Literature Reviews

https://doi.org/10.31288/oftalmolzh202336170

Preconditioning-induced retinal protection appears promising: a review

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Like human and animal myocardium and cerebral nervous tissue, the human and animal retina has powerful intrinsic adaptive mechanisms which can improve cell protection under adverse conditions. These endogenous protective mechanisms are capable of improving the resistance of retinal cells to adverse factors and can be triggered by brief episodes of different subthreshold stimuli (ischemia, photobiomodulation, hypothermia, etc.) preceding a subsequent more sustained injury, a process which is called preconditioning. Results of years of laboratory and clinical studies have demonstrated amazing cardiac protection and cerebral protection opportunities from preconditioning. Although the results of in vivo preclinical studies of retinal conditioning are promising, the benefits from the found effects of preconditioning on the protection of the human retina are still to be assessed. The overview presented highlights some aspects of the research and use of protective effects of preconditioning in various fields of medicine. The results of laboratory studies of endogenous mechanisms of retinal cell protection are discussed herein, along with our recommendations for prospective areas of future use of the effects in clinical ophthalmology for retinal protection.

Keywords:
preconditioning, ischemic injury, phototoxic injury, heart, brain, retina

The protection of human tissues and organs from ischemic damage is a key trend of contemporary medicine.[1, 2] Tissues with a high sensitivity against ischaemia, i.e. myocardium, central nervous system and retina, present the most promising targets for therapeutic application of novel protection techniques. Results of studies indicated that these tissues have powerful endogenous adaptive mechanisms which can improve both the resistance to ischemic damage and post-ischemic recovery.[3-9]

As early as 1964, Dahl and Balfour [10], in an experimental rat study, found that a brief preliminary exposure to anoxia improved brain tissue tolerance to subsequent longer anoxia, which the authors suggested was due to increased anaerobic glycolysis.[11] Murry and colleagues (1986) [12] reported that brief episodes of subthreshold coronary ischemia-reperfusion preceding a subsequent more sustained period of coronary artery occlusion in dogs protected or "preconditioned" the heart and limited infarct size to 25% of that seen in the control group. The authors suggested that the protective effect of preconditioning may be due to reduced adenosine triphosphate (ATP) depletion and/or to reduced catabolite accumulation during the sustained occlusion.[11]

Recent decades have demonstrated significant progress in identifying endogenous reactions protecting against ischemia and using them in routine clinical practice. Numerous animal studies and subsequent clinical studies have demonstrated that tissue preconditioning with brief sublethal ischemic stimuli is a powerful endogenous protection from cerebral ischemia, ischemic myocardial injury and their consequences.[12-15] The concept of ischemic preconditioning (IPC) implied that a brief subcritical ischemic challenge could mobilize intrinsic protective mechanisms, increasing tolerance against subsequent critical ischemia.[1, 11]

It was found later that applying brief repeat periods of ischemia to remote organs, including the kidney, intestine and skeletal muscle, can also protect the heart from subsequent myocardial infarction.[16, 17, 18, 19, 20]. This is called remote IPC and is less invasive and simpler to implement in clinical practice than classical preconditioning.[21, 22, 23]. There have been reports [24, 25, 26, 27] on the use of remote IPC to elicite protective response not only in the heart, but also in other target organs (the brain, kidneys and lungs).

In addition, studies have demonstrated that not only a brief subcritical ischemic challenge, but also hypoxia,

hypothermia, hyperthermia, light, inhalational anesthetics and other chemical substances can trigger endogenous protection from ischemic or other damage.[28, 29, 30, 31, 32]. Cross-preconditioning refers to prior exposure to a noxious stress other than ischemia that confers ischemic tolerance.

The mechanisms underlying preconditioning protective effects are numerous, intricate and still poorly understood. Preconditioning triggers adaptive cellular and tissue responses which prepare the tissue to a subsequent actual ischemic damaging event. Preconditioning can be subdivided into early and late mechanisms, depending on whether the effect appears immediately after the nonlethal stress or with a delay of some hours or days. [1] Early protective mechanisms can be induced within minutes of exposure to preconditioning stimuli and are associated with generation of signaling molecules which bind to specific membrane receptors and activate a number of signaling intracellular responses.[1, 6, 33] Late protective mechanisms of preconditioning result from gene activation and de novo protein synthesis.[1, 6, 33] In addition, beneficial effects of preconditioning are due to the attenuation of damage-inducing processes including excitotoxicity, oxidative stress, metabolic dysfunction, inflammation, and necrotic and apoptotic cell death.[6]

The protective signals of remote preconditioning are transferred from distant tissues to the target organ likely through humoral, neuronal and immune pathways. After being transmitted to the target organ, these protective signals use a final common pathway to induce tolerance to damaging effects.[9]

Interestingly, numerous studies have reported on the activation of endogenous adaptive mechanisms (in the myocardium, brain and retina) by inducing brief non-lethal episodes of ischemia or other stimuli to the target organ not only prior to, but also during, or even after an episode of sustained lethal injury - a phenomenon termed preconditioning, perconditioning or postconditioning, respectively.[8, 34, 35, 36, 37, 38, 39]

Preconditioning-induced cardiac tissue protection

Adaptation of the myocardium to ischemia after ischemic preconditioning have been observed in patients with unstable angina and those in the setting of coronary artery bypass surgery and in the setting of coronary angioplasty.[40, 41, 42] In patients with transient episodes of preinfarction angina, as compared with those without, thrombolytic therapy resulted in more rapid reperfusion and smaller infarcts.[43, 44] It is believed that, in this case, IPC is a potential protective mechanism against myocardial infarction.[42]

Yellon and colleagues [41] reported a study examining the effects of a preconditioning protocol of two cycles of 3 min of global ischemia (induced by intermittent cross-clamping the aorta and pacing the heart at 90 beats/min) followed by 2 min of reperfusion before a 10-min period of global ischemia and ventricular fibrillation. Changes in ATP content from needle biopsies of left ventricular muscle were used as the end point in this study. It was found that patients subjected to this preconditioning protocol had better preservation of ATP levels during the subsequent global ischemic period. Based on these findings, the authors concluded that the biochemical changes observed in the human hearts in response to the IPC in their study were almost identical to those observed in canine hearts by Murry and colleagues.[41] Others [45, 46, 47] confirmed the efficacy of IPC in the setting of coronary angioplasty. Some researchers [48, 49] supposed the beneficial effect of IPC in ischemia- and reperfusion-induced arrhythmias. Cohen and colleagues [50] and Sun and colleagues [51] reported on IPC-induced alleviation in transient coronary occlusion (also called myocardial stunning) in rabbits and pigs, respectively. Therefore, IPC may be considered as a powerful phenomenon that provides potent therapeutic myocardial protection in humans.[33]

Remote IPC has also reported to be beneficial for cardioprotection. Others [21, 23, 52] cardioprotective and prognostic effects have demonstrated of remote IPC on myocardial injury in patients undergoing coronary artery bypass graft surgery and children undergoing surgical repair of congenital heart defects, with improvements in the release of serum biomarkers of ischemic injury and perioperative myocardial protection. In patients undergoing elective open abdominal aortic aneurysm repair, remote IPC reduces the incidence of postoperative myocardial injury, myocardial infarction, and renal impairment, with improvements in the release of serum biomarkers of myocardial injury and renal function.[26] A number of studies [53, 54, 55, 56, 57, 58], however, have found no substantial difference in the release of serum biomarkers of ischemic injury or clinical treatment outcomes between patients undergoing remote IPC and controls.

Therefore, the results of clinical studies are highly variable in terms of the beneficial effects of remote IPC, and the mechanisms underlying the protective effects of remote IPC are not completely understood. Nevertheless, a number of factors (like administration of particular medications and anesthesia protocols) have been confirmed to have an impact on the efficacy of IPC.[46, 47, 59] Myocardium from diabetic patients taking long-term oral sulfonylurea hypoglycemic agents (non-specific inhibitors of the ATP-sensitive potassium (K_{ATP}) channel) has been shown to be resistant to the protection by IPC.[60] In addition, the impact of the aforementioned factors has not always been taken into account in clinical study protocols and controlled, which could affect study results.[61]

Preconditioning-induced brain protection

Moncayo and colleagues [62] found that patients with transient ischemic attacks (TIAs) lasting 10 to 20 minutes before cerebral infarction (CI) had a more favorable outcome than those without TIAs before CI. They suggested that ischemic tolerance may play a role in patients with ipsilateral TIAs before CI, allowing better recovery from

a subsequent ischemic stroke. More recently, Wegener and colleagues [63] concluded that the beneficial effect of TIAs on lesion size in apparent diffusion coefficient (ADC) and T2 suggests the existence of endogenous neuroprotection in the human brain.

Chan and co-authors [64] evaluated the effects of ischemic preconditioning, produced by 2 min proximal temporary artery occlusion and 30 min reperfusion, on brain tissue gases and acidity during clipping of cerebral aneurysm. The results of their study suggested that ischemic preconditioning attenuates tissue hypoxia during subsequent artery occlusion, and brief occlusion of the proximal artery may be a simple maneuver for brain protection during complex cerebrovascular surgery. Others [65] also suggested that repetitive brief occlusion of the proximal artery with intermittent reperfusion reduced the risk of stroke compared with uninterrupted ischemia of similar duration in patients undergoing surgical repair of ruptured intracranial aneurysms.

It is believed that remote IPC also has neuroprotective effects. Thus, Meng and colleagues [66] provided a proof-of-concept that brief repetitive bilateral arm ischemic preconditioning may be an effective way to improve cerebral perfusion and reduce recurrent strokes in patients with symptomatic atherosclerotic intracranial arterial stenosis. In a study by Sales and colleagues [67], remote IPC in patients with brain tumors undergoing elective surgical resection was induced by inflating a blood pressure cuff placed on the upper arm three times for 5 min at 200 mmHg in the treatment group after induction of anesthesia. The authors concluded that application of remote IPC was associated with reduced incidence of postoperative ischemic tissue damage in patients undergoing elective brain tumor surgery. Sangeetha and colleagues [25] concluded that remote IPC was feasible and safe in patients with aneurysmal subarachnoid hemorrhage (aSAH) and resulted in a lower incidence of cerebral vasospasm and better functional outcome.

Preconditioning-induced retinal protection

The results of the studies on the mechanisms underlying preconditioning and adaptation to myocardial and cerebral ischemia made it possible to hypothesize that the retinal neural tissue may be preconditioned. A number of experimental studies on the mechanisms underlying retinal protection by preconditioning have confirmed this hypothesis (Table 1).

In an experimental in vitro study, Caprioli and colleagues [68] concluded that the neuroprotective effect of hyperthermia and sublethal hypoxia suggests that heat shock proteins confer protection against ischemic and excitotoxic retinal ganglion cell death. More recently, Roth and colleagues [69] demonstrated the capacity of brief preconditioning ischemic episodes to attenuate subsequent retinal ischemic injury in rats. Retinal ischemia for 5 minutes constituted the preconditioning stimulus. To assess the time course of preconditioning, animals underwent a 5-minute preconditioning episode and then 60 minutes of ischemia 1, 24, 72, or 168 hours later; or they underwent a 5-minute sham experiment and 60 minutes of ischemia 24 hours later. In contrast to the nonpreconditioned rats preconditioned rats had complete recovery of the a- and b-waves compared with preischemic baseline amplitudes, and ischemia-induced histologic damage was completely prevented when preconditioning was performed 24 or 72 hours (but not 168 hours) before ischemia. Separation of preconditioning and 60 minutes of ischemia by 1 hour caused an even greater impairment of functional retinal recovery compared with that seen in sham-preconditioned rats.[69] Obviously, those authors observed late, but not early effects of IPC.

Sakamoto and colleagues [70] aimed to clarify whether early IPC could be observed in the rat retina by histological examination. Animals underwent a 5-minute preconditioning episode and then 60 minutes of ischemia 5, 10, 20, 30, 40, 50 or 60 minutes later. Five minutes of preconditioning ischemia 20-40 minutes (but not 5, 10, 50 or 60 minutes) before 60 min of sustained ischemia completely prevented the retinal tissue damage induced by the sustained ischemia.[70] Therefore, early protective effects of IPC were demonstrated in the rat retina.

Multiple mechanisms have been reported to potentially underlie the preconditioning-induced retinal protection from injury/disease including: binding of adenosine to its A_1 and A_{2a} receptors, activation of protein kinase C, induction of heat-shock protein 27 (Hsp27), upregulation of erythropoietin receptor (EPO-R), activation of nitric oxide synthase (NOS), opening of mitochondrial K_{ATP} channels, and inhibition of mitogen-activated protein kinases (MAPKs) [70-78]. Zhang and colleagues [76] used a rat model to investigate the effect of IPC on apoptosis after ischemia and some of the key proteins involved in the apoptotic cascade. They concluded that IPC protects the rat retina against ischemic injury, in part, by attenuating caspase activation, diminishing apoptosis-related gene expression and by altering protein phosphorylation. Studies [79] have provided evidence that hypoacetylation associated with ischemic injury results from the selective rise in histone deacetylase (HDAC)1/2 activity and that neuroprotection induced by IPC is mediated in part by suppressing HDAC activity. In addition, it was found [80] that the inhibitory effects of IPC on inflammatory leukocyte–endothelium interactions in the postischemic rat retina would partially contribute to the neuroprotective effect on the ischemic insult. The results of a study by Nishiyama and colleagues [81] suggested that the mechanism of preconditioned retinal ischemia may be related to retinal Müller cells which have a pivotal role in the maintenance of retinal homeostasis and regulate the glutamate/glutamine metabolic cycle.

It is noteworthy that the protective effect of IPC is transient. IPC may be induced and its protective effect be allowed to dissipate, and then be reinduced by later repeated application.[71] Differences in the intensity, duration, and/or frequency of a particular stress stimulus determine

Table 1. Experimental studies of preconditioning-based retinal protection

Authors	Year	Animals	Type of preconditioning stimuli	Time between preconditioning and insult	Damaging factor	Effect revealed
Faktorovich E.G., et al[86]	1992	rats	Needle introduced subretinally	48 hours	light, 7 days, 1300-1800 lux	Mechanical preconditioning stimulus causes a decrease in the amount of phototoxic injury to photoreceptors
Roth S., et al[69]	1998	rats	5-min ischemia	1 hour	60-min ischemia	Deterioration in the structure and electrical function of the retina following ischemic injury
			5-min ischemia	24, 72 hours	60-min ischemia	Complete functional and histologic protection against ischemic damage in the retina by previous preconditioning with nondamaging ischemia
			5-min ischemia	168 hours	60-min ischemia	No functional and histologic protection against ischemic damage
Liu C., et al[89]	1998	rats	Light, 12 hours, 1400 lux	48 hours	light, 7 days, 1400 lux	Preconditioning with light evokes a protective response against light damage in the retina
Li B., et al[71]	1999	rats	5-min ischemia	24 hours	60-min ischemia	The neuroprotective effects of IPC in the retina are lost over time but may be reinduced by subsequent application of the IPC stimulus. The role of adenosine as a mediator of IPC was confirmed.
Lin J., et al[82]	1999	rats	5-min ischemia	24 hours	30-min ischemia	Prevention of post-ischemic retinal hypoperfusion and complete recovery of retinal electrical function
			5-min ischemia	24 hours	60-min ischemia	Prevention of post-ischemic retinal hypoperfusion
			5-min ischemia	24 hours	75-min ischemia	Post-ischemic retinal hypoperfusion
Nishiyama T., et al[81]	2000	rats	5-min ischemia	24 hours	60-min ischemia	Role of Müller cells in IPC-induced protection was demonstrated
Nonaka A., et al[80]	2001	rats	5-min ischemia	24 hours	60-min ischemia	IPC inhibits inflammatory leukocyte–endothelium interactions in the postischemic rat retina
Zhang C., et al[76]	2002	rats	8-min ischemia	24 hours	45-min ischemia	IPC attenuates apoptotic cell death in the rat retina
Toprak A.B., et al[112]	2002	rats	5-min ischemia	24 hours	60-min ischemia	IPC protects retinal structure from ischemic damage
Casson R.J., et al[83]	2003	rats	5-min ischemia	24 hours	light, 48 hours, 2000 lux	IPC protects photoreceptors against light-induced injury
Sakamoto K., et al[70]	2004	rats	5-min ischemia	5 and 10 min	60-min ischemia	Deterioration of retinal structure following ischemia
			5-min ischemia	20, 30, and 40 min	60-min ischemia	Protective effect of early IPC in the retina. IPC protects retinal structure from ischemic damage.
			5-min ischemia	50 and 60 min	60-min ischemia	Deterioration of retinal structure following ischemia
Ozbay D., et al[111]	2004	rats	5-min ischemia	48 hours	30-min ischemia	Retinal sections from rats in the IPC group showed a well-preserved retinal structure

Table 1 (continuation). Experimental studies of preconditioning-based retinal protection

Authors	Year	Animals	Type of preconditioning stimuli	Time between preconditioning and insult	Damaging factor	Effect revealed
Dreixler J.C., et al[77]	2009	rats	8-min ischemia	24 hours	ischemia	The role of EPO-R in IPC-induced retinal protection from ischemia was determined
Albarracin R., et al[90]	2011	rats	Light 670 nm, 60 mW/cm^2 3 min for 5 days	24 hours	light, 24 hours, 1000 lux	Preconditioning with photobiomodulation protects retinal structure and function from phototoxic damage
Zhu Y., et al[98]	2012	mice	Hypoxia with 11% O_2, 2 hours for 2 weeks	72 hours	Intraocular hypertension, 3-10 weeks	Hypoxic preconditioning protects retinal ganglion cells from apoptosis and reduces axonal damage
Salido E.M., et al[88]	2013	rats	20-min hypothermia	24 hours	40-min ischemia	Hypothermic preconditioning protects retinal structure and function from ischemic damage
Fan J., et al[79]	2016	rats	5-min ischemia	24 hours	45-min ischemia	Retinal neuroprotection induced by IPC is mediated in part by suppressing HDAC activity.
Brandli A., et al[84]	2016	rats	5 min×2 remote ischemia	15-30 min	light, 24 hours, 1000 lux	Remote IPC protects retinal photoreceptors against bright light–induced photoreceptor degeneration
Iliescu D.A., et al[96]	2018	rats	60-min sevoflurane 2%	1 hour	light, 60 min, 20000 lux	Sevoflurane preconditioning protects the structure and function of retinal photoreceptors and bipolar cells from the damaging effect of photostress

whether that stimulus is too weak to elicit any response, of sufficient magnitude to serve as a preconditioning trigger, or too robust and therefore harmful.[6] One of the disadvantages of IPC, which cannot be ignored, is that IPC is capable of leading to serious damage with only small changes in the timing, durations, and location of sublethal ischemic insults.[9] In addition, IPC's protective effect depends on the duration of a subsequent ischemic episode. [82]

An experimental study by Casson and colleagues [83] have demonstrated that IPC can protect the retina not only against ischemic injury, but also against phototoxic injury. They concluded that IPC upregulates basic fibroblast growth factor (bFGF), glial fibrillary acidic protein (GFAP) and the apoptosis regulator Bcl-2, and these factors may be involved in the protective response.

Retinal protective effect can be also triggered by remote IPC. In a rat study by Brandli and colleagues [84], repeated episodes of ischemia and reperfusion in one hind limb of rats were applied to the retina. Remote ischemic preconditioning was found to protect the retina against phototoxic injury, with preservation of retinal electrical activity and histological structure and a reduction in photoreceptor apoptosis caused by light damage. Others have reported evidence of the potential to use cross-preconditioning stimuli to protect retinal cells against ischemic and phototoxic injury. It has been reported [85, 86] that mechanical trauma of the retina may act as a preconditioning stimulus and create conditions for subsequent photoreceptor protection from phototoxic injury. Findings of a study by Wen and colleagues [87] strongly suggested that increases in endogenous basic fibroblast growth factor (bFGF) and/or ciliary neurotrophic factor (CNTF) play key roles in mechanical injury-induced photoreceptor rescue.

Salido and colleagues [88] investigated hypothermia as a potential preconditioning stimulus to protect the rat retina from ischemia/reperfusion damage. Twenty-four hours before ischemia, animals were exposed to a brief period of global or ocular hypothermia (i.e. hypothermic preconditioning, HPC). Fourteen days after ischemia, they were subjected to electroretinography (ERG) and histological analysis. There was ERG evidence that global or ocular HPC afforded significant functional protection in eyes exposed to ischemia/reperfusion injury. Global or ocular HPC significantly preserved retinal structure and ganglion cell count. The authors [88] gave their reasons for proposing a glutamate-dependent mechanism of HPC-induced retinal neuroprotection from ischemia, with retinal Müller cells being a putative target for the protective effect of HPC on ischemia/reperfusion damage. Therefore, when applied systemically to the whole body or when applied locally, preconditioning stimuli can activate endogenous retinal protection.[8]

Preconditioning with bright light can be used to attenuate the consequences of phototoxic retinal injury.

Liu and colleagues [89] used a preconditioning paradigm to show that rats preconditioned with fluorescent light became resistant to subsequent light damage. They found that preconditioning induced an increase in bFGF and CNTF, stimulated the phosphorylation of extracellular signal-regulated protein kinases (Erks), and reduced the extent of retinal structural damage. They also suggested an important role of Müller cells in preconditioning-induced photoreceptor protection. Albarracin and colleagues [90, 91] reported that 670-nm light-emitting-diode photobiomodulation is protective against bright-light–induced retinal degeneration in rats. Treatment with 670-nm light led to reductions in (a) the extent of phototoxic injury to photoreceptors, retinal pigment epithelial and Müller cells, and (b) the levels of markers of inflammatory stress in the retina.

Pharmacological preconditioning (e.g. with inhalation anesthetics) can be used to protect the retina against phototoxic damage. Some inhalation anesthetics have been found to improve the preconditioning effect and to be independently capable of protecting the tissues of the heart, kidneys and other organs by reducing ischemia/reperfusion damage and inducing anti-inflammatory, anti-necrotic and anti-apoptotic effects.[2, 57, 93-95] There have been reports on the protective effect of inhalational anesthetic preconditioning against light-induced injury. It is believed that inhalation anesthetics provide neuroprotection by attenuating neuronal excitotoxicity, inflammation and apoptocis. Iliescu and colleagues [96] explored the effect of sevoflurane anesthetic preconditioning on a model of light-induced retinal degeneration in diabetic rats. Results showed that sevoflurane has a protective effect on light-induced neuroretinal degeneration proved by significantly less variations of the ERG before and after photostress.

The results of mouse studies by Zhu and colleagues [97, 98] described a novel form of sustained retinal ischemic tolerance, wherein endogenous adaptive responses triggered by repeated episodes of sublethal hypoxia afford protection against apoptosis of retinal ganglion cells and axonal injury many weeks after the preconditioning stimulus. The authors [97, 98] suppose that the ability to induce such a sustained, cell death–resistant phenotype may be therapeutically advantageous, not only for protecting the vision of glaucoma patients, but for saving neurons in other neurodegenerative diseases as well.

Experimental studies have demonstrated retinal protective ability of cross-preconditioning stimuli in the form of hyperthermia [99], hyperbatic oxygenation [100], oxidative stress [101], and aerobic exercise [102].

Effective preconditioning stimuli are numerous and diverse, suggesting that a downstream convergence of signalling pathways promotes this protective response. [6] Despite the amount of knowledge available on preconditioning, it is still unknown whether neuroprotective effects from various protective response-inducing stimuli may be potentiated.

The success of laboratory studies in the field of retinal conditioning have not been confirmed by clinical trials, and the beneficial effects of preconditioning are not used in the practice of ophthalmology.[8] Cardiac and cerebral conditioning, however, are used in cardiac surgery and neurosurgery to protect cardiac tissue and brain, respectively, from ischemic injury.[23, 26, 66, 67, 103] Hypothetically, vitreoretinal surgery may be a promising field for further research on the use of preconditioning for retinal protection. During vitrectomy, insufficient blood pressure and/or elevated intraocular pressure (IOP) result in decreased perfusion pressure, leading to additional perioperative ischemic retinal and optic nerve injury.[104] In addition, light from a fiberoptic endoilluminator used in vitrectomy can cause retinal phototoxic damage.[105] It is not uncommon that vitrectomy procedures take a long time (120 minutes or longer).[106, 107] Therefore, it is supposed that the use of some variants of cross-preconditioning (e.g., in the form of photobiomodulation or local ocular hypothermia) before surgery would contribute to induction of neuroprotective mechanisms and create conditions for retinal protection against perioperative ischemic and phototoxic injury.[108-110]

It is, however, obvious that a set of biochemical and biophysical processes taking place in the retina of eyes with a vitreoretinal disease (especially under conditions of surgery) is rather diverse, and one type of preconditioning strategy may not induce enough retinal protection. It is likely that in patients having a vitreoretinal surgery it would be reasonable to combine different available retinal protection approaches while avoiding the use of factors that can deteriorate this protection (e.g., intraoperative elevated IOP, deep hypothermia, and/or phototoxic illumination sources).

Conclusion

Short-time application of different types of subthreshold preconditioning stimuli capable of mobilizing intrinsic protective mechanisms in tissues (ischemia, photobiomodulation, hypothermia, etc.) can improve the resistance of retinal cells to ischemic or phototoxic injury. Although the protective effects of preconditioning have been not realized in clinical ophthalmology, the considerable experience accumulated in experimental studies of different types of retinal preconditioning, and the results of clinical applications of preconditioning in heart surgery and neurosurgery allow us to suppose that this approach to retinal protection may be promising. Further laboratory and clinical studies are required to allow for the application of the beneficial effects of preconditioning for additional retinal protection prior to vitreoretinal surgery. With this in mind, it is reasonable to take notice of easy-to-use types of preconditioning (like photobiomodulation or local hypothermia) which could be used within hours or days prior to planned surgery.

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Disclosures

Received 06.02.2023
Accepted 27.02.2023

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***Author Contributions:** The authors confirm the following contributions to the article—study conception and design: NP; data collection and analysis: OZ, AK; drafting of the manuscript: OZ. All authors read and approved the final manuscript.*

***Funding Sources:** This research received no special grant from any funding agency in the public, commercial, or non-for-profit sectors.*

***Conflict of interest:** All authors have read the journal's Author Agreement and Conflict of Interest policy. The authors have no potential conflict of interest to declare.*

***Abbreviations:** PC, preconditioning; IPC, ischemic preconditioning; TIA, transient ischemic attack*