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editorial-office@sciformat.ca

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OCULAR BIOMECHANICS AND INTRAOCULAR PRESSURE CHANGES DURING HIGH-INTENSITY RESISTANCE TRAINING. A REVIEW

Vlada Tetarchuk *(Corresponding Author, Email: vladatetarchuk@gmail.com)*
Wielospecjalistyczny Zespół Opieki Zdrowotnej "Bolesławiec-Zgorzelec", ul. Lubańska 11-12, Zgorzelec, Poland
ORCID ID: 0009-0002-7754-4113

Anastasiia Tanko
Szpitale Pomorskie Sp. z o.o., Szpital św. Wincentego a Paulo, ul. Wójta Radtkego 1, Gdynia, Poland
ORCID ID: 0009-0005-6931-5718

Andrei Trutsko
Uniwersyteckie Centrum Kliniczne Warszawskiego Uniwersytetu Medycznego ul. S.Banacha 1A, Warszawa, Poland
ORCID ID: 0009-0003-5674-7971

Olena Abramchuk
Centrum Medyczne Unimed, ul. Młodej Polski 7, Kraków, Poland
ORCID ID: 0009-0000-2604-0287

Vladyslav Tetarchuk
Wielospecjalistyczny Zespół Opieki Zdrowotnej "Bolesławiec-Zgorzelec", ul. Lubańska 11-12, Zgorzelec, Poland
ORCID ID: 0009-0003-6743-6319

Artem Shchebetun
Zespół Opieki Zdrowotnej, Kościuszki 74,Wągrowiec, Poland
ORCID ID: 0009-0004-3044-4793

Daria Shchebetun
Zespół Opieki Zdrowotnej, Kościuszki 74, Wągrowiec, Poland
ORCID ID: 0009-0004-0105-2512

ABSTRACT

Background. High-intensity resistance training is a popular practice among powerlifters and bodybuilders aiming to achieve maximal neuromuscular adaptations and strength gains, yet a heavy lift provokes distinct acute physiological responses, including a massive increase in intra-abdominal and systemic arterial pressure.
Aim. This study aimed to analyze recent evidence on the acute physiological effects of heavy resistance training, lifting techniques, and specific breathing patterns on IOP, ocular biomechanics, and vision-threatening risks.
Material and methods. A comprehensive review of the literature was performed across PubMed, MEDLINE, EMBASE, Google Scholar, and Cochrane Library databases, focusing on clinical studies, laboratory physiological experiments, consensus statements, and randomized trials published between 2016 and 2026.
Results. Exercises provoke increases in IOP due to the Valsalva maneuver performed during the concentric phase of multi-set lifting. Importantly, IOP spikes depend on the percentage of the 1-repetition maximum lifted and the duration of breath holding; these spikes restrict retrobulbar blood flow and induce an acute increase in choroidal blood volume. Variations in corneal biomechanics during resistance training are responsible for measurement errors of IOP with standard contact tonometry. A steep rise in IOP during heavy lifting followed by equalization can damage the lamina cribrosa in patients with pre-existing risk factors.
Conclusions. Heavy resistance training is generally safe for the eyes but requires optimization of loading parameters, breathing patterns, and coordination of breathing movements with the Valsalva maneuver. Sports medicine professionals should develop personalized recommendations for their patients based on baseline corneal biomechanics, choroidal blood flow, and optic disc anatomy.

KEYWORDS
Resistance Training, Intraocular Pressure, Ocular Biomechanics, Valsalva Maneuver, Glaucoma

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Introduction

Recent declarations from international athletic bodies emphasize that evaluating visual system integrity and monitoring ocular mechanics under physiological stress should be standard components of comprehensive medical checks for elite athletes [1]. When managing competitive lifters, designing an optimal strength program requires a deep understanding of structural boundaries that extends far beyond muscle adaptations. Although high-resistance loading is widely recognized as a primary stimulus for eliciting maximum force production and neuromuscular adaptations [2], this modality subjects the eye's fluid compartments and soft-tissue structures to profound internal forces [3].

The extreme exertion necessary to overcome large external loads generates massive, temporary spikes in systemic vascular perfusion and pressures within major body cavities [2]. When an athlete contracts the abdominal wall muscles while attempting to exhale against a closed airway, a technique known as the Valsalva maneuver, the resulting internal pressure protects the spine by forming a highly rigid torso cylinder. Nevertheless, this significant increase in intrathoracic pressure alters normal hemodynamics by restricting venous blood flow returning to the heart. This resistance causes venous congestion, forcing a retrograde transmission of elevated pressure up through the jugular pathways into cranial vascular networks [5]. Concurrently, arterial pathways exhibit sharp spikes, with mean arterial pressure frequently exceeding 300 mmHg during the concentric phase of heavy squats or deadlifts [2].

The primary responsibility of ophthalmologists and sports physicians is to identify how these substantial cardiorespiratory spikes impact intraocular fluid balance, alter tissue elasticity, and modify the physical forces acting on the back of the eye [1]. Investigating how near-maximal lifting influences ocular biomechanics is

critical for establishing evidence-based safety guidelines and preventing long-term optic nerve complications in power sports [3].

Modern sports cardiology and exercise science increasingly highlight the distinction between static isometric straining and dynamic aerobic routines. While steady-state cardiovascular activities usually induce a mild decline or stabilization in intraocular pressure due to metabolic acidosis and improved venous outflow, high-load resistance training introduces opposite forces [6]. The sharp rise in intrathoracic pressure generated during heavy concentric lifting transmits rapidly through the valveless venous plexus of Batson, directly elevating cranial, epidural, and orbital venous pressures [16]. This backpressure severely limits passive fluid clearance from the eye, setting the stage for acute spikes in intraocular tension [4].

Understanding these intraocular changes requires examining the eye as an enclosed, viscoelastic structural system. The corneoscleral shell maintains structural shape while housing fluid-filled chambers and vascular beds. When internal blood volume or hydrostatic fluid pressures fluctuate rapidly, the outer collagen matrix of the eye stretches and deforms according to its specific compliance properties [9]. In powerlifting, where multi-set protocols to failure are routine, these acute physical deformities repeat regularly, raising critical questions regarding long-term structural remodeling of the anterior and posterior segments [3].

In addition to structural deformation, systemic vascular alterations significantly impact ocular tissue perfusion. Extreme spikes in mean arterial pressure (MAP) occurring alongside elevated intraocular pressure (IOP) create dynamic shifts in ocular perfusion pressure (OPP) [15]. While native autoregulatory mechanisms in healthy optic nerve microvessels attempt to maintain stable blood delivery, sudden pressure spikes during maximal lifting sets test the upper functional limits of vascular self-regulation [16]. For athletes with pre-existing vascular dysregulation, myopia, or early structural weakness of the optic disc, these acute hemodynamic surges may contribute to subtle, cumulative ischemic stress [12].

Despite growing interest in powerlifting, cross-training, and high-intensity body conditioning across diverse age demographics, clinical guidelines addressing ocular safety during heavy strength workouts remain limited. Traditional ophthalmic screenings rarely account for acute exercise-induced pressure surges, evaluating eye health exclusively under resting conditions [1]. Consequently, establishing a comprehensive theoretical framework that links physical exertion, thoracic pressure waves, corneal biomechanics, and optic disc strain is essential for protecting visual health without unnecessary restriction of athletic training [3].

A primary clinical concern is determining how acute ocular hypertension during heavy lifting translates into long-term biological risk. While transient IOP surges lasting several seconds may appear clinically harmless in healthy young athletes, repetitive exposure over years of competitive training could induce structural fatigue in vulnerable collagen structures [18]. The lack of standardized screening protocols for high-risk athletes—such as those with high myopia or a family history of primary open-angle glaucoma—leaves strength-training individuals vulnerable to undetected, progressive optic nerve damage [19].

Furthermore, standard clinical instruments utilized to measure IOP, such as Goldmann applanation tonometry, rely on static mathematical assumptions that do not reflect dynamic physiological states [11]. Exercise-induced systemic changes, including altered blood serum osmolarity, core body temperature elevations, and altered corneal hydration, directly modify corneal stiffness [10]. As a result, static resting measurements fail to reflect peak IOP levels occurring during maximal physical exertion, leading to potential underestimation of true intraocular pressure spikes experienced during intense training [4].

Finally, the complex interaction between breathing mechanics, lifting postures, and central nervous system feedback loops remains under-explored in routine sports medicine practice. Athletes frequently perform compulsory isometric bracing and glottic closure during heavy multi-joint lifts, unaware of how breath control modifies internal cranial fluid dynamics [22]. Resolving these diagnostic gaps requires integrating biomechanical engineering principles, clinical ophthalmology, and sports ergonomics to formulate clear, evidence-based recommendations for athletes and coaches.

The objective of this manuscript is to evaluate scientific data regarding the structural and biomechanical properties of the corneoscleral shell, focusing on its viscoelastic behavior, volumetric alterations in the anterior chamber, and posterior segment hemodynamic shifts during heavy straining. Specifically, this paper examines the mechanisms behind exercise-induced intraocular pressure (IOP) spikes, the confounding and misestimation of cornea-dependent IOP measurements during exertion, variations in the translaminar pressure gradient, and the risk of permanent damage to the optic nerve head in strength-training populations.

Methodology

To capture and critically evaluate the complex interplay between heavy resistance exercise, ocular biomechanics, intraocular fluid dynamics, and optic nerve strain, this review employed a multidisciplinary literature synthesis across clinical ophthalmology, sports medicine, vascular physiology, and ocular tissue engineering. The investigative timeline was strategically bounded between 2016 and 2026 to ensure that synthesized empirical evidence captures the present generation of high-precision dynamic tonometry, advanced optical coherence tomography (OCT) imaging, corneal biomechanical analyzers, and continuous wireless telemetric IOP monitoring. Early foundational publications were selectively retained only when they provided essential theoretical frameworks necessary for interpreting ocular viscoelasticity and vascular autoregulatory mechanics.

Search Strategy and Information Sources

Information retrieval was executed across major international databases: PubMed, Google Scholar, EMBASE, MEDLINE, and the Cochrane Database of Systematic Reviews. The electronic search strategy incorporated targeted, Boolean-linked keyword strings designed to capture the intersection of resistance training hemodynamics, intraocular pressure surges, and ocular structural mechanics, including:

- ("intraocular pressure" OR "IOP spikes" OR "ocular hypertension") AND ("resistance training" OR "weightlifting" OR "powerlifting")
- ("Valsalva maneuver" OR "intrathoracic pressure") AND ("choroidal thickness" OR "ocular blood flow" OR "retrobulbar hemodynamics")
- ("corneal biomechanics" OR "corneal hysteresis" OR "corneal resistance factor") AND ("exercise-induced serum shifts" OR "tonometric artifact")
- ("translaminar pressure gradient" OR "lamina cribrosa strain") AND ("cerebrospinal fluid pressure" OR "optic disc deformation")
- ("primary open-angle glaucoma" OR "high myopia") AND ("exercise safety guidelines" OR "telemetric contact lens monitoring")

Inclusion and Exclusion Criteria

- Retrieved articles were screened and deemed eligible for inclusion if they satisfied at least one of the following operational inclusion criteria:

- Evaluated shifts in intraocular pressure, aqueous humor clearance dynamics, or acute subfoveal choroidal thickness changes driven directly by resistance exercise or heavy isometric straining;
- Examined structural alterations, translaminar pressure gradient fluctuations, optic nerve head compression, or scleral extracellular matrix remodeling stemming from acute thoracic pressure spikes;
- Investigated functional characteristics of corneal biomechanical properties (corneal hysteresis, corneal resistance factor), tonometric correction formulas, or real-time continuous telemetric IOP monitoring in active lifters;
- Documented changes in systemic arterial blood pressure, mean arterial pressure, retrobulbar blood flow autoregulation, or ocular perfusion pressure during variable resistance intensities.

- Conversely, publications were systematically excluded if they met any of the following criteria:

- Did not directly address visual science, ocular tissue biomechanics, resistance exercise protocols, or sports ophthalmology guidelines;
- Evaluated static, non-straining physical activities (e.g., low-intensity steady-state walking or passive stretching) lacking Valsalva mechanics and high-load thoracic pressure generation;
- Consisted of commercial product manuals, non-refereed fitness blogs, popular media articles, or opinion-based commentary lacking structured empirical metrics;
- Manuscripts without available full English text.

Finally, investigations focusing solely on clinical medical rehabilitation populations (e.g., post-stroke gait training or post-cardiac surgery rehabilitation) were kept outside the scope of this review. The physiological profiles, intra-abdominal pressure dynamics, and lifting intensities of clinical rehabilitation populations lack the maximal strain characteristics, volume metrics, and Valsalva forces typical of healthy recreational and athletic strength-training populations.

Results

The primary homeostatic metric impacted by high-intensity weightlifting is the dynamic regulation of intraocular pressure (IOP) [3]. In healthy individuals at rest, aqueous humor hydrodynamics are strictly governed by Goldmann's steady-state model, wherein IOP is determined by the rate of ciliary body aqueous humor secretion, episcleral venous pressure (EVP), and trabecular outflow facility [6]. Under normal resting conditions, aqueous humor drains continuously through the conventional trabecular meshwork into Schlemm's canal and subsequently into the episcleral venous system. However, during heavy resistance exercise, this delicate clearance system experiences immediate mechanical and hemodynamic disruption. The forced expiration against a closed glottis characteristic of the Valsalva maneuver rapidly elevates intrathoracic and intra-abdominal pressures, compressing the superior and inferior vena cava. Because the venous system connecting the thoracic cavity to the head and orbit lacks anatomical valves, this centralized backpressure transmits retrogradely through the internal jugular veins and cavernous sinus into the superior ophthalmic vein and episcleral venous plexus [5, 7]. Episcleral venous pressure spikes from a baseline of 8–10 mmHg to values exceeding 30–35 mmHg, completely abolishing the hydrostatic gradient required for passive aqueous outflow through Schlemm's canal [6].

Concurrently, the posterior compartment of the eye undergoes rapid hemodynamic engorgement. The choroid—a highly vascular coat supplying the outer retina—contains an extensive network of fenestrated capillaries and larger blood vessels that lack autonomic autoregulatory sphincters [7]. During maximal forced exertion, retrograde venous backpressure forces a surge of systemic blood into the uveal tract, causing rapid expansion of subfoveal choroidal volume and an acute increase in choroidal thickness [8]. Because the surrounding outer sclera is semi-rigid and unyielding, this internal vascular engorgement creates an inward hydraulic displacement, compressing the vitreous chamber and pushing the anteriorly situated lens-iris diaphragm forward [9]. The combination of stalled trabecular outflow and acute intraocular volume expansion produces steep, immediate surges in IOP. Continuous tonometric evaluations indicate that near-maximal powerlifting sets provoke transient IOP peaks of 40–45 mmHg [4, 5]. Notably, these intraocular hypertensive surges collapse rapidly back toward baseline levels within milliseconds of glottis release and weight rack completion [4].

The magnitude and physiological strain of these acute intraocular pressure spikes are directly moderated by specific exercise variables:

Percentage of 1-Repetition Maximum (1RM): Exercise intensity serves as a primary determinant of IOP elevation. Submaximal workloads below 60–70% of 1RM provoke modest pressure increases primarily driven by systemic arterial elevation. In contrast, loading protocols exceeding 80–85% of 1RM require compulsory muscular bracing and maximum intra-abdominal pressure generation [5]. The massive intrathoracic pressure waves generated during heavy powerlifts (such as squats, deadlifts, and bench presses) generate significantly higher peak IOP surges than those recorded during lighter, high-repetition sets [2, 4]. The distinct biomechanical drivers, corneal impacts, and optic nerve strain characteristics across exercise modalities are systematically detailed in Table 1.

Table 1. Biomechanical Drivers and Optic Nerve Strain Across Different Exercise Modalities

Exercise Modality	Primary Vascular Driver	Impact on Corneal Damping	Peak Optic Nerve Strain
Maximal Powerlifts (Squats, Deadlifts)	Severe thoracic venous block; rapid choroidal blood pooling [2, 5, 7].	Acute drop in corneal hysteresis due to systemic serum shifts [10].	Extreme during the immediate post-lift weight release window [14].
High-Volume Sets (To Failure)	Sustained, lower-amplitude accumulation of head venous backpressure [5, 21].	Transient alterations in dynamic visual tracking and ocular surface stability [23].	Moderate but prolonged over extended set durations [4].
Postural Exercises (Decline Leg Press)	Combined impact of centralized chest pressure and gravitational fluid pooling [5, 8].	Stable baseline values; heavily dominated by high internal pressure [4].	Elevated and continuous throughout the entire exercise duration [8].
Isometric Holds (Heavy Bracing)	Low-clearance, steady-state internal ocular hypertension [4, 21].	Minor alterations and drying from blink suppression [10].	Low to moderate; balanced by sustained CSFP elevations [13].

Duration of Breath Holding and Glottic Mechanics: The temporal profile of breath retention correlates linearly with intraocular pressure accumulation [4]. Prolonged isometric breath-holding during multi-second concentric stick points sustains jugular venous congestion, stalling retrobulbar blood flow and prolonging acute intraocular hypertension [6, 7]. Conversely, maintaining a continuous expiratory flow during heavy exertion blunts peak thoracic pressure waves, significantly reducing the magnitude of acute IOP spikes [22].

Accurately measuring intraocular pressure during heavy exercise presents significant clinical and diagnostic challenges due to the dynamic viscoelastic behavior of the central cornea [10]. The human cornea is a highly organized collagenous tissue exhibiting distinct biomechanical parameters, primarily corneal hysteresis (CH)—a metric reflecting tissue viscoelastic damping and energy dissipation—and corneal resistance factor (CRF), which quantifies total structural resistance [10]. High-intensity strength training provokes rapid systemic physiological shifts, including elevated core temperature, heavy fluid loss via perspiration, altered serum osmolarity, and temporary corneal stromal dehydration [10, 11, 22]. Stromal fluid loss alters inter-collagen fibrillar spacing, transiently increasing corneal rigidity while reducing its native viscoelastic damping capacity (CH) [11]. Standard applanation tonometry instruments, such as Goldmann tonometers or electronic rebound pens, rely on the Imbert-Fick principle, which assumes a thin, perfectly flexible, and purely elastic corneal membrane of uniform thickness [3]. When applied to an acutely stiffened, moisture-depleted cornea during physical exertion, these devices encounter increased structural resistance during mechanical applanation. Consequently, contact tonometers over-read the true fluid pressure within the anterior chamber, introducing substantial clinical measurement artifacts that complicate true IOP estimation [11].

However, while anterior corneal artifacts obscure precise clinical measurements, the true pathogenic threat of exercise-induced ocular hypertension manifests deeper within the globe [10, 12]. The rapid accumulation of intraocular fluid pressure directly translates into mechanical stress that propagates retrogradely toward posterior ocular structures [9, 12]. Consequently, this sudden dynamic hydrostatic force shifts the primary site of structural vulnerability from the dynamic outer cornea to the unyielding tissues of the posterior pole [9, 12]. This biomechanical shift concentrates compressive strain directly onto the optic nerve head [12].

At the posterior segment, acute ocular hypertension exerts severe physical forces on the optic nerve head, specifically at the lamina cribrosa—a delicate, perforated collagenous meshwork spanning the scleral canal that transmits unmyelinated retinal ganglion cell (RGC) axons [12]. The mechanical load acting across the lamina cribrosa is governed by the translaminar pressure gradient (TLPG), defined as the hydrostatic difference between internal intraocular pressure and surrounding retrobulbar subarachnoid cerebrospinal fluid pressure (CSFP) [12]. During active straining under a heavy lift, thoracic pressure waves elevate CSFP and IOP simultaneously [13]. The concurrent rise in CSFP provides a protective hydrostatic counter-pressure within the optic nerve sheath, limiting structural displacement of the lamina cribrosa during peak loading [12].

However, a critical mechanical vulnerability occurs during the immediate post-lift recovery window [14]. Following weight release and sudden glottis opening, CSFP drops back to baseline levels almost instantaneously alongside thoracic depressurization [13]. In contrast, IOP clearance is significantly delayed because preceding venous congestion has compromised active trabecular meshwork drainage pathways [4]. This imbalance creates a brief window of marked TLPG enlargement, characterized by elevated internal IOP acting against a un-supported retrobulbar space [14]. The resulting pressure differential forces the lamina cribrosa to bow posteriorly, generating shear strain on passing RGC axons and compressing capillary networks within the prelaminar optic disc [15].

Maintaining adequate metabolic supply to optic nerve tissues during these extreme mechanical states depends on stable mean ocular perfusion pressure (MOPP), calculated using the standard hemodynamic equation:

During maximal powerlifts, systemic mean arterial pressure (MAP) can exceed 300 mmHg [2], driving a massive increase in calculated perfusion pressure that tests retinal vascular autoregulation [15, 16]. While healthy retinal microvessels dynamically constrict to protect capillary beds from hyperperfusion damage, sudden drops in systemic pressure post-lift—occurring alongside persistently high IOP—can induce transient ocular hypoperfusion [15]. In athletes with pre-existing structural weakness, thinned scleral canal architecture, or altered scleral cross-linking (such as individuals with high axial myopia [9]), repetitive exposure to high TLPG fluctuations accelerates structural breakdown at the posterior pole [12].

At the cellular level, repeated mechanical strain activates lamina cribrosa astrocytes and resident optic nerve head microglia via mechanosensitive cation channels, including Piezo1 and TRPV4 [17]. Mechanotransduction pathways trigger upregulation of matrix metalloproteinases (MMP-2 and MMP-9), accelerating collagen turnover and remodeling healthy extracellular matrix scaffolding into a stiffened, fibrotic tissue network [18]. Over extended training careers, this structural stiffening reduces the optic disc's ability to cushion transient pressure changes, elevating long-term vulnerability to axonal transport blockage, optic neuropathy, and primary open-angle glaucoma (POAG) [12, 19].

Discussion

Findings of this systematic review confirm that high-intensity resistance training exerts profound, transient biomechanical and vascular influences on ocular physiology. While steady-state aerobic activities typically induce a mild reduction or stabilization in intraocular pressure (IOP) mediated by systemic metabolic acidosis, lactate accumulation, and enhanced trabecular clearance [6], heavy resistance exercise provokes acute intraocular hypertensive spikes [3, 4]. The core physiological driver of these acute surges is the forced Valsalva maneuver performed during maximal concentric lifting attempts [5]. By elevating intrathoracic and intra-abdominal pressures to reinforce spine stability under heavy external loads [2], the lifter creates a centralized vascular blockage that directly impedes venous return to the right atrium. Because the cranial, jugular, and orbital venous networks lack anatomical valves, this centralized backpressure is transmitted directly retrogradely into the episcleral venous plexus and the choroidal vasculature [5, 7].

Under steady-state resting conditions, intraocular pressure is strictly governed by the classic Goldmann equation, wherein equilibrium IOP is determined by the balance between aqueous humor secretion by the ciliary body epithelium, trabecular outflow facility, episcleral venous pressure (EVP), and non-trabecular uveoscleral drainage [6]. During maximal resistance exercise, this equilibrium is disrupted simultaneously across multiple points of the pathway. Acute jugular backpressure elevates EVP from resting baselines of 8–10 mmHg to transient values exceeding 30–35 mmHg [5, 6]. Because passive aqueous humor outflow through Schlemm's canal depends entirely on a favorable hydrostatic pressure gradient between the anterior chamber and episcleral veins, a sharp rise in EVP effectively halts conventional trabecular drainage [6]. At the same time, the posterior segment experiences rapid blood pooling within the valve-free choroidal capillary network, causing subfoveal choroidal thickness to expand rapidly under peak strain [7, 8]. This acute volume expansion within the posterior eye exerts an internal hydraulic push, displacing the vitreous humor forward and compressing internal anterior chamber geometry against the semi-rigid outer scleral coat [9]. Together, blocked fluid clearance and acute intraocular vascular engorgement produce transient IOP spikes reaching 40–45 mmHg during heavy 1RM powerlifts [4, 5].

A central question in exercise ophthalmology is whether these transient, second-scale intraocular hypertensive surges pose a true clinical threat to ocular structural integrity. In healthy, young individuals with normal corneoscleral viscoelasticity, the eye possesses inherent buffering capacities [9, 10]. The sclera acts as a dynamic biomechanical shock absorber, expanding slightly under transient internal pressure elevations to dissipate mechanical strain [9]. However, repetitive exposure to high-amplitude pressure spikes over years of competitive strength training can alter this baseline structural buffering [18]. Cyclic mechanical stretching of human scleral fibroblasts upregulates the expression of pro-fibrotic cytokines, stimulating collagen cross-linking and extracellular matrix remodeling [18]. Over time, this progressive scleral stiffening reduces scleral compliance, diminishing the globe's capacity to buffer future intraocular pressure surges and transferring a higher proportion of internal hydrostatic strain directly onto vulnerable optic nerve head tissues [12, 18].

The biomechanical vulnerability of the posterior pole is fundamentally governed by the translaminar pressure gradient (TLPG)—the hydrostatic pressure differential across the lamina cribrosa between internal IOP and retrobulbar cerebrospinal fluid pressure (CSFP) [12]. During the active straining phase of a heavy lift, thoracic pressure waves elevate both CSFP and IOP concurrently [13]. The simultaneous rise in CSFP provides a protective hydrostatic cushion within the optic nerve sheath, supporting the posterior surface of the lamina cribrosa and limiting its structural displacement during peak loading [12, 13]. However, a critical mechanical vulnerability opens during the immediate post-lift recovery transition [14]. When the athlete releases the barbell, opens the glottis, and gasps for air, thoracic depressurization causes CSFP to collapse back to baseline levels almost instantaneously [13]. In contrast, IOP clearance displays a delayed decay curve because preceding venous backpressure has temporarily stalled active trabecular outflow [4, 6]. This temporal mismatch generates a transient window of marked TLPG enlargement, wherein elevated internal IOP acts

against a depressurized retrobulbar subarachnoid space [14]. The resulting unbalanced pressure gradient forces the lamina cribrosa to displace posteriorly, generating shear strain across exiting unmyelinated retinal ganglion cell (RGC) axons and compressing capillary networks within the prelaminar optic disc [15].

Beyond extracellular matrix remodeling and macro-vascular perfusion shifts, the cellular metabolic impact of transient intraocular pressure surges on retinal ganglion cell (RGC) axons introduces a profound neuro-ophthalmic risk vector during heavy resistance exertion. Unmyelinated RGC axons passing through the narrow pores of the lamina cribrosa exhibit an extraordinarily high density of mitochondria to sustain the energy-intensive sodium-potassium ATPase pump activity required for continuous action potential propagation. When an acute thoracic pressure spike during a maximal lift elevates intraocular pressure to 40–45 mmHg while simultaneously swelling subfoveal choroidal thickness, the resulting biomechanical shear forces pinch these unmyelinated axonal bundles against the stiffened collagenous laminar beams [12]. This mechanical compression creates an acute physical bottleneck that directly impairs both anterograde and retrograde axoplasmic transport [14]. Crucially, anterograde transport delivers essential neurotrophic factors—such as brain-derived neurotrophic factor (BDNF) and nerve growth factor (NGF)—from the retina to the superior colliculus and lateral geniculate nucleus, while retrograde transport recycles cellular waste and signaling endosomes back to the cell soma. Interruption of this vital axoplasmic flow, even for transient multi-second intervals during a sticking-point concentric effort, leads to localized accumulation of mitochondria, organelle congestion, and focal axonal swelling within the unmyelinated prelaminar optic nerve head [15]. Furthermore, this localized axonal pinch disrupts mitochondrial respiratory chain efficiency, provoking excessive production of reactive oxygen species (ROS) and inducing micro-environmental oxidative stress. Concurrently, mechanosensitive activation of optic nerve head astrocytes triggers an immediate release of intracellular calcium stores, which spreads via gap junctions to neighboring glial networks, amplifying neuro-inflammatory cytokine production (such as TNF-alpha and IL-1 beta) [17]. In healthy, well-conditioned athletes with robust baseline antioxidant capacity and optimal microvascular autoregulation, these transient metabolic disruptions are rapidly cleared during post-lift rest intervals through endogenous neuroprotective mechanisms [15]. However, in lifters exhibiting silent mitochondrial dysfunction, low baseline systemic antioxidant capacity, or genetic susceptibility to optic nerve head degeneration, the cumulative impact of thousands of repetitive axoplasmic transport interruptions across years of high-volume powerlifting can lower the threshold for RGC apoptosis [12]. Over time, this chronic micro-trauma converts transient bioenergetic distress into irreversible structural thinning of the retinal nerve fiber layer (RNFL), establishing a novel physiological pathway wherein un-mitigated high-load isometric straining acts as an environmental accelerant for subclinical neuro-degenerative damage at the posterior pole [14, 17].

From an diagnostic and ergonomic perspective, accurately evaluating IOP during heavy exercise is further complicated by corneal biomechanical artifacts [10]. Central corneal thickness (CCT), corneal hysteresis (CH), and corneal resistance factor (CRF) directly influence applanation tonometry measurements [10,11]. Intense physical exertion induces profuse sweating, serum hyperosmolarity, elevated facial cutaneous temperature, and mild corneal stromal dehydration [10, 22]. Stromal hydration loss increases temporary corneal stiffness while reducing its native viscoelastic energy dissipation capacity (CH) [11]. Standard Goldmann applanation tonometry (GAT) and electronic rebound devices rely on static mathematical assumptions derived from the Imbert-Fick law, which assumes a perfectly flexible, non-yielding membrane [3]. When applied to an acutely stiffened, moisture-depleted cornea during physical effort, contact tonometers encounter increased structural resistance during applanation, producing an artificially inflated digital pressure reading [11]. Consequently, static resting tonometry routinely fails to capture an athlete's true intraocular fluid status during live training, emphasizing the need for advanced dynamic biomechanical measurements [10, 20].

Individual risk stratification must account for pre-existing ocular pathologies and anatomical variations. Athletes with high axial myopia (> −6.00 diopters or axial length > 26.0 mm) represent a particularly vulnerable subgroup [9]. Highly myopic eyes exhibit generalized scleral thinning, elongated optic canal geometry, and reduced baseline scleral rigidity [9]. When exposed to acute IOP surges and TLPG enlargement during heavy lifting, myopic eyes experience higher structural strain across the lamina cribrosa than emmetropic controls, increasing risk for scleral staphyloma expansion, chorioretinal stretch, and optic disc damage [9, 12]. Similarly, individuals with a genetic predisposition to POAG, thinned baseline corneal hysteresis, or pre-existing glaucoma should be managed with heightened clinical vigilance when participating in high-intensity strength disciplines [19].

Mitigating these vision-threatening risks does not require prohibiting resistance training, which remains a cornerstone of neuromuscular conditioning, bone mineral density maintenance, and overall athletic health [2].

Instead, clinical management should focus on optimizing exercise execution mechanics and breathing protocols [22]. Substituting prolonged isometric breath-holding with continuous expiratory techniques (such as forced exhalation during the sticking point of a concentric lift) prevents extreme intrathoracic pressure accumulation, allowing uninterrupted jugular venous drainage and blunting peak IOP spikes [4, 22]. Additionally, lifting posture optimization plays a key role: avoiding prone, inverted, or head-down decline positions (e.g., decline leg press or inverted hyperextensions) eliminates gravitational hydrostatic vectors that exacerbate choroidal venous engorgement [8, 21]. Finally, incorporating structured inter-set recovery periods allows complete clearance of jugular venous congestion and restores normal aqueous humor drainage prior to subsequent lifting bouts [3, 6].

Advanced diagnostic technologies offer promising avenues for real-time risk assessment in power sports. Continuous telemetric monitoring using sensor-equipped soft contact lenses enables non-invasive, wireless recording of real-time corneal curvature and ocular circumference alterations during live strength sets [20]. Coupled with regular spectral-domain optical coherence tomography (SD-OCT) evaluations of lamina cribrosa depth, nerve fiber layer thickness, and baseline corneal hysteresis profiling [10, 12], sports medicine specialists can construct personalized risk matrices for elite lifters. Implementing these evidence-based clinical safeguards ensures that athletes can maximize muscular strength performance while protecting long-term visual health [1, 3].

As structured systematically in Table 2, mitigating these visual risks requires specific behavioral adaptations and clinical monitoring strategies.

Table 2. Summary of Practical Clinical Guidelines and Behavioral Prioritization for Athletes

Clinical Guideline / Recommendation	Scientific & Physiological Rationale
Continuous Expiratory Techniques	Eliminates prolonged isometric breath-holding; keeps peak thoracic pressures low to allow uninterrupted jugular venous return [4, 22].
Lifting Posture Optimization	Avoids compressed, prone, or declined orientations that add hydrostatic gravity vectors, minimizing severe choroidal engorgement [8].
Baseline Biomechanical Screening	Assesses central corneal thickness and corneal hysteresis to identify pre-existing systemic measurement errors [10, 11].
Optic Nerve Topography Monitoring	Utilizes regular optical coherence tomography (OCT) scans to map changes in the lamina cribrosa and optic nerve head [12, 14, 21].
High-Risk Stratification	Screens for high axial myopia [9] and genetic primary open-angle glaucoma (POAG) traits [19] to protect vulnerable posterior pole structures [12].
Advanced Wearable Monitoring	Integrates sensor-equipped soft contact lenses to capture real-time wireless corneal curvature changes during live training [20].
Structured Inter-Set Recovery	Facilitates proper aqueous humor drainage clearance and stabilizes ocular perfusion pressure between heavy lifting bouts [3, 6].

Conclusion

Heavy resistance training provides an extremely effective avenue for building physical strength and neuromuscular performance [2], but it can expose the internal structures of the eye to rapid, profound physiological transitions [3]. The temporary surges of intraocular pressure observed during heavy lifting are primarily driven by the execution of the Valsalva maneuver [4], which results in acute cranial venous congestion and rapid choroidal vascular expansion against the semi-rigid scleral coat [7, 9]. The eye's ability to safely buffer these transient pressure loads is significantly dictated by its underlying viscoelastic properties, including corneal hysteresis and scleral compliance [10, 18].

To preserve long-term visual fields without compromising athletic gains, strength athletes should avoid prolonged, forced breath-holding [4], optimize their lifting postures [8], and undergo comprehensive, sport-specific ophthalmic screenings [12]. Innovative wearable sensors, such as soft silicone-hydrogel contact lenses

incorporating built-in micro-sensors, show immense clinical promise by transmitting real-time corneal curvature data wirelessly to capture the exact millisecond peak of eye pressure during a lift [20].

Ultimately, the successful management of a strength athlete requires a unified, interdisciplinary approach where the athlete's distinct ocular anatomy, systemic blood pressure profiles, breathing mechanisms, and specific sporting demands are systematically optimized together [1, 3].

REFERENCES

1. Moe, M. C., Özmert, E., Baudouin, C., Binadra, A., Crafoord, S., Jo, Y., Kiratli, H., Moore, M., Pitsiladis, Y. P., Rolle, U., Tan, B., Yanik, Ö., Budgett, R., Erdener, U., Steffen, K., & Engbretsen, L. (2023). International Olympic Committee (IOC) consensus paper on sports-related ophthalmology issues in elite sports. *BMJ Open Sport & Exercise Medicine, 9*(3), Article e001644. https://doi.org/10.1136/bmjsem-2023-001644
2. de Vos, N. J., Singh, N. A., Ross, D. A., Stavrinos, T. M., Orr, R., & Fiatarone Singh, M. A. (2008). Continuous hemodynamic response to maximal dynamic strength testing in older adults. *Archives of Physical Medicine and Rehabilitation, 89*(2), 343–350. https://doi.org/10.1016/j.apmr.2007.08.130
3. Hackett, D. A., Li, J., Wang, B., Way, K. L., Cross, T., & Tran, D. L. (2024). Acute effects of resistance exercise on intraocular pressure in healthy adults: A systematic review. *Journal of Strength and Conditioning Research, 38*(2), 394–404. https://doi.org/10.1519/JSC.0000000000004668
4. Vaghefi, E., Shon, C., Reading, S., Sutherland, T., Borges, V., Phillips, G., Niederer, R. L., & Danesh-Meyer, H. (2021). Intraocular pressure fluctuation during resistance exercise. *BMJ Open Ophthalmology, 6*(1), Article e000723. https://doi.org/10.1136/bmjophth-2021-000723
5. Vera, J., Jiménez, R., Redondo, B., Torrejón, A., De Moraes, C. G., & García-Ramos, A. (2019). Effect of the level of effort during resistance training on intraocular pressure. *European Journal of Sport Science, 19*(3), 394–401. https://doi.org/10.1080/17461391.2018.1505959
6. Jakubowska, M., Dziegciarczyk, A., Włodarczyk, A., Wiater, Z., Brankowska, K., Swoboda, K., Domagała, S., Olejnik, M., Kądziołka, W., & Górka, P. A. (2026). Physical activity as a modulator of intraocular pressure: Relevance for sport participation and eye health. *Quality in Sport, 53*, Article 69807. https://doi.org/10.12775/QS.2026.53.69807
7. Arnold-Vangsted, A., Boberg-Ans, L. C., Cehofski, L. J., van Dijk, E. H. C., Grauslund, J., Hansen, M. S., Kiilgaard, H. C., Klefter, O. N., Krogh Nielsen, M., Sevik, M. O., & Subhi, Y. (2025). Impact of the Valsalva manoeuvre on the choroid: A systematic review with meta-analyses. *Acta Ophthalmologica, 103*(2), 127–135. https://doi.org/10.1111/aos.16793
8. Sayin, N., Kara, N., Pekel, G., & Altinkaynak, H. (2015). Choroidal thickness changes after dynamic exercise as measured by spectral-domain optical coherence tomography. *Indian Journal of Ophthalmology, 63*(5), 445–450. https://doi.org/10.4103/0301-4738.159884
9. Jia, X., Yu, J., Liao, S. H., & Duan, X. C. (2016). Biomechanics of the sclera and effects on intraocular pressure. *International Journal of Ophthalmology, 9*(12), 1824–1831. https://doi.org/10.18240/ijo.2016.12.21
10. Antończyk, A., Kubiak-Nowak, D., Borawski, W., Gloster, A., Janeczek, M., & Pasławska, U. (2020). The effect of changes in cardiovascular activity on corneal biomechanics and pulsation in rabbits. *Scientific Reports, 10*(1), Article 22249. https://doi.org/10.1038/s41598-020-79219-9
11. Palamar, M., Dag, M. Y., & Yagci, A. (2015). The effects of Valsalva manoeuvre on Ocular Response Analyzer measurements. *Clinical and Experimental Optometry, 98*(5), 447–450. https://doi.org/10.1111/cxo.12303
12. Siaudvytyte, L. (2019). Translaminar pressure gradient. In I. Januleviciene & A. Harris (Eds.), Biophysical properties in glaucoma (pp. 67–82). Springer, Cham. https://doi.org/10.1007/978-3-319-98198-7_5
13. Liu, S. M., Wang, N. L., Zuo, Z. T., Chen, W. W., Yang, D. Y., Li, Z., & Cao, Y. W. (2018). The effect of increased intra-abdominal pressure on orbital subarachnoid space width and intraocular pressure. *Neural Regeneration Research, 13*(2), 353–359. https://doi.org/10.4103/1673-5374.226407
14. Zhang, J., Liu, Y., Liu, L., Li, L., & Qian, X. (2022). Morphological changes of glial lamina cribrosa of rats suffering from chronic high intraocular pressure. *Bioengineering, 9*(12), Article 741. https://doi.org/10.3390/bioengineering9120741
15. Boltz, A., Told, R., Napora, K. J., Palkovits, S., Werkmeister, R. M., Schmidl, D., Popa-Cherecheanu, A., Garhöfer, G., & Schmetterer, L. (2013). Optic nerve head blood flow autoregulation during changes in arterial blood pressure in healthy young subjects. *PLoS ONE, 8*(12), Article e82351. https://doi.org/10.1371/journal.pone.0082351
16. Piórkowska, I., Piotrowski, K., Jerczak, M., Pluszyński, T., Padula, N., Polewka, M., Polańska, A., Paczosa, K., & Przybył, F. (2026). The impact of high-intensity resistance training on intraocular pressure and ocular hemodynamics: A literature review. *Quality in Sport, 54*, Article 70413. https://doi.org/10.12775/QS.2026.54.70413
17. Garcia-Sanchez, J., Lin, D., & Liu, W. W. (2024). Mechanosensitive ion channels in glaucoma pathophysiology. *Vision Research, 223*, Article 108473. https://doi.org/10.1016/j.visres.2024.108473

18. Qiu, C., Chen, M., Yao, J., Sun, X., Xu, J., Zhang, R., Wang, X., Li, G., & Qian, S. (2018). Mechanical strain induces distinct human scleral fibroblast lineages: Differential roles in cell proliferation, apoptosis, migration, and differentiation. *Investigative Ophthalmology & Visual Science, 59*(6), 2401–2410. https://doi.org/10.1167/iovs.18-23855
19. Schuhmann, V., Küttel, J., Van Eijgen, J., Gugleta, K., & Hanssen, H. (2026). Physical activity and exercise in primary open-angle glaucoma: A scoping review. *Frontiers in Physiology, 17*, Article 1834649. https://doi.org/10.3389/fphys.2026.1834649
20. Pennisi, V., van den Bosch, J. J. O. N., Neustaeter, A., Ehmer, A., Thieme, H., Hoffmann, M. B., & Choritz, L. (2025). Assessment of the effect of physical activity on intraocular pressure using a novel implanted telemetric pressure sensor. *British Journal of Ophthalmology, 109*(10), 1138–1143. https://doi.org/10.1136/bjo-2023-324034
21. Krobot Cutura, N., Mrak, M., Cutura, D. M., Petric Vickovic, I., & Ruzic, L. (2024). Evaluating intraocular pressure alterations during large muscle group isometric exercises with varying head and body positions. *International Journal of Environmental Research and Public Health, 21*(4), Article 476. https://doi.org/10.3390/ijerph21040476
22. Vera, J., Perez-Castilla, A., Redondo, B., De La Cruz, J. C., Jiménez, R., & García-Ramos, A. (2020). Influence of the breathing pattern during resistance training on intraocular pressure. *European Journal of Sport Science, 20*(2), 157–165. https://doi.org/10.1080/17461391.2019.1617354
23. Liu, Z., Cui, L., Lu, X., Zhao, S., & Dong, Y. (2026). Dynamic changes in cortical activation patterns during incremental load among athletes of different sports types. *Brain and Behavior, 16*(7), Article e71631. https://doi.org/10.1002/brb3.71631