood constituents and blood vessels in hyperbaric conditions.

In a later investigation of the macular functions of frequent air divers, colour vision abnormalities, which were sometimes quite severe, were amongst the visual abnormalities reported in addition to some visual field changes. In these cases, despite the aforementioned abnormalities, visual acuity remained normal and angiographic lesions were not found. It was suggested that some individuals might be more predisposed than others to macular dysfunction during diving²⁰. However, in an investigation comparing retinal fluorescein angiograms of 55 professional naval divers with those of 24 non-diver servicemen, no significant differences were observed²¹.

Ocular surgery

Diving is hazardous for patients who have undergone ocular surgery for three main reasons:

1. the water is a potential source of infection;
2. gas in intraocular cavities may be affected by pressure changes; and
3. face mask barotrauma may result in corneal and conjunctival complications.

In eyes which have previously undergone surgery and contain gas bubbles in the anterior chamber or vitreous cavity, haemorrhages of the retina, uvea and vitreous may occur in response to pressure-induced alterations in the volume of the gas bubble. To prevent this, patients with intraocular gas bubbles should be advised not to dive.

With the increasing availability of refractive surgery, optometrists may be approached for advice from patients who wish to dive. There is a theoretical potential for corneal rupture in divers following surgery, however, the pressure

Table 1
Diving complications and ophthalmic manifestations¹⁵

DIVING COMPLICATION	VISUAL SYMPTOMS
Decompression sickness	Nystagmus Diplopia Visual field defects Scotoma Homonymous hemianopia Ocular pain Cortical blindness Convergence insufficiency Central retinal artery occlusion Optic neuropathy
Arterial gas embolism	Hemianopia Cortical blindness Central retinal arterial occlusion
Gas bubble formation under contact lenses	Corneal oedema
Displaced contact lens	Blurred vision
Contact lens adherence syndrome	Blurred vision Recurrent ocular irritation
Chemical agents to reduce mask fogging	Epithelial keratopathy Blurred vision Photophobia Tearing Blepharospasm
Facemask barotrauma	Oedema Bruising Sub-conjunctival haemorrhage Orbital haemorrhage (rare)

differential required to produce such an effect is extremely high. Radial keratotomy (RK) decreases the strength of the cornea and may increase the risk of serious injury if the eye is subjected to subsequent barotrauma such as face mask squeeze. Despite this theoretical risk, there have been no reports of a traumatic rupture of the cornea following diving after RK. To minimise risk, divers who have had this procedure should wait at least three months after surgery before returning to diving and should be careful to avoid a face mask squeeze. Individuals who have procedures that do not involve incisions in the cornea, such as PRK or LASIK are less likely to experience problems and may dive two weeks following surgery. There are no reports of complications arising following cataract surgery as a result of diving, and the normal recommended recovery period is sufficient.

Mountain climbing

Background visual disturbances following high altitude exposure were first reported in 1969. Later, the term 'high altitude retinal haemorrhage' was introduced encompassing all high altitude retinal haemorrhages and vascular engorgement. Hypoxia at high altitude can lead to increased retinal blood flow. When a hypoxic vascular bed is accompanied by raised retinal venous pressure due to other stresses, such as extreme physical exertion and the valsalva manoeuvre during mountain climbing, the result is a predisposition to intraretinal haemorrhage. In a case report of a 31-year old male

who underwent a Himalayan expedition, glare and decreased vision in twilight, together with intraretinal haemorrhages, tortuosity of dilated arterioles and of the venules, were described. The authors stipulated that all of these retinal changes were reversible within weeks and that high altitude retinopathy can be prevented by ascending slowly and use of supplemental oxygen²². The effects of altitude on the vision of military personnel undergoing a strict exercise regime were investigated²³. The results showed an overall reduction in visual acuity at high altitude and a decrease in electroretinographic (ERG) photopic flicker response which was suggestive of a shift in retinal cone physiology which could resort in subtle effects on vision.

Two Himalayan expeditions were invited to test colour vision perception with the desaturated D15 test at various altitudes and no significant effect on colour vision was noted²⁴. High-altitude retinopathy may occur at high altitudes even in well-acclimatised climbers. In a study of several climbers where retinal capillary blood flow was measured using the Heidelberg Retina Flowmeter before ascent and after descent, two climbers who returned from the expedition with bilateral retinal haemorrhage showed significantly increased retinal blood flow parameters. The authors proposed that this phenomenon might be an indication of mountaineers with a tendency towards ocular vascular dysregulation, and that an insufficiency of the normal autoregulatory response of the retinal circulation under

conditions of chronic hypoxia may play an important part in high-altitude retinopathy²⁵.

Mountain climbers should be carefully advised following ocular surgery. In particular, high-altitude exposure in the early post-operative period following LASIK can lead to visual acuity fluctuations due to a temporary myopic shift in refractive error²⁶.

Conclusion

Virtually all tissues and systems of the human body have been shown to be responsive to programmes of exercise and the eye is no exception. While the intraocular pressure lowering effect of exercise is beneficial, the haemodynamic alterations, particularly in diseased eyes where autoregulation is deficient, may exacerbate visual symptoms. Consequently, optometrists and ophthalmologists should be aware of the pros and cons of exercise, and some extreme sports, on the healthy and diseased eye.

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References

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