LEARNING OBJECTIVES

• Special Focus: A detailed review on the aspects of neuro-protection, lifestyle modifications and Valsalva-type activities as well as globe compression, sleep apnea and complementary medicines
• What's New: A comprehensive example of translating theory and laboratory findings in neuroprotection to clinical practice: the Glucose Story
• Clinical Issues: A review summary of essential requirements needed for the development of neuro-protective strategies. A discussion of potentially promising compounds and currently available data.
• Practical Tips: An expert discussion on how lifestyle habits like practising yoga may influence IOP

Main topic: “Non IOP lowering treatments”

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Special Focus:
Non-IOP-Lowering and Unconventional IOP-Lowering Treatments for Glaucoma

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Core Concepts
- IOP lowering is the only proven therapy for glaucoma at present, but other therapies are frequently used by patients or recommended by ophthalmologists.
- Often ophthalmologists are unaware that their patients use unconventional therapies.
- Marijuana can lower IOP, it is not recommended for glaucoma because of its short-term action and adverse effects.
- Regular exercise provides a sustained IOP lowering and has other health benefits.
- There is insufficient evidence to support the use of vitamins, minerals, or herbal medicines for treating glaucoma.
- Neuroprotection may be clinically useful, based on one trial, but this needs to be confirmed.
- Squeezing or pushing on the eyes can raise the IOP to very high levels, and should be avoided in patients with glaucoma.

Introduction
The only proven treatment to halt or slow the progression of glaucomatous optic neuropathy is to lower the intraocular pressure (IOP). However, in many patients, IOP reduction is insufficient to prevent vision loss. There may be three reasons for this failure:
1) There is progression despite the maximal IOP decrease achievable, whether by medical treatment, laser, or surgery.
2) It can be difficult to reduce IOP in some patients because of poor response to treatments, e.g. non-responders to medications.
3) The risks of large IOP reduction are too great in some patients, e.g. asthma from topical beta-adrenergic blockers or endophthalmitis after trabeculectomy.

For these reasons, there has been great interest in alternative methods to decrease or halt the progression of glaucoma. There are two major categories of such methods. The first includes unconventional methods to lower the intraocular pressure, e.g. alternative drugs such as marijuana. The second category includes treatments that do not affect IOP at all, but nonetheless aim to decrease the rate of progression of vision loss.

The use of complementary and alternative methods to halt or to slow glaucomatous progression has been reviewed several times in the last decade. Many glaucoma sub-specialists and comprehensive ophthalmologists use alternative medicines to treat glaucoma, although not at high frequencies. In their 2002 review, Rhee and colleagues found approximately one in 20 glaucoma patients used medications such as vitamins, herbal remedies, marijuana, acupuncture, or similar alternative therapies. Rhee et al concluded that these approaches were not supported by sufficient evidence.

Interestingly, ophthalmologists may be unaware that their patients take complementary and alternative medications for their glaucoma. A study by Wan and colleagues demonstrated that although about 11% of patients currently used such therapies, 62% of this group had not told their ophthalmologist that they do so. Among Canadian ophthalmologists, Bower and colleagues found that about 20% felt that complementary and alternative medicine had a role in therapy for glaucoma, and about one in 10 recommended such therapy. Clearly there is a need for ophthalmologists to be aware of what complementary and alternative therapies are being used by patients, and their relative risk-benefit ratio.

Alternative methods to lower IOP.
Marijuana
The most well-known example of an alternative drug to reduce IOP is marijuana. People who smoke, ingest, or intravenously receive Delta-9-tetrahydrocannabinol (THC), the active ingredient of marijuana, have significant lowering of IOP. The mechanism seems to be from both cannabinoid and beta-adrenergic receptors within the eye.

With this unconventional approach to reduce IOP, the major problem is the side effects: primarily psychological and systemic hypotension. In one long-term study, all subjects undergoing a trial of marijuana to lower IOP eventually abandoned its use. Attempts to deliver the drug through other routes and thus to bypass effects on the central nervous system have so far been mostly unsuccessful. While a topical delivery of THC should avoid most psychological effects, it does not work well because of poor penetration into the anterior chamber. There are ongoing attempts to design other drugs that would have the same effect as THC on lowering the pressure, but without the systemic and cognitive side effects.

Another major problem with marijuana for glaucoma is that the half-life of the drug itself is very short, usually 3-4 hours. Most current drugs that are used for glaucoma are taken one or two times per day; a drug needing to be taken 6 to 8 times a day is not patient-friendly and would likely lead to decreased adherence. Finally, there has recently been an excellent review of the health effects of marijuana, including those associated with inhalation of smoke and addiction, which concluded that it is truly deleterious to health and well being. This makes the cost-benefit ratio for the use of this drug disappointing compared with other treatments, or even other complementary and alternative treatments. Unsurprisingly, the American Glaucoma Society declared that marijuana is not recommended for the treatment of glaucoma.

Palmitoylethanolamide is a drug that has some of the same effects of cannabinoids (as found in marijuana) without binding to cannabinoid receptors. A clinical trial of oral palmitoylethanolamide in a small number of patients with “normal-tension glaucoma” was performed, comparing it with no treatment. Patients given the drug showed remarkable improvement in visual field parameters with a concurrent decrease in
IOP. This is a provocative result, which should be confirmed in a larger group.

Exercise

Exercise lowers IOP. A recent meta-analysis, published in abstract form\(^1\) reviewed 63 studies that involved exercise and IOP, and found ten that they selected for further review. They concluded “there is a clear effect of exercise on the reduction of IOP ranging from 1 to 5 mmHg.” Both active and sedentary participants responded similarly to the effects of exercise, with the duration of exercise making a difference only for mild but not moderate intensity.

Exercise has to be continued to be effective to reduce IOP. Passo and colleagues studied nine sedentary glaucoma suspects before and after three months of exercise training\(^2\). They found a 30% increase in aerobic capacity and a 20% decrease in IOP. Once the exercise was stopped and the subjects return to their baseline physical state, IOP returned to the initial value by three weeks. Nonetheless, the health benefits of exercise are strong enough to make this approach a good one for physically able patients.

Other nonconventional therapies

Herbal medications may reduce IOP. The most commonly studied medication is vitamin C (ascorbic acid) at high doses. It has a short-term IOP lowering effect, but does not last very long. Other herbal remedies are not thought to lower IOP, but have been studied for their ability to decrease progression. They are listed below in the section on non-IOP lowering therapies.

Meira-Freitas and colleagues studied the role of acupuncture on IOP\(^3\). They found no significant difference between acupuncture versus sham treatment of healthy volunteers in effects on IOP.

Non-IOP lowering therapies

Vitamins and herbal remedies

Most ophthalmologists have been asked by patients whether they should take “eye vitamins” for their glaucoma. Often they are referring to drugs recommended for the prevention of progression of macular degeneration. Such vitamin-mineral drugs are based on the Age-Related Eye Disease Study (AREDS) formulations. Although these formulations have been shown useful for moderate to severe macular degeneration, there is no evidence that they are helpful for glaucoma. Not only is there insufficient evidence that thiamine, vitamin B\(_2\), vitamin A, or vitamin E are helpful for glaucoma, but the latter two have the potential for adverse effects (greater risks of increased intracranial pressure and lung cancer in smokers, respectively).

Herbal remedies have been studied for years with respect to their ability to affect the health of the optic nerve, either directly via positive effects on retinal ganglion cells and their axons or through vascular effects. Examples include ginkgo biloba, and bilberry. Unfortunately, the evidence supporting these therapies is not scientifically rigorous. Wilkinson and Fraunfelder have reviewed most of the important studies in this area\(^4\) and the role of specific antioxidants, as well as nonspecific antioxidants as found in herbal extracts, e.g. from Ayurvedic medicine, have been summarized by Anand et al\(^5\). One mechanism of action may be a direct effect on retinal cells, although again, this remains to be proven in randomized clinical trials.

Neuroprotection

Another approach is pharmacological protection of the retinal ganglion cell and its axon via a neuroprotective mechanism\(^6\). There is substantial preclinical evidence for this approach over some decades. More recently, there have been multiple small clinical studies of neuroprotective drugs and two large studies. The largest study examined the ability of memantine to decrease the progression of glaucoma in two parallel trials of more than 2000 patients. The results of the study have not been published, but press releases from Allergan, the pharmaceutical company that funded the study, indicated that the primary outcome measure in each of the trials was not met. There are no published data on specific results from this study, including other outcome measures and subgroup analyses.

A smaller study of 178 patients tested whether the alpha-2 adrenergic agonist brimonidine could slow the rate of visual field deterioration in subjects with normal pressure glaucoma\(^7\). Fewer patients on brimonidine worsened over time, compared with those receiving timolol. This occurred despite similar decreases in IOP with the two drugs. However, the results of this study need to be confirmed before they can be accepted as hard evidence\(^8\).

Decreasing Valsalva

The Valsalva maneuver is commonly performed during everyday living. It results in increased intrathoracic venous pressure with the consequence of increased IOP. The IOP response to Valsalva maneuver is variable\(^9\). Patients with glaucoma who play brass or woodwind instruments commonly exhibit increased IOP while playing, making this a potential risk factor for progression. However, it is unclear whether the average glaucoma patient needs to avoid the Valsalva maneuver in order to decrease to progression rate as Valsalva also raises intra-cranial pressure and this might ameliorate the effects of increased IOP.

Detection and treatment of sleep apnea

Sleep apnea syndrome is being increasingly recognized in the general population, both as a result of improved diagnostic acumen and the rising prevalence of obesity in the population. Early reports suggested a strong relationship between obstructive sleep apnea and glaucoma. However, this has been controversial. More recent studies using sophisticated epidemiological techniques have shown conflicting results. Stein and colleagues surveyed a database containing more than 2 million individuals in the United States in a managed care network\(^10\). More than 150,000 had sleep apnea, but the frequency of open-angle glaucoma when adjusted for other features was the same in the sleep apnea and non-sleep apnea groups. Similarly, Apel and colleagues looked at a large French cohort in a multicenter prospective study, and found no difference between the incidence of glaucoma with or without sleep apnea when followed over several years\(^11\). Note that it was important to adjust for covariates such as age, body mass, etc. On the other hand, a population-based cohort retrospective study in Taiwan performed by Lin and colleagues showed a hazard ratio of 1.67 for the risk of glaucoma in the sleep apnea group versus the non-sleep apnea group\(^12\).

It is unclear why these studies show different results; they might reflect an effect of sleep apnea on the morphology of the angle, with Asian patients at greater risk of angle closure than among Caucasians. It is thus unclear whether the treatment of sleep apnea would be helpful in glaucoma, and might be significant only in certain individuals. Certainly it would improve patients’ ability to perform visual fields accurately and would help their general health, including cardiovascular, respiratory, and cerebral function.

Avoiding lid squeezing or putting pressure on the globe

Glaucomatous progression can occur despite IOP in the population “normal” range. However, the pressure measured by the physician in the office is a single “still frame” from a continuously running
movie. Second-to-second variations and particularly spikes in IOP is associated with progression of visual loss. We know from the work of Downs and colleagues that monkeys with continuous IOP monitoring have IOP fluctuations of 10 mm Hg or more that are most likely the results of blinks and saccades. More troubling are the effects of squeezing or rubbing the eyes. The lack of a reliable and accurate 24-hour monitoring of IOP means that we cannot assess this possibility in patients. Nonetheless, it is reasonable to ask patients to avoid squeezing or rubbing their eyes and to consider wearing a shield over their eyes when sleeping if they know that they sleep with their eye(s) buried in the pillow or pressing on their hand.

Avoidance of smoking

A meta-analysis published in 2004 examined a possible association between cigarette smoking and glaucoma, and reported a hazard ratio of 1.37 (i.e. a 37% greater risk of developing glaucoma in current smokers). A few years later a systematic review found minimal evidence to support a link and considered the quality of most studies performed so far to be poor. More recently, the Rotterdam study found no evidence for an association between smoking and glaucoma, similar to the negative results seen in the Beaver Dam Eye Study. The Blue Mountains Eye Study showed a very slightly higher IOP in current smokers versus non-smokers (16.34 vs. 16.04 mm Hg). The Latino Eye Study did not show an association between smoking and glaucoma, but the confidence limits were wide and a small effect could have been present but not detected.

With this evidence, preventing glaucomatous progression does not a jus-
tify telling patients to stop smoking, but should be recommended for the value to their overall health.

Summary

A variety of unconventional interventions can be considered to prevent further visual loss in glaucoma patients. The best evidence supports avoidance of mechanical causes of increased IOP (e.g. squeezing and rubbing) or extended Val-salva maneuvers, and for exercise. Neur-roprotective therapies and cannabinoid-related methods to lower IOP may join the armamentarium for glaucoma management in the future.

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What’s New
Taking Retinal Neuroprotection from the laboratory to the clinic: The Glucose Story

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Core Concepts
- Naturally-occurring and inducible models of glaucoma have been used by researchers to delineate that the initial site of damage to retinal ganglion cells (RGCs) is at the optic nerve head and that this likely involves local metabolic compromise.
- In models of retinal neuron damage, preventing energy deprivation by pre-existing hyperglycaemia or intraocular delivery of glucose leads to a robust protection of RGCs.
- Hyperglycaemia also robustly protects RGCs in a rat model of laser-induced glaucoma.
- Topical glucose delivery to pseudophakic POAG patients causes temporary but significant improvement in psychophysical visual parameters such as contrast sensitivity.

Glucoma and neuroprotection
In chronic neurological diseases, like primary open-angle glaucoma (POAG), neuroprotection can be conceptualized as the reduction in the rate of neuronal loss1. Currently, intraocular pressure (IOP) reduction is the only evidence-based neuroprotective strategy for all forms of glaucoma, including so-called "normal tension" glaucoma. Although IOP reduction is generally a successful strategy, some patients continue to progress despite optimal medical and surgical management. Alternative neuroprotective treatment strategies that directly protect retinal ganglion cells (RGCs), including their axons is highly clinically desirable2.

Animal models of glaucoma and RGC death
Since it is unfeasible to study the initiation or progression of glaucoma at a cellular level in humans, researchers have devised a number of experimental animal models to understand better the underlying pathology (Table 1). There are also a number of other related models of RGC injury, which specifically investigate certain features of glaucoma such as models of acute IOP elevation, vessel ligation or physical RGC perikarya/axon damage2,3.

What do laboratory studies tell us about the pathogenesis of glaucoma?
Naturally-occurring glaucoma models are useful in delineating progression

Table 1: Commonly employed animal models of glaucoma

<table>
<thead>
<tr>
<th>Type of Model</th>
<th>Animal</th>
<th>Procedure</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>Dogs (Beagles)</td>
<td>n/a</td>
<td>Begins at 6-12 months Autosomal recessive Excavation of ONH</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Mutant mouse, DBA/2J</td>
<td>n/a</td>
<td>Iris pigment dispersal syndrome Maximum IOP at 11 months Sectorial RGC apoptosis</td>
</tr>
<tr>
<td>Induced</td>
<td>(Cynomolgus) Macaque monkeys</td>
<td>Argon laser-induced photocoagulation of TM</td>
<td>IOP elevation up to 11 months RGC loss Excavation of ONH</td>
</tr>
<tr>
<td>Induced</td>
<td>Mouse</td>
<td>Argon laser-induced coagulation of episcleral and limbal veins</td>
<td>Elevation of IOP from 13-20mmHg at 4 weeks RGC apoptosis by 4 weeks</td>
</tr>
<tr>
<td>Induced</td>
<td>Rat</td>
<td>Diode laser coagulation of TM and/or episcleral veins</td>
<td>Both treatments together caused IOP elevation of 6-10mmHg RGC loss up to 70% (9 weeks)</td>
</tr>
<tr>
<td>Induced</td>
<td>Rat</td>
<td>Episcleral/limbal vein cauterisation</td>
<td>IOP elevated for 3-4 months ERG a/b wave down by 3 months Focal loss of RGCs</td>
</tr>
<tr>
<td>Induced</td>
<td>Rat</td>
<td>Hypertonic saline injection into episcleral veins</td>
<td>IOP elevations of 7-28mmHg Focal RGC axon loss for low IOP elevation; total axon damage for high IOPs.</td>
</tr>
</tbody>
</table>

Notes: TM, trabecular meshwork; ONH, optic nerve head

of the disease or whether a neuroprotective treatment strategy has clinical applicability. However, inducible models provide an opportunity to investigate pathogenesis using an experimentally controlled temporal profile. By using a combination of these approaches, it has been demonstrated that RGC death is apoptotic, axons deteriorate via Wallerian degeneration, and the initial insult to RGCs likely occurs not in the retinal-based perikaryon, but in the axon at the optic nerve head (ONH), where fibres exit the eye through the lamina cribrosa.

It is not clear how these mechanisms initiate RGC death, but local energetic alterations in axons, caused either by elevated metabolic demands as the metabolically fragile axons attempt to cope with increased stresses, or by decreased energy production due to lower blood flow, may play a pathogenic role. In any event, it is likely that an early consequence of changes in the local environment is a compromised RGC metabolism. Specific experimental energy deprivation to RGCs leads to their death by apoptosis, which is also the case in glaucoma.

If energy failure is part of the problem, is energy delivery part of the solution?

In light of these concepts, maintaining the energy supply to RGCs, by ensuring the continuing source of essential energy substrates even if the vascular supply is compromised, might prevent their death in glaucoma. Unusually, compared with other CNS tissues, which produce most of their adenosine triphosphate (ATP) from oxidative phosphorylation (OXPHOS), the retina makes a considerable portion of its ATP via glycolysis, even in the presence of oxygen (aerobic glycolysis). It has been hypothesised that this is due to the heavy biosynthetic load of the photoreceptors, as they constantly renew their outer segment discs. Glucose is thought to be the major energy substrate for all retinal cells. However, elevated blood glucose (hyperglycaemia) might be detrimental to hypoxic brain neurons.

We investigated retinal neuronal protection with induced hyperglycaemia. Rats were made hyperglycaemic by injecting streptozotocin, which is toxic to the insulin-producing beta cells of the pancreas. When animals were made hyperglycaemic before induction of either chronic hypoperfusion, or acute ischaemia, there was a remarkably robust preservation of both retinal structure and function. Following the success of these studies, further tests were conducted in a more relevant experimental model of glaucoma, where IOP was chronically elevated in rats by using a diode laser to photocoagulate the trabecular meshwork and limbal veins. Again, pre-existing hyperglycaemia was able to prevent much of the loss of RGCs seen at two weeks post-pressure elevation. Again the degree of protection was remarkable, with some 50% of RGC perikarya and axons preserved relative to non-hyperglycaemic controls.

From the laboratory to the clinic: Retinal neuroprotection in patients

As we demonstrated that intensive topical glucose therapy in pseudophakic patients elevated the vitreous glucose concentration, we determined whether glucose could rapidly affect vision in glaucoma patients. Two cohorts of non-diabetic pseudophakic POAG patients
were recruited (total 40 eyes from 23 patients split into two separate studies). Eyes randomly received drops of 50% glucose or either physiological or equiosmolar saline as control, every 5 minutes for 1 hour, and contrast sensitivity was assessed as a relevant psychophysical measure of retinal function/recovery, as this parameter is impaired in POAG. Glucose had a profound effect: instillation of this monosaccharide significantly improved mean contrast sensitivity at 12 cycles/degree by 0.26 and 0.40 log units respectively in the two studies compared with saline (Fig. 1). The glucose had no effect on IOP, refraction or central corneal thickness. The effect indicates temporary neurorecovery of “sick” RGCs at the retinal level. Studies continue into most efficient means to supply glucose to deliver a sustained retinal protective effect.

Concluding Remarks

That glucose can offer temporary visual function recovery for POAG patients is a remarkable finding, representing the culmination of a wealth of research. Whether or not this represents a genuine therapeutic pathway is unclear. However, the results provide further evidence:

(1) of energy metabolism compromise in at least some patients with POAG
(2) that a portion of RGCs are sick but able to recover
(3) that visual psychophysical parameters of neurorecovery can be measured rapidly and inexpensively in clinical studies.

These results motivate further bioenergetic-based neuroprotective studies in glaucoma to provide a better understanding of RGC metabolism.

References

Glucoma encompasses a spectrum of progressive optic neuropathies characterized by pathological degeneration of non-myelinated retinal ganglion cells (RGCs), with structural damage at the optic nerve head. Irrespective of the multitude of potential initiating insults, the common theme in the pathogenesis of glaucoma is the triggering of a cascade resulting in accelerated apoptosis of the optic nerve head. Irrespective of the mechanism of neuronal cell death have been identified and explored, leading to numerous research areas targeting mechanisms for neuroprotection in glaucoma:

- Neurotrophic factors
- Calcium Channel antagonists
- Anti-oxidants/Free radical scavengers
- NMDA (N-Methyl-D-aspartate) Receptor antagonists
- Immune mediators
- Apoptosis inhibitors
- Gene therapy
- Stem cells

For any of these areas to be effectively translated to clinical practice, a number of criteria need to be fulfilled when evaluating a glaucoma neuro-protective agent's potential:

- Have a specific and relevant receptor target in the retina or optic nerve;
- Have adequate penetration to reach the retina or optic nerve in pharmacologically effective concentrations;
- Evidence of enhanced neuronal survival or decreased neuronal damage in animal models;
- Demonstrated efficacy in clinical trials

Despite laboratory evidence that several agents may provide neuroprotection in glaucoma, there is currently a lack of published human clinical trials on the use of the majority of these proposed agents. Trials in neuroprotection in glaucoma present unique challenges related to the study population and end point selection. The ideal glaucoma management paradigm will likely combine risk factor modification, IOP reduction, and the use of neuroprotective agents.
sion glaucoma, Ginkgo Biloba extract appeared to improve the visual field or slowed visual field progression.\textsuperscript{14, 15}

In a separate study, no effect on visual field was demonstrated with the use of Gingko Biloba.\textsuperscript{16}

Brimonidine has been studied specifically as a neuroprotectant. In a long term, randomized clinical trial, patients with low-tension glaucoma treated with brimonidine tartrate 0.2% were less likely to have visual field progression than those treated with timolol maleate 0.5%. IOP levels were comparable between groups, suggesting a non-IOP related neuro-protective mechanism of action.\textsuperscript{17}

Currently, we are some way from translating the advances in laboratory modelling to implementation of neuroprotection for glaucoma in our clinics. Nevertheless, in the future, neuroprotective agents will offer the potential for a new treatment paradigm in the management of glaucoma. A disease with multifactorial mechanisms will most likely benefit from a multifactorial treatment approach incorporating individualised treatment strategies, which will likely combine risk factor modification, IOP reduction and the use of neuroprotective agents.

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Practical Tips:

Avoiding Inversions - Intraocular Pressure Changes and Common Yoga Poses

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Core concepts

- Elevated intraocular pressure (IOP) is the most important known modifiable risk factor for glaucoma onset and progression.
- IOP increases on assuming a body position other than seated or upright.
- Yoga has become a popular practice in the western world.
- IOP increases during and following the sirsasana (headstand) posture in healthy and glaucoma subjects. Previous yoga studies have tested only the headstand position, but other common yoga positions also increase IOP.
- Four positions tested showed a significant increase in IOP in all subjects, ranging from a 20% to 73.7% increase in IOP.
- Although the effect of practicing these positions on glaucoma progression remains to be investigated, yoga practitioners should be aware of the significant increase in IOP during these common positions, especially those with severe glaucoma.

Intraocular pressure (IOP) increases on assuming a body position other than seated or upright. This increase is directly related to the inclination of the body toward the complete inverted position. IOP begins to rise upon assuming a head down position and with the body vertical, which results in doubling of the IOP, and IOP remains elevated as this position is maintained. The extent of IOP fluctuations are correlated to the change of position based on angle (ninety degrees upright or inverted) and the length of time maintained.

IOP begins to rise upon assuming a head down position and with the body vertical, which results in doubling of the IOP. In 1998 an estimated 15 million American adults had performed yoga at least once. Studies have shown an increase in IOP during and following sirsasana (headstand) posture in healthy and glaucoma subjects. In a recent study, we identified IOP changes during four standard yoga poses in glaucoma patients and healthy control subjects. Inverted positions increase IOP significantly; however common yoga positions have been incompletely investigated. Yoga practitioners need to know the result of common yoga positions and IOP. The four poses tested using the Reichert Model 30 Pneumatonometer were Adho Mukha Svanasana, Uttanasana, Halasana, and Viparita Karani. (Figure 1) We measured the IOP prior to the pose in a seated position, immediately at the start of the pose, 2 minutes into the pose, immediately after assuming a seated position, and 10 minutes later in a seated position for a final IOP.

The study included 10 subjects (9 women; mean age: 62.3 ± 15.6 years) with primary open-angle glaucoma and 10 healthy individuals (8 women; mean age: 36.3 ± 12.8 years). Within the glaucoma group and within the control group, IOP increased significantly for all 4 yoga positions (repeated-measures ANOVA; all P<0.01) (Table 1).

In a recent study, we identified IOP changes during four standard yoga poses in glaucoma patients and healthy control subjects. Inverted positions increase IOP significantly; however common yoga positions have been incompletely investigated. Yoga practitioners need to know the result of common yoga positions and IOP. The four poses tested using the Reichert Model 30 Pneumatonometer were Adho Mukha Svanasana, Uttanasana, Halasana, and Viparita Karani. (Figure 1) We measured the IOP prior to the pose in a seated position, immediately at the start of the pose, 2 minutes into the pose, immediately after assuming a seated position, and 10 minutes later in a seated position for a final IOP.

Aside from the Halasana position, which reached borderline significance (P=0.08), there was no significant difference between glaucomatous and healthy eyes regarding the IOP response to position changes. The Adho Mukha Svanasana position showed the highest IOP increase: a 66.3% increase for glaucoma subjects and a 73.7% increase for control subjects during this pose. The Uttanasana position showed a 56.5% increase for glaucoma subjects and a 44.9% increase for control subjects. The Halasana position showed a 30.8% increase for glaucoma subjects and a 23.1% increase for control subjects. The Viparita Karani position showed a 19.9% increase for glaucoma subjects and a 20.6% increase for control subjects.

Previous studies have tested only the headstand position, as shown; other positions also increase IOP. All four positions showed a significant increase in IOP in all subjects. Yoga practitioners...
should be aware of the significant increase in IOP during these common positions, especially glaucoma subjects with severe disease. Yoga instructors should also be aware of practitioners who suffer from glaucoma and are performing yoga with them, modified positions perhaps should be suggested and used.

References

Figure 1. Four Yoga Positions Tested.
STATEMENT OF NEED AND PROGRAM DESCRIPTION
Recent months and years have seen significant advances in our understanding of glaucoma. Much has been learned, not only about damage mechanisms and pathogenesis, but also about diagnosis and management. Treatment options – both medical and surgical – continue to expand. This program will review this new knowledge with an emphasis on incorporating recent insights into day-to-day practice.

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