

Intraocular Pressure Changes During Prolonged Dry Static Apnea in Breath-Hold Divers

Pavla Potěšilová

Lensi Turnov s.r.o

Zuzana Schreiberová

University Hospital Olomouc

Martin Šín

Palacký University Olomouc

Marta Karhanová

`marta.karhanova@fnol.cz`

Palacký University Olomouc

Research Article

Keywords: apnea, breath-hold diving, episcleral venous pressure, glaucoma, intraocular pressure

Posted Date: October 19th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10989210/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Purpose

To evaluate dynamic changes in intraocular pressure (IOP) during prolonged dry static apnea (STA) in breath-hold divers and investigate the potential mechanisms underlying these changes.

Methods

This prospective observational study included 40 breath-hold divers (8 women and 32 men). IOP, peripheral oxygen saturation (SpO₂), heart rate (HR), and blood pressure (BP) were repeatedly measured at baseline, during apnea, and during recovery.

Results

Participants performed breath-holds lasting 3.9 ± 1.0 minutes (1:45–5:38). IOP increased significantly during STA ($P < 0.0001$), rising from 16.4 ± 2.0 mmHg at baseline to 29.0 ± 5.8 mmHg (18.6–43.8) at maximal apnea (mean increase, 12.6 ± 4.9 mmHg), and returned to baseline within 60 seconds after resumption of breathing. SpO₂ decreased significantly ($P < 0.0001$), reaching $85.9 \pm 10.1\%$ (58–98), and normalized within 60 seconds. HR did not change significantly during apnea ($P = 0.226$) but remained elevated during recovery ($P = 0.021$). BP increased significantly ($P < 0.0001$), with mean systolic and diastolic BP rising to 170 ± 21 mmHg (138–223) and 111 ± 13 mmHg (90–145), respectively, at maximal apnea. Changes in IOP correlated positively with systolic BP and negatively with SpO₂.

Conclusion

Dry static apnea induces a significant increase in intraocular pressure in breath-hold divers, followed by rapid normalization after apnea. The mechanism underlying this response is likely multifactorial, with increased episcleral venous pressure as a possible major contributing mechanism.

KEY POINTS

- Prolonged dry static apnea causes a marked, time-dependent increase in intraocular pressure.
- Intraocular pressure returns rapidly to baseline after resumption of breathing.
- The response is likely multifactorial, with increased episcleral venous pressure as a possible major contributing mechanism.

INTRODUCTION

The effect of breathing techniques on intraocular pressure (IOP) has been widely investigated. However, results remain inconsistent. While some studies reported no significant short-term effect of breathing exercises on IOP [1], others demonstrated either a decrease [2] or an increase [3].

In contrast, long-term effects of breathing techniques appear to be more consistent, with several studies suggesting a potential reduction in IOP [4,5].

An important question remains whether and how IOP changes during breath-holding. Previous studies suggest that simple breath-holding during IOP measurement does not significantly affect IOP [6,7], even after deep inspiration [6]. However, breath-holding during measurement is often accompanied by a Valsalva maneuver, which may lead to a transient increase in IOP [8]. Similarly, studies investigating breathing patterns during resistance training have shown that IOP increases more markedly when a Valsalva maneuver is performed in combination with breath-holding [9,10].

Physiological IOP values range between 10 and 21 mmHg [11–13], and elevated IOP is one of the most important risk factors for glaucoma [12,14–16], one of the leading causes of blindness worldwide [15,17,18].

To the best of our knowledge, no previous study has investigated how IOP changes during prolonged breath-holding. Breath-hold divers (BHDs) are able to sustain apnea for extended periods, with some individuals exceeding 10 minutes [19]. Such prolonged apnea is accompanied by complex cardiovascular and respiratory responses [20,21].

Although breath-hold diving has been practiced for centuries, particularly by Japanese Ama and Korean Haenyeo divers, its popularity has increased substantially in recent decades [22]. The only study evaluating the relationship between breath-hold diving and glaucoma reported slightly higher IOP values in Haenyeo divers compared with controls; however, the difference was minimal (approximately 1 mmHg) and remained within the normal range. No significant differences in visual field parameters were observed despite a higher cup-to-disc ratio in divers [23].

In this context, dry static apnea (STA), defined as breath-holding performed at rest on land, represents a suitable and controlled model for investigating ocular responses to prolonged breath-holding.

The aim of this study was to evaluate changes in IOP during dry STA in BHDs, explore their potential physiological mechanisms, and assess their possible clinical implications.

MATERIALS AND METHODS

Participants

This prospective observational study included 40 BHDs (8 women and 32 men), with a mean age of 38 ± 7.7 years (24–60 years). One eye per participant was included in the analysis, comprising 22 right eyes

(55%) and 18 left eyes (45%). Participants were engaged in freediving at either a professional or recreational level.

Exclusion criteria included a history of glaucoma or ocular hypertension, prior ocular trauma, refractive error greater than ± 5.00 dioptres, previous ocular surgery (cataract surgery, strabismus surgery, or pars plana vitrectomy), corneal disease, and acute ocular infection. Refractive surgery was not considered an exclusion criterion because absolute changes in IOP were evaluated. Contact lens use was not permitted for at least 24 hours before measurements. All medical history data were obtained using a questionnaire.

Ethical Approval

The study protocol was approved by the Ethics Committee of University Hospital Olomouc and the Faculty of Medicine and Dentistry, Palacký University Olomouc (No. 103/26; approved April 13, 2026). Written informed consent was obtained from all participants, and the study was conducted in accordance with the Declaration of Helsinki. The study was registered at ClinicalTrials.gov (NCT07604428).

Experimental Protocol

All measurements were performed by the same investigator. Before measurements, each participant performed breathing exercises and thoracic stretching according to their usual routine. Participants were then placed in a supine position and remained at rest for at least 2 minutes. Hyperventilation before breath-holding was not allowed. The use of a nose clip and techniques aimed at increasing lung volume, including glossopharyngeal insufflation (GI), were permitted.

Measurements

Baseline IOP and vital parameters, including peripheral oxygen saturation (SpO_2), heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP), were recorded.

IOP was measured using a portable rebound tonometer (Icare Pro; Icare Finland Oy, Helsinki, Finland) in the supine position. Participants were instructed to fixate straight ahead. During STA, repeated IOP measurements were obtained. IOP was measured in both eyes, and the eye measured second was included in the analysis.

SpO_2 and HR were continuously monitored using a pulse oximeter (Beurer PO 80; Beurer Healthcare, Germany). The sensor was placed on the right index finger, and recording was initiated at the onset of apnea.

Blood pressure (BP) was measured using an automated sphygmomanometer (Omron M6 Comfort; Omron Healthcare, Kyoto, Japan) placed on the left arm. The last successfully recorded SBP and DBP

values during apnea were used as the values at maximal apnea, even if they were not obtained immediately before the end of apnea.

Mean arterial pressure (MAP) was calculated from SBP and DBP using the formula:

$$\text{MAP} = \text{DBP} + 1/3 (\text{SBP} - \text{DBP}).$$

Data Analysis

As measurements could not be obtained simultaneously, data were grouped into predefined time intervals for statistical analysis. Measurements at baseline and maximal apnea were designated as T0 and Tmax, respectively.

Time intervals during STA were categorized as follows: 1–2 minutes (T1–2), 2–3 minutes (T2–3), 3–4 minutes (T3–4), and 4–5 minutes (T4–5). The 0–1 minute interval and time points beyond 5 minutes were excluded from the interval-based analysis because of insufficient measurements but retained in the overall analysis. Recovery measurements were obtained at 30 and 60 seconds (R30s and R60s), with BP measured at 60 and 120 seconds (R60s and R120s).

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version 23 (IBM Corp., Armonk, NY, USA). Data normality was assessed using the Shapiro–Wilk test. Changes over time during STA were evaluated using regression analysis. Recovery values (30 and 60 seconds after apnea; BP: 1 and 2 minutes) were compared with baseline using the paired t-test or the Wilcoxon signed-rank test, as appropriate. Associations between measured parameters were evaluated using Spearman correlation analysis. $P < 0.05$ was considered statistically significant. Continuous variables were presented as mean $\pm$ standard deviation or median (range), as appropriate.

Changes over time were illustrated using line graphs showing mean $\pm$ standard deviation. If multiple measurements were obtained within the same predefined time interval for a single participant, their mean value was used for statistical analysis and data visualization. IOP changes over time were additionally evaluated by linear regression using all individual IOP measurements and their original measurement times.

RESULTS

Participant Characteristics

Mean baseline IOP was 16.4 ± 2.0 mmHg (12.8–21.2), HR 69 ± 11 beats/min (50–98), SBP 126 ± 15 mmHg (97–186), DBP 78 ± 9 mmHg (59–101), and MAP 94 ± 10 mmHg (72–129). SpO₂ was 99% in all participants. The mean duration of maximal STA was 3.9 ± 1.0 minutes (1:45–5:38) (Fig. 1A). The mean freediving experience was 4.3 ± 3.2 years (0.5–15.0 years) (Fig. 1B).

Intraocular Pressure

IOP increased significantly during STA ($P < 0.0001$). The median increase in IOP per minute was 3.1 mmHg (1.2–6.3) (Fig. 2). The mean IOP at T_{max} was 29.0 ± 5.8 mmHg (18.6–43.8), corresponding to a mean increase of 12.6 ± 4.9 mmHg (3.5–25.1) from baseline. Linear regression analysis demonstrated a significant positive relationship between IOP and time ($r = 0.733$), indicating a progressive increase in IOP over time (Fig. 3). Although IOP remained significantly elevated at 30 seconds after apnea ($P < 0.0001$), it decreased rapidly, with a mean difference of 2.8 mmHg relative to baseline. IOP returned to baseline levels in all participants within 60 seconds after apnea.

Peripheral Oxygen Saturation

SpO₂ decreased significantly during STA ($P < 0.0001$). The median decrease in SpO₂ per minute was 3.0% (–9.1 to –0.3) (Fig. 4A). The mean SpO₂ at T_{max} was $85.9 \pm 10.1\%$ (58–98). SpO₂ remained significantly lower at 30 seconds after apnea compared with baseline ($P < 0.0001$), with a median difference of 4.5%. In 11 participants, SpO₂ continued to decrease after apnea, and in 5 of them, this decrease persisted for an additional 30 seconds. SpO₂ returned to baseline levels in all participants within 60 seconds after apnea.

Heart Rate

HR did not change significantly during STA ($P = 0.226$). The median change in HR per minute was –0.7 beats/min (–15.1 to 13.0) (Fig. 4B). A transient HR increase occurred immediately after apnea onset ($P < 0.0001$), with a median HR of 90 beats/min (51–140) at 1 second. The mean HR at T_{max} was 62 ± 13 beats/min (37–98). HR remained significantly elevated at 30 seconds after apnea compared with baseline ($P = 0.001$) and did not return to baseline levels within 60 seconds ($P = 0.021$).

Blood Pressure

BP increased significantly during STA ($P < 0.0001$). The median increase per minute was 10.7 mmHg for SBP (5.5–29.3) (Fig. 5A), 9.3 mmHg for DBP (3.9–22.9) (Fig. 5B), and 9.6 mmHg for MAP (5.4–24.9). At T_{max}, mean BP values were 170 ± 21 mmHg (138–223) for SBP, 111 ± 13 mmHg (90–145) for DBP, and 137 ± 12 mmHg (117–157) for MAP. Although BP decreased rapidly after apnea, values remained significantly elevated at 60 seconds. The median differences at 60 seconds were 12.0 mmHg for SBP ($P < 0.0001$), 5.0 mmHg for DBP ($P = 0.001$), and 7.4 mmHg for MAP ($P < 0.0001$). At 120 seconds, DBP no longer differed significantly from baseline ($P = 0.073$), whereas SBP ($P = 0.001$) and MAP ($P = 0.004$) remained significantly elevated.

Associations Between IOP and Physiological Parameters

Significant correlations were observed between changes in IOP and selected physiological parameters. Changes in IOP correlated negatively with SpO₂ at T_{max} ($r = -0.409$) (Fig. 6A). Increases in IOP correlated positively with increases in SBP at T₂–3 ($r = 0.474$) and T₃–4 ($r = 0.502$) (Fig. 6B), as well as

at T_{max} ($r = 0.357$). A weaker positive correlation was observed between changes in IOP and MAP at T₂₋₃ ($r = 0.374$) and T_{max} ($r = 0.320$). A moderate negative correlation between changes in IOP and HR was observed at T₃₋₄ ($r = -0.482$).

DISCUSSION

Previous studies investigating the effect of breath-holding on IOP have focused primarily on very short apneas lasting several seconds [6,9,10]. However, to the best of our knowledge, no previous study has evaluated IOP dynamics during prolonged apnea in BHDs, and only limited data are available regarding its relationship with glaucomatous disease [23].

IOP During Prolonged STA

In our study, IOP demonstrated a clear time-dependent increase during STA, consistent across participants. Although increases in BP occurred in parallel with the rise in IOP, this association does not necessarily imply a direct causal relationship.

Therefore, additional mechanisms are likely involved in the observed increase in IOP. These complex interactions are illustrated by representative individual recordings (Fig. 7), demonstrating the temporal evolution of the measured parameters during STA.

A recent study reported an increase in IOP during simulated normobaric hypoxia [24]. Although a comparable decrease in oxygen saturation was observed, IOP increased by only approximately 1 mmHg, whereas the mean increase in our study was 12.6 mmHg. These findings suggest that reduced SpO₂ alone is unlikely to be the primary driver of IOP elevation in BHDs. Moreover, the mechanisms underlying IOP changes associated with hypoxemia remain incompletely understood.

IOP may be more strongly influenced by the partial pressure of carbon dioxide (pCO₂), which increases during apnea in parallel with the decline in SpO₂ [20,21,25]. Although pCO₂ was not measured in our study, its dynamics in BHDs are well described. Under normal conditions, pCO₂ is approximately 40 mmHg [25], and during breath-holding after maximal inspiration without hyperventilation, it exceeds 50 mmHg [26,27].

The effect of hypercapnia on IOP rise has been described previously, for example during general anesthesia [28,29]. Hypercapnia induced by breathing in a closed circuit may provide a useful comparison; in that study, pCO₂ increased to approximately 55 mmHg and IOP by approximately 4 mmHg [30].

The mechanisms underlying this phenomenon are not fully understood and are likely multifactorial. Elevated pCO₂ may induce vasodilation in the choroidal circulation, leading to an increase in intraocular blood volume. In addition, hypercapnia may increase venous pressure, thereby contributing to IOP elevation. Another potential mechanism involves increased aqueous humor (AH) production due to enhanced ultrafiltration associated with increased perfusion of the ciliary body [30]. Furthermore,

respiratory acidosis and the associated increase in bicarbonate levels may influence AH dynamics and further contribute to elevated IOP [31].

Acute IOP increases associated with BP elevation have been described during isometric exercise [32,33]. A linear relationship between changes in BP and IOP has been reported; however, the underlying pathophysiological mechanism linking these variables remains unclear [32].

The choroid is thought to exhibit myogenic autoregulation [34] and to respond to sympathetic nervous system activation [35,36]. In both cases, choroidal vasoconstriction contributes to maintaining relatively stable choroidal blood flow (ChBF) [37]. This is further supported by measurements of choroidal vascular resistance [38]. These findings suggest that ChBF is not substantially influenced by increases in BP [33,39]. Accordingly, our results do not support a direct causal relationship between BP elevation and the observed increase in IOP.

Venous Mechanisms of IOP Elevation

Another important factor that may contribute to elevated IOP in BHDs is an increase in central venous pressure (CVP). CVP is transmitted to the eye, leading to venous congestion within the choroidal circulation and an increase in choroidal volume, which may result in elevated IOP. At the same time, episcleral venous pressure (EVP) increases, creating resistance to AH outflow and thereby further contributing to IOP rise [8].

To our knowledge, EVP has not yet been measured in BHDs. However, a study has demonstrated a 162% increase in internal jugular venous pressure (IJVP) during STA of approximately 5 minutes in competitive BHDs [40]. Further evidence indicates that the increase in IJVP depends on lung volume prior to apnea, with IJVP being approximately 10 mmHg higher at total lung capacity (TLC) compared with STA performed at functional residual capacity (FRC) [41]. It is therefore possible that during GI, IJVP may be even higher than at TLC, potentially further contributing to the observed increase in IOP.

The mechanisms underlying the increase in IJVP can be inferred from previous studies. During GI, intrapulmonary and transpulmonary pressures increase, leading to transient compression of the heart and intrathoracic vessels, displacement of blood from the thorax to the periphery, and a reduction in venous return [42]. During STA, further cardiovascular responses occur. Alongside bradycardia, peripheral vasoconstriction, and increased BP, BHDs exhibit a reduction in cardiac output and venous return [43], which may promote blood pooling in the venous system proximal to the heart. A similar mechanism has been described in conditions associated with venous congestion, such as heart failure, where blood accumulation in the episcleral venous system has been reported [44].

Splenic contraction has also been described during dry STA [45], during which approximately 100–125 mL of erythrocytes are released into the circulation [46]. This relatively small increase in blood volume may contribute to elevated CVP; however, it is unlikely to play a dominant role compared with other mechanisms.

With progressive hypoxia and hypercapnia, BHDs develop involuntary breathing movements (IBMs), characterized by diaphragmatic contractions that attempt to restore ventilation during apnea [47,48]. The onset of IBMs marks the transition from the “easy-going” phase to the “struggle phase” [49], during which intrathoracic pressure fluctuates between positive and negative values [50].

These fluctuations may further augment venous pressure and contribute to the elevated IOP observed toward the end of apnea. This interpretation is supported by previous work suggesting that blood pooling in the cerebral venous system results from increased back-pressure and vascular resistance caused by compression of intrathoracic vessels at high lung volumes and further amplified by IBMs [41].

Taken together, these findings suggest that IOP elevation during STA in BHDs could be primarily driven by an increase in EVP, leading to impaired AH outflow. The onset of IBMs likely contributes to further increases in EVP, while the magnitude of EVP may be additionally influenced by the use of GI and the duration of IBMs. Venous congestion within the choroidal circulation and the effects of hypercapnia may also contribute to IOP elevation. However, this hypothesis requires further experimental validation.

Systemic Physiological Responses During STA

During our study, we observed several physiological responses associated with the diving reflex [20,51]. This response is triggered by breath-holding itself but is further augmented by immersion in cold water [43,52]. As measurements were performed on land, the magnitude of this response may have been attenuated.

In elite BHDs, arterial oxygen saturation can decrease to values below 50% during prolonged apnea [53,54]. Although such extreme desaturation was not observed in our cohort, a substantial decrease in SpO₂ occurred.

SpO₂ declined predominantly toward the end of apnea, and in some participants, the lowest values were reached after apnea termination. This delayed nadir is likely explained by circulatory delay between the lungs and the peripheral measurement site, as previously described [55,56]. SpO₂ has been shown to reach its minimum approximately 15–20 seconds after the end of apnea [56]. In our cohort, SpO₂ returned to baseline values within 60 seconds.

In elite BHDs, the diving reflex is more pronounced, resulting in more efficient oxygen utilization and slower desaturation compared with untrained individuals [57,58]. Given the heterogeneous composition of our cohort, including participants with varying levels of experience, interindividual differences in desaturation patterns may have influenced the observed SpO₂ dynamics.

Bradycardia is a well-described component of the diving reflex, with HR commonly decreasing to approximately 40 beats/min in BHDs and to as low as 20 beats/min in elite divers [22]. This response is mediated by increased vagal activity and is typically accompanied by a reduction in cardiac output [59,60].

Despite this, we did not observe a significant decrease in HR during STA in our cohort. However, approximately half of the participants exhibited HR values below 60 beats/min, and in one individual, HR decreased below 40 beats/min. This may be explained by the absence of facial immersion in cold water, as well as by the stress associated with repeated IOP measurements, which could have attenuated the expected bradycardic response.

A transient increase in HR observed immediately after maximal inspiration prior to apnea is likely related to pre-apnea breathing maneuvers [61,62]. To prolong breath-hold duration, BHDs often use GI (“lung packing”), which increases lung volume beyond TLC [63–66]. When using this technique, lung volume may increase by up to approximately 47% [64,67], leading to elevated transpulmonary pressure [42,68]. GI has been associated with a transient hypotensive phase followed by increases in HR and BP [67,68].

The transient hypotensive phase described above was not captured in our study, likely due to the lack of continuous BP monitoring and the short duration of this response. In contrast, the subsequent increase in BP was well documented, although in many cases values became unmeasurable by our device immediately before the end of STA.

BP increases with the duration of apnea and appears to be independent of HR changes, both in dry conditions and during cold water immersion [69]. With prolonged STA, sympathetic activity increases, leading to peripheral vasoconstriction and a progressive rise in BP [52]. Additional sympathetic activation is mediated by chemoreceptor stimulation in response to developing hypoxemia and hypercapnia [70].

The maximal BP values observed in our study (SBP 223 mmHg, DBP 145 mmHg) were slightly higher than those reported in a study investigating BP and HR changes during dry STA (SBP 200 mmHg, DBP 120 mmHg) [61], which may be explained by the longer mean duration of STA in our cohort. Toward the end of STA, we observed a marked increase in BP, consistent with previous studies [53,55,61]. This pronounced rise in BP may be partly attributed to the Cushing reflex [41].

Limitations

This study has several limitations. First, the inclusion of BHDs with varying experience resulted in a relatively heterogeneous cohort, which may have influenced the results. A more homogeneous group of highly trained BHDs with longer breath-hold durations might exhibit even higher IOP values.

Another limitation was the lack of continuous measurement of key parameters, particularly IOP and BP. Measurement of IOP toward the end of STA was technically challenging due to the presence of IBMs, and BP values were often unmeasurable at this stage using our device.

A further limitation is the absence of direct measurements of pCO₂ and EVP; therefore, their contribution to IOP elevation is inferred based on physiological considerations. Additional insights could be gained by stratifying participants according to the use of GI, as well as by documenting the onset of IBMs and the duration of the “struggle phase.”

Finally, all measurements were performed at rest under dry conditions and therefore do not reflect the dynamic aspects of diving or the additional physical factors associated with depth exposure.

CONCLUSION

Despite these limitations, this is, to the best of our knowledge, the first study to evaluate dynamic IOP changes during prolonged dry STA in BHDs in a relatively large cohort. Our findings raise the possibility that repeated transient IOP elevations during prolonged apnea may have implications for glaucoma risk. Future studies should experimentally confirm the relationship between IOP and EVP during apnea in BHDs and investigate the long-term impact of repeated transient IOP elevations on the optic nerve and visual field.

Declarations

Funding

This study was supported by the Ministry of Health of the Czech Republic – Conceptual Development of Research Organization (FNOL, 00098892).

Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

Ethics Approval

The study protocol was approved by the Ethics Committee of University Hospital Olomouc and the Faculty of Medicine and Dentistry, Palacký University Olomouc (No. 103/26; approved on April 13, 2026), and adhered to the tenets of the Declaration of Helsinki.

Consent to Participate

Informed consent was obtained from all individual participants included in the study.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

P.P. and M.K. conceived and designed the research; P.P. performed the experiments and collected the data; P.P. analyzed the data; P.P. and M.K. interpreted the results of the experiments; P.P. prepared the figures; P.P. drafted the manuscript; P.P., Z.S., M.Š., and M.K. edited and revised the manuscript; P.P., Z.S.,

References

1. Paidimarri S, Bhardwaj R, Pathak-Ray V. Can Breathing Exercise or Pranayama Affect the Intraocular Pressure in the Short-term? *J Glaucoma*. 2021;30:e18–20.
<https://doi.org/10.1097/IJG.0000000000001682>
2. Kulkarni A, Kamath Y, Shetty L, Kuzhuppilly NIR. The Effect of Specific Techniques of Nasal Breathing [Pranayama] on Intra-Ocular Pressure in Normal Individuals, a Randomized Trial. *Clin Ophthalmol*. 2022;16:4047–54. <https://doi.org/10.2147/OPHTH.S389495>
3. Morya AK, Shrivastava AK, Janti SS, Tejaswini A, Gupta R, Gurnani B, et al. Effect of Asanas in Yoga on Intraocular Pressure of Practicing Healthy Individuals: a Prospective Observational Study. *Maedica (Bucur)*. 2023;18:238–45. <https://doi.org/10.26574/maedica.2023.18.2.238>
4. Dada T, Gwal RS, Mahalingam K, Chandran DS, Angmo D, Gupta S, et al. Effect of “365 Breathing Technique” on Intraocular Pressure and Autonomic Functions in Patients With Glaucoma: A Randomized Controlled Trial. *J Glaucoma*. 2024;33:149–54.
<https://doi.org/10.1097/IJG.0000000000002356>
5. Udenia H, Mittal S, Agrawal A, Singh A, Singh A, Mittal SK. Yogic Pranayama and Diaphragmatic Breathing: Adjunct Therapy for Intraocular Pressure in Patients With Primary Open-angle Glaucoma: A Randomized Controlled Trial. *J Glaucoma*. 2021;30:115–23.
<https://doi.org/10.1097/IJG.0000000000001697>
6. Liu W-C, Lee S-M, Graham AD, Lin MC. Effects of Eye Rubbing and Breath Holding on Corneal Biomechanical Properties and Intraocular Pressure. *Cornea*. 2011;30:855–60.
<https://doi.org/10.1097/ICO.0b013e3182032b21>
7. Lam AKC, Lam C-H. Effect of Breath-Holding on Pulsatile Ocular Blood Flow Measurement in Normal Subjects. *Optom Vis Sci*. 2004;81:597–600. <https://doi.org/10.1097/01.opx.0000141795.95597.98>
8. Dos Santos MG, Makk S, Berghold A, Eckhardt M, Haas A. Intraocular pressure difference in Goldmann applanation tonometry versus Perkins hand-held applanation tonometry in overweight patients. *Ophthalmology*. 1998;105:2260–3. [https://doi.org/10.1016/S0161-6420\(98\)91226-X](https://doi.org/10.1016/S0161-6420(98)91226-X)
9. Vieira GM. Intraocular Pressure Variation During Weight Lifting. *Arch Ophthalmol*. 2006;124:1251. <https://doi.org/10.1001/archopht.124.9.1251>
10. Vera J, Perez-Castilla A, Redondo B, De La Cruz JC, Jiménez R, García-Ramos A. Influence of the breathing pattern during resistance training on intraocular pressure. *Eur J Sport Sci*. 2020;20:157–65. <https://doi.org/10.1080/17461391.2019.1617354>
11. Alward WLM. Medical Management of Glaucoma. *N Engl J Med*. 1998;339:1298–307.
<https://doi.org/10.1056/NEJM199810293391808>

12. Baser G, Karahan E, Bilgin S, Unsal U. Evaluation of the effect of daily activities on intraocular pressure in healthy people: is the 20 mmHg border safe? *Int Ophthalmol*. 2018;38:1963–7. <https://doi.org/10.1007/s10792-017-0684-2>
13. Wang YX, Xu L, Wei WB, Jonas JB. Intraocular pressure and its normal range adjusted for ocular and systemic parameters. *The Beijing Eye Study 2011*. *PLoS ONE*. 2018;13:e0196926. <https://doi.org/10.1371/journal.pone.0196926>
14. Asrani SG, McGlumphy EJ, Al-Aswad LA, Chaya CJ, Lin S, Musch DC, et al. The relationship between intraocular pressure and glaucoma: An evolving concept. *Prog Retin Eye Res*. 2024;103:101303. <https://doi.org/10.1016/j.preteyeres.2024.101303>
15. Allison K, Patel D, Alabi O. Epidemiology of Glaucoma: The Past, Present, and Predictions for the Future. *Cureus*. 2020;12:e11686. <https://doi.org/10.7759/cureus.11686>
16. Reis TF, Paula JS, Furtado JM. Primary glaucomas in adults: Epidemiology and public health: A review. *Clin Exp Ophthalmol*. 2022;50:128–42. <https://doi.org/10.1111/ceo.14040>
17. Qi T, Liu H, Fröhn L, Löw K, Cursiefen C, Prokosch V. Understanding Glaucoma: Why it Remains a Leading Cause of Blindness Worldwide. *Klin Monbl Augenheilkd*. 2025;242:712–7. <https://doi.org/10.1055/a-2617-1575>
18. Wang Z, Xue CC, Li Y, Wu Y, Pan Z, Li F, et al. Global Glaucoma Prevalence: Burden and Projection to 2060. *Am J Ophthalmol*. 2026;283:324–35. <https://doi.org/10.1016/j.ajo.2025.12.013>
19. Vrdoljak D, Hubbard CD, Coombs GB, Lovering AT, Drvis I, Foretic N, et al. Dynamics of Preparatory Apneas and Their Influence on Maximal Dry Static Apnea in Breath-Hold Divers. *J Funct Morphol Kinesiol*. 2025;10:471. <https://doi.org/10.3390/jfmk10040471>
20. Ferretti G. Extreme human breath-hold diving. *Eur J Appl Physiol*. 2001;84:254–71. <https://doi.org/10.1007/s004210000377>
21. Bain AR, Drvis I, Dujic Z, MacLeod DB, Ainslie PN. Physiology of static breath holding in elite apneists. *Exp Physiol*. 2018;103:635–51. <https://doi.org/10.1113/EP086269>
22. Tetzlaff K, Muth C-M. Physiological Challenges and Adaptations in Competitive Freediving. *Dtsch Z Sportmed*. 2024;75:207–15. <https://doi.org/10.5960/dzsm.2024.613>
23. Lee E, Yang JW, Kwon JD. The Effect of Breath-Hold Diving on Intraocular Pressure, Optic Nerve, Visual Field in Korean Haenyeo (Female Divers). *J Korean Ophthalmol Soc*. 2012;53:1480–7. <https://doi.org/10.3341/jkos.2012.53.10.1480>
24. Najmanová E, Pluháček F, Botek M, Krejčí J, Jarošová J. Intraocular Pressure Response to Short-Term Extreme Normobaric Hypoxia Exposure. *Front Endocrinol (Lausanne)*. 2019;9:785. <https://doi.org/10.3389/fendo.2018.00785>
25. Parkes MJ. Breath-holding and its breakpoint. *Exp Physiol*. 2006;91:1–15. <https://doi.org/10.1113/expphysiol.2005.031625>
26. Lin YC, Lally DA, Moore TO, Hong SK. Physiological and conventional breath-hold breaking points. *J Appl Physiol*. 1974;37:291–6. <https://doi.org/10.1152/jappl.1974.37.3.291>

27. Bain AR, Ainslie PN, Barak OF, Hoiland RL, Drvis I, Mijacika T, et al. Hypercapnia is essential to reduce the cerebral oxidative metabolism during extreme apnea in humans. *J Cereb Blood Flow Metab.* 2017;37:3231–42. <https://doi.org/10.1177/0271678X16686093>
28. Samuel JR, Beaugie A. Effect of carbon dioxide on the intraocular pressure in man during general anaesthesia. *Br J Ophthalmol.* 1974;58:62–7. <https://doi.org/10.1136/bjo.58.1.62>
29. Petounis AD, Chondreli S, Vadaluka-Sekioti A. Effect of hypercapnea and hyperventilation on human intraocular pressure general anaesthesia following acetazolamide administration. *Br J Ophthalmol.* 1980;64:422–5. <https://doi.org/10.1136/bjo.64.6.422>
30. Kielar RA, Teraslinna P, Kearney JT, Barker D. Effect of changes in Pco₂ on intraocular tension. *Invest Ophthalmol Vis Sci.* 1977;16:534–7.
31. Xie Y, Yang Y, Han Y, Yang D, Sun Y, Wang X, et al. Association Between Arterial Blood Gas Variation and Intraocular Pressure in Healthy Subjects Exposed to Acute Short-Term Hypobaric Hypoxia. *Transl Vis Sci Technol.* 2019;8:22. <https://doi.org/10.1167/tvst.8.6.22>
32. Castejon H, Chiquet C, Savy O, Baguet J-P, Khayi H, Tamisier R, et al. Effect of Acute Increase in Blood Pressure on Intraocular Pressure in Pigs and Humans. *Invest Ophthalmol Vis Sci.* 2010;51:1599. <https://doi.org/10.1167/iovs.09-4215>
33. Polska E, Simader C, Weigert G, Doelemeyer A, Kolodjaschna J, Scharmann O, et al. Regulation of Choroidal Blood Flow during Combined Changes in Intraocular Pressure and Arterial Blood Pressure. *Invest Ophthalmol Vis Sci.* 2007;48:3768. <https://doi.org/10.1167/iovs.07-0307>
34. Kiel JW. Choroidal Myogenic Autoregulation and Intraocular Pressure. *Exp Eye Res.* 1994;58:529–43. <https://doi.org/10.1006/exer.1994.1047>
35. Bill A, Linder M, Linder J. The protective role of ocular sympathetic vasomotor nerves in acute arterial hypertension. *Bibl Anat.* 1977;16:30–5.
36. Reiner A, Fitzgerald MEC, Del Mar N, Li C. Neural control of choroidal blood flow. *Prog Retin Eye Res.* 2018;64:96–130. <https://doi.org/10.1016/j.preteyeres.2017.12.001>
37. Polak K, Polska E, Luksch A, Dorner G, Fuchsjäger-Mayrl G, Findl O, et al. Choroidal blood flow and arterial blood pressure. *Eye.* 2003;17:84–8. <https://doi.org/10.1038/sj.eye.6700246>
38. Riva CE, Titze P, Hero M, Movaffaghy A, Petrig BL. Choroidal blood flow during isometric exercises. *Invest Ophthalmol Vis Sci.* 1997;38:2338–43.
39. Luksch A, Polska E, Imhof A, Schering J, Fuchsjäger-Mayrl G, Wolzt M, et al. Role of NO in Choroidal Blood Flow Regulation during Isometric Exercise in Healthy Humans. *Invest Ophthalmol Vis Sci.* 2003;44:734. <https://doi.org/10.1167/iovs.02-0177>
40. Bain AR, Ainslie PN, Hoiland RL, Barak OF, Drvis I, Stembridge M, et al. Competitive apnea and its effect on the human brain: focus on the redox regulation of blood-brain barrier permeability and neuronal-parenchymal integrity. *FASEB J.* 2018;32:2305–14. <https://doi.org/10.1096/fj.201701031R>
41. Stembridge M, Hoiland RL, Bain AR, Barak OF, Drvis I, MacLeod DB, et al. Influence of lung volume on the interaction between cardiac output and cerebrovascular regulation during extreme apnoea. *Exp Physiol.* 2017;102:1288–99. <https://doi.org/10.1113/EP086429>

42. Loring SH, O'Donnell CR, Butler JP, Lindholm P, Jacobson F, Ferrigno M. Transpulmonary pressures and lung mechanics with glossopharyngeal insufflation and exsufflation beyond normal lung volumes in competitive breath-hold divers. *J Appl Physiol.* 2007;102:841–6.
<https://doi.org/10.1152/japplphysiol.00749.2006>
43. Andersson JPA, Linér MH, Fredsted A, Schagatay EKA. Cardiovascular and respiratory responses to apneas with and without face immersion in exercising humans. *J Appl Physiol.* 2004;96:1005–10.
<https://doi.org/10.1152/japplphysiol.01057.2002>
44. Koç Ş. Episcleral Venous Tortuosity Indicates Increased Ventricular Filling Pressure in Heart Failure with Reduced Ejection Fraction. *J Updates Cardiovasc Med.* 2020;8:113–22.
<https://doi.org/10.32596/ejcm.galenos.2020.02.08>
45. Persson G, Lodin-Sundström A, Linér MH, Andersson SHA, Sjögren B, Andersson JPA. Splenic contraction and cardiovascular responses are augmented during apnea compared to rebreathing in humans. *Front Physiol.* 2023;14:1109958. <https://doi.org/10.3389/fphys.2023.1109958>
46. Baković D, Eterović D, Saratlija-Novaković Z, Palada I, Valic Z, Bilopavlović N, et al. Effect of human splenic contraction on variation in circulating blood cell counts. *Clin Exp Pharmacol Physiol.* 2005;32:944–51. <https://doi.org/10.1111/j.1440-1681.2005.04289.x>
47. Breskovic T, Lojpur M, Maslov PZ, Cross TJ, Kraljevic J, Ljubkovic M, et al. The influence of varying inspired fractions of O₂ and CO₂ on the development of involuntary breathing movements during maximal apnoea. *Respir Physiol Neurobiol.* 2012;181:228–33.
<https://doi.org/10.1016/j.resp.2012.03.007>
48. Duke JW, Hubbard CD, Vrdoljak D, Coombs GB, Lovering AT, Drvis I, et al. Respiratory muscle strength pre- and post-maximal apneas in a world champion breath-hold diver. *Journal of Applied Physiology.* 2025;138:66–72. <https://doi.org/10.1152/japplphysiol.00671.2024>
49. Hubbard CD, Vrdoljak D, Coombs GB, Cross TJ, Taylor BJ, Lovering AT, et al. Reduction in respiratory muscle strength following a series of preparatory and maximal static, dry breath-holds. *Journal of Applied Physiology.* 2025;139:1235–45. <https://doi.org/10.1152/japplphysiol.00256.2025>
50. Cross TJ, Kavanagh JJ, Breskovic T, Zubin Maslov P, Lojpur M, Johnson BD, et al. The Effects of Involuntary Respiratory Contractions on Cerebral Blood Flow during Maximal Apnoea in Trained Divers. *PLoS ONE.* 2013;8:e66950. <https://doi.org/10.1371/journal.pone.0066950>
51. Malinowski KS, Pawłowski R, Winklewski PJ. Neurocardiac diving reflex dynamics: assessment and prediction. *The European Physical Journal Special Topics.* 2026;
<https://doi.org/10.1140/epjs/s11734-026-02187-0>
52. Gooden BA. Mechanism of the human diving response. *Integr Physiol Behav Sci.* 1994;29:6–16.
<https://doi.org/10.1007/BF02691277>
53. Heusser K, Dzamonja G, Tank J, Palada I, Valic Z, Bakovic D, et al. Cardiovascular regulation during apnea in elite divers. *Hypertension.* 2009;53:719–24.
<https://doi.org/10.1161/HYPERTENSIONAHA.108.127530>

54. Dujic Z, Breskovic T, Bakovic D. Breath-hold diving as a brain survival response. *Transl Neurosci*. 2013;4:302–13. <https://doi.org/10.2478/s13380-013-0130-5>
55. Andersson JPA, Linér MH, Rünow E, Schagatay EKA. Diving response and arterial oxygen saturation during apnea and exercise in breath-hold divers. *J Appl Physiol*. 2002;93:882–6. <https://doi.org/10.1152/jappphysiol.00863.2001>
56. Breskovic T, Uglesic L, Zubin P, Kuch B, Kraljevic J, Zanchi J, et al. Cardiovascular changes during underwater static and dynamic breath-hold dives in trained divers. *J Appl Physiol*. 2011;111:673–8. <https://doi.org/10.1152/jappphysiol.00209.2011>
57. Ferretti G, Costa M, Ferrigno M, Grassi B, Marconi C, Lundgren CE, et al. Alveolar gas composition and exchange during deep breath-hold diving and dry breath holds in elite divers. *J Appl Physiol*. 1991;70:794–802. <https://doi.org/10.1152/jappl.1991.70.2.794>
58. Delahoche J, Delapille P, Lemaître F, Verin E, Tourny-Chollet C. Arterial Oxygen Saturation and Heart Rate Variation During Breath-Holding: Comparison between Breath-Hold Divers and Controls. *Int J Sports Med*. 2005;26:177–81. <https://doi.org/10.1055/s-2004-820976>
59. Lemaître F, Lafay V, Taylor M, Costalat G, Gardette B. Electrocardiographic aspects of deep dives in elite breath-hold divers. *Undersea Hyperb Med*. 2013;40:145–54.
60. Alboni P, Alboni M, Gianfranchi L. Diving bradycardia: a mechanism of defence against hypoxic damage. *J Cardiovasc Med (Hagerstown)*. 2011;12:422–7. <https://doi.org/10.2459/JCM.0b013e328344bcde>
61. Perini R, Tironi A, Gheza A, Butti F, Moia C, Ferretti G. Heart rate and blood pressure time courses during prolonged dry apnoea in breath-hold divers. *Eur J Appl Physiol*. 2008;104:1–7. <https://doi.org/10.1007/s00421-008-0771-1>
62. Elia A, Gennser M, Eiken O, Keramidas ME. Systemic and regional hemodynamic and (de)oxygenation responses across repeated breath-holds. *Journal of Applied Physiology*. 2025;139:1019–28. <https://doi.org/10.1152/jappphysiol.00508.2025>
63. Schipke JD, Kelm M, Siegmund K, Muth T, Sievers B, Steiner S. “Lung packing” in breath hold-diving: An impressive case of pulmo–cardiac interaction. *Respir Med Case Rep*. 2015;16:120–1. <https://doi.org/10.1016/j.rmcr.2015.09.007>
64. Heusser K, Dzamonja G, Breskovic T, Steinback CD, Diedrich A, Tank J, et al. Sympathetic and cardiovascular responses to glossopharyngeal insufflation in trained apnea divers. *J Appl Physiol*. 2010;109:1728–35. <https://doi.org/10.1152/jappphysiol.00522.2010>
65. Simpson G, Ferns J, Murat S. Pulmonary effects of „lung packing“ by buccal pumping in an elite breath-hold diver. *SPUMS J*. 2003;33:122–6.
66. Scott TKM, Vrijdag XCE, van Waart H, Harris RJ, Trubridge W, Tillmans F, et al. Arterial blood gas changes in progressively deeper breath-hold dives. *Journal of Applied Physiology*. 2026;141:180–9. <https://doi.org/10.1152/jappphysiol.00324.2026>
67. Dzamonja G, Tank J, Heusser K, Palada I, Valic Z, Bakovic D, et al. Glossopharyngeal insufflation induces cardioinhibitory syncope in apnea divers. *Clin Auton Res*. 2010;20:381–4.

<https://doi.org/10.1007/s10286-010-0075-5>

68. Potkin R, Cheng V, Siegel R. Effects of glossopharyngeal insufflation on cardiac function: an echocardiographic study in elite breath-hold divers. *J Appl Physiol*. 2007;103:823–7.
<https://doi.org/10.1152/jappphysiol.00125.2007>
69. Andersson J, Schagatay E, Gislén A, Holm B. Cardiovascular responses to cold-water immersions of the forearm and face, and their relationship to apnoea. *Eur J Appl Physiol*. 2000;83:566–72.
<https://doi.org/10.1007/s004210000317>
70. Somers VK, Mark AL, Zavala DC, Abboud FM. Contrasting effects of hypoxia and hypercapnia on ventilation and sympathetic activity in humans. *J Appl Physiol*. 1989;67:2101–6.
<https://doi.org/10.1152/jappl.1989.67.5.2101>

Figures

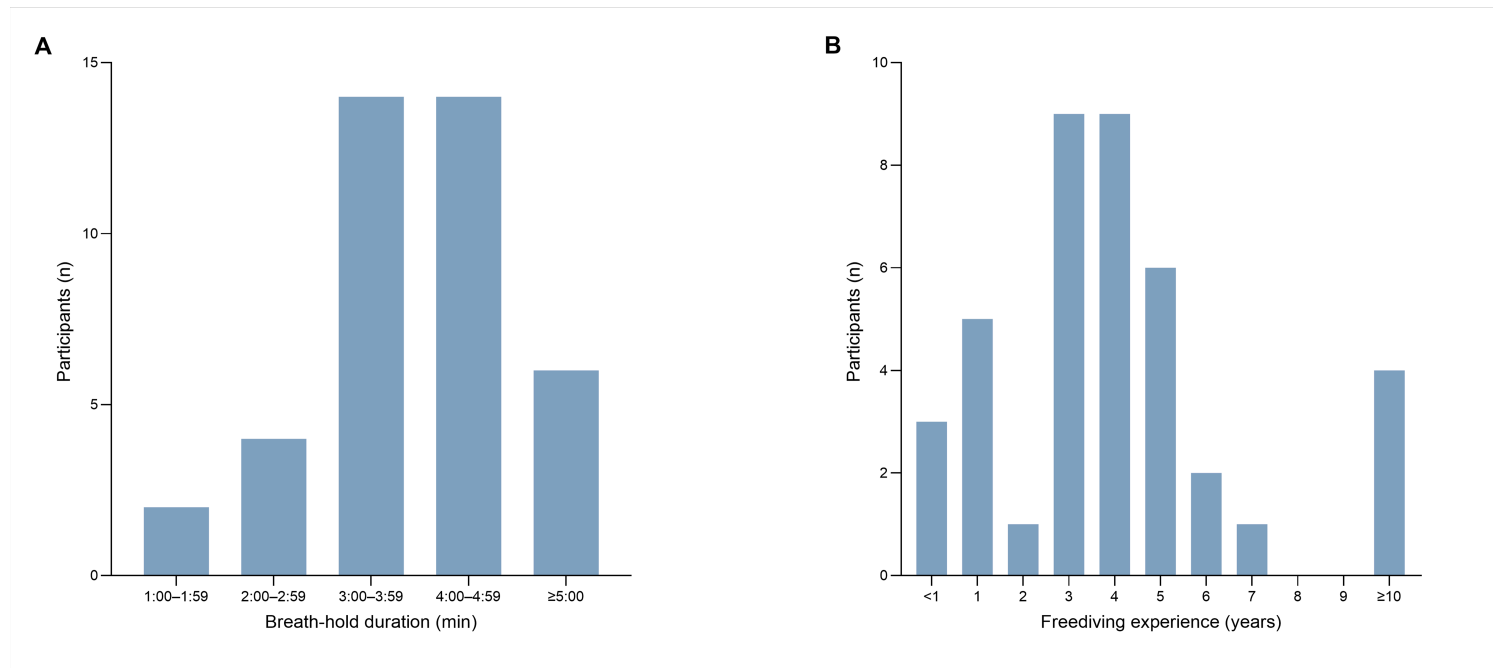


Figure 1

Distribution of breath-hold duration (A) and freediving experience (B) among participants.

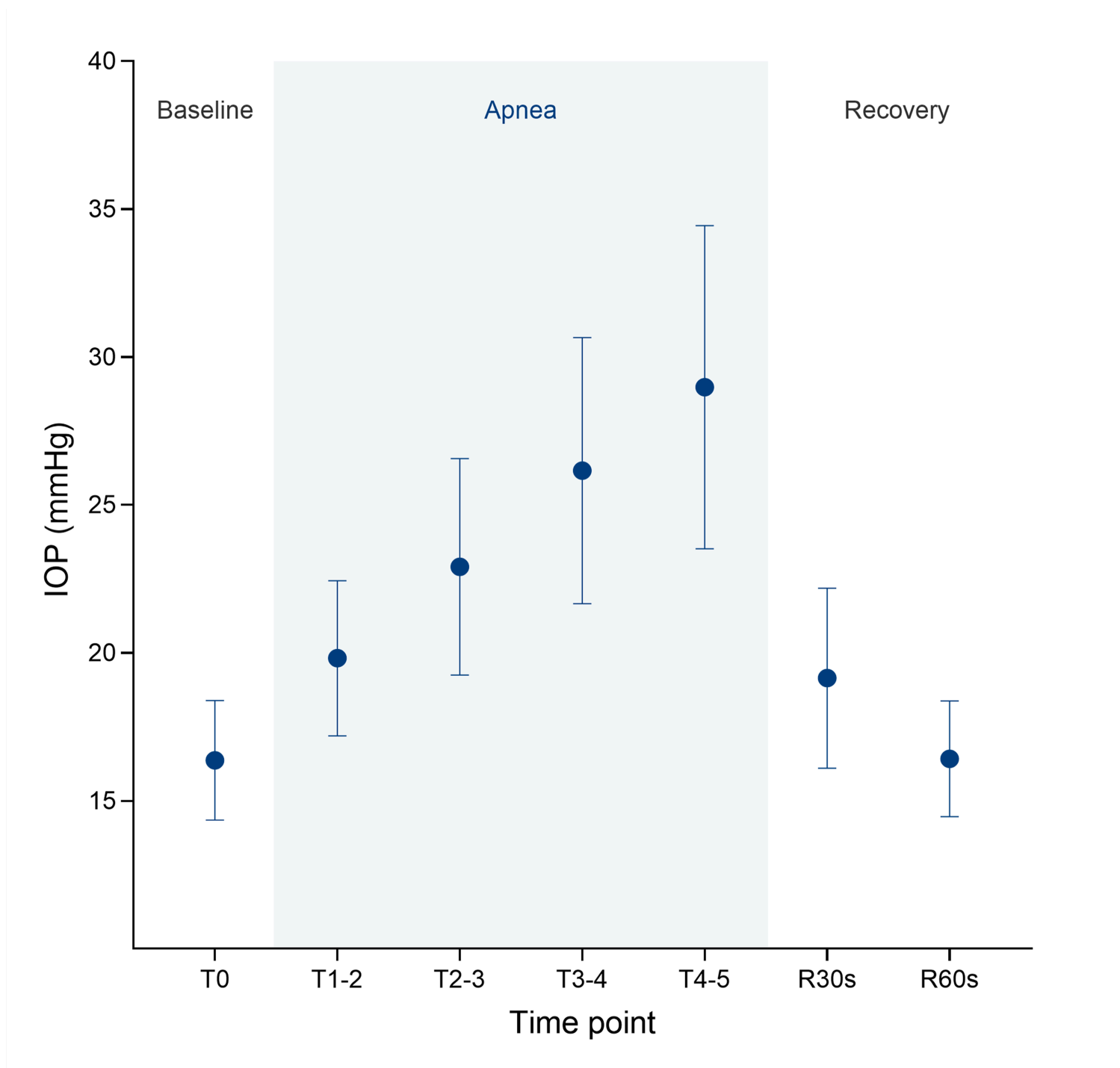


Figure 2

Changes in intraocular pressure (IOP) during dry static apnea (STA) in breath-hold divers. T0 represents baseline; T1–2, T2–3, T3–4, and T4–5 represent 1-min intervals during STA; R30s and R60s represent measurements 30 and 60 s after resumption of breathing, respectively. When multiple measurements were obtained within the same time interval for an individual participant, their mean value was used. Values are presented as mean ± SD.

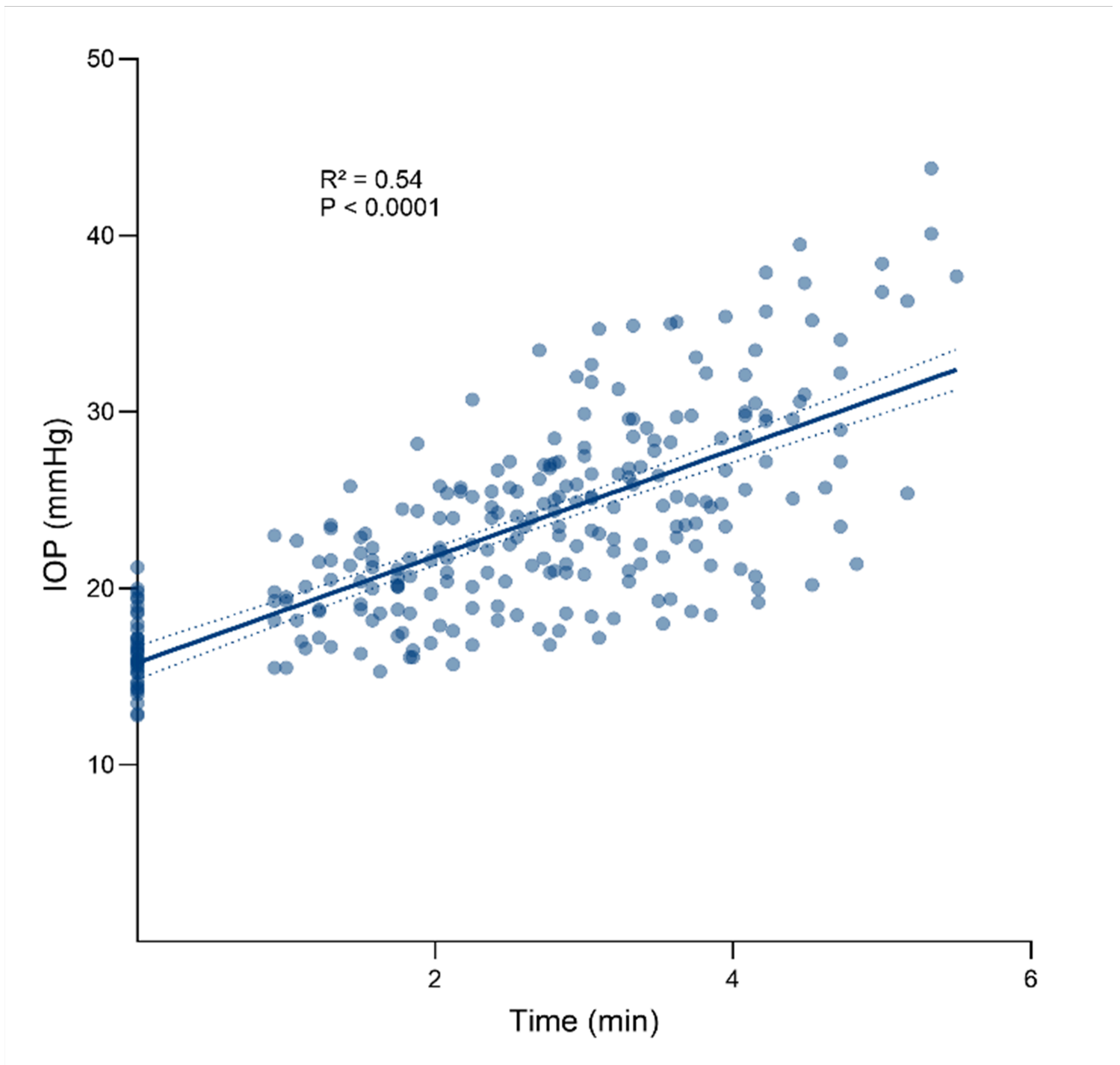


Figure 3

Linear regression analysis showing the relationship between intraocular pressure (IOP) and apnea duration during dry static apnea. Each point represents an individual IOP measurement at its original measurement time. The solid line represents the linear regression line, and the dashed lines indicate the 95% confidence interval. The coefficient of determination (R^2) and P value are shown in the figure.

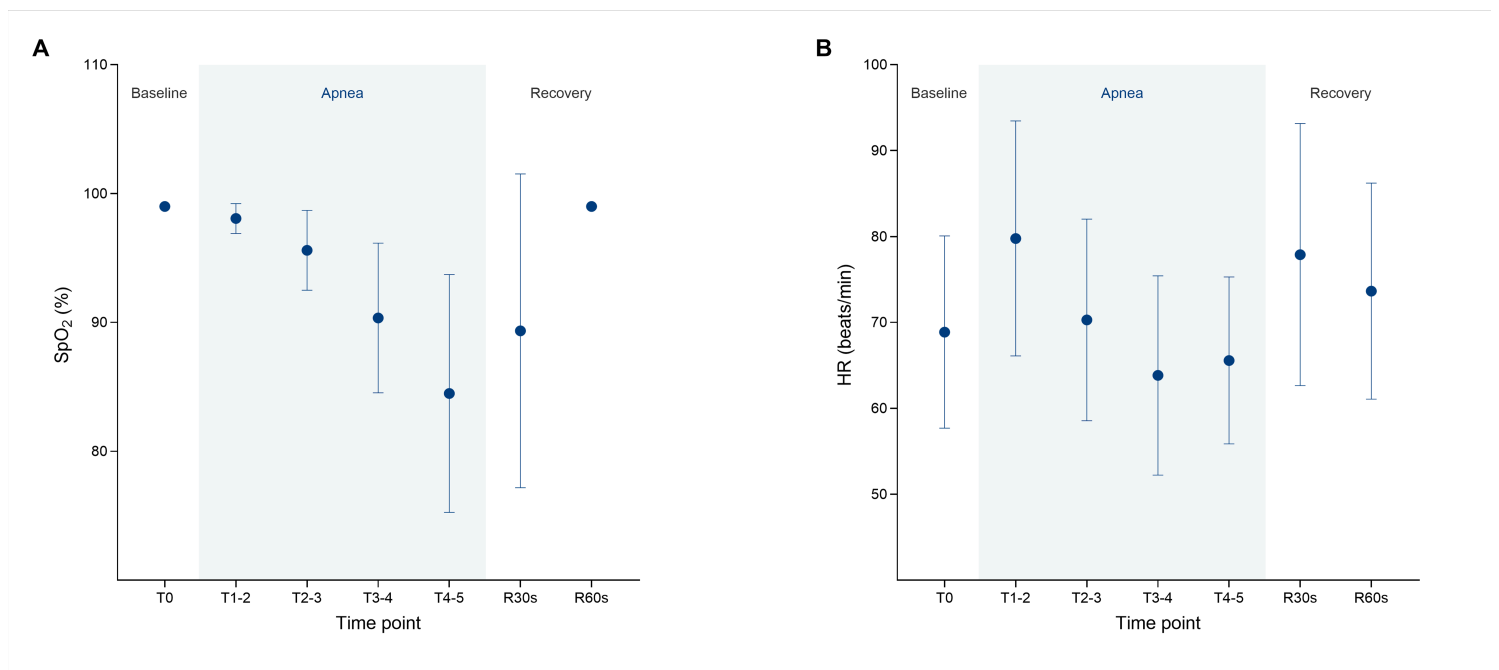


Figure 4

Changes in peripheral oxygen saturation (SpO₂) (A) and heart rate (HR) (B) during dry static apnea (STA) in breath-hold divers. T0 represents baseline; T1–2, T2–3, T3–4, and T4–5 represent 1-min intervals during STA; R30s and R60s represent measurements 30 and 60 s after resumption of breathing, respectively. When multiple measurements were obtained within the same time interval for an individual participant, their mean value was used. Values are presented as mean ± SD.

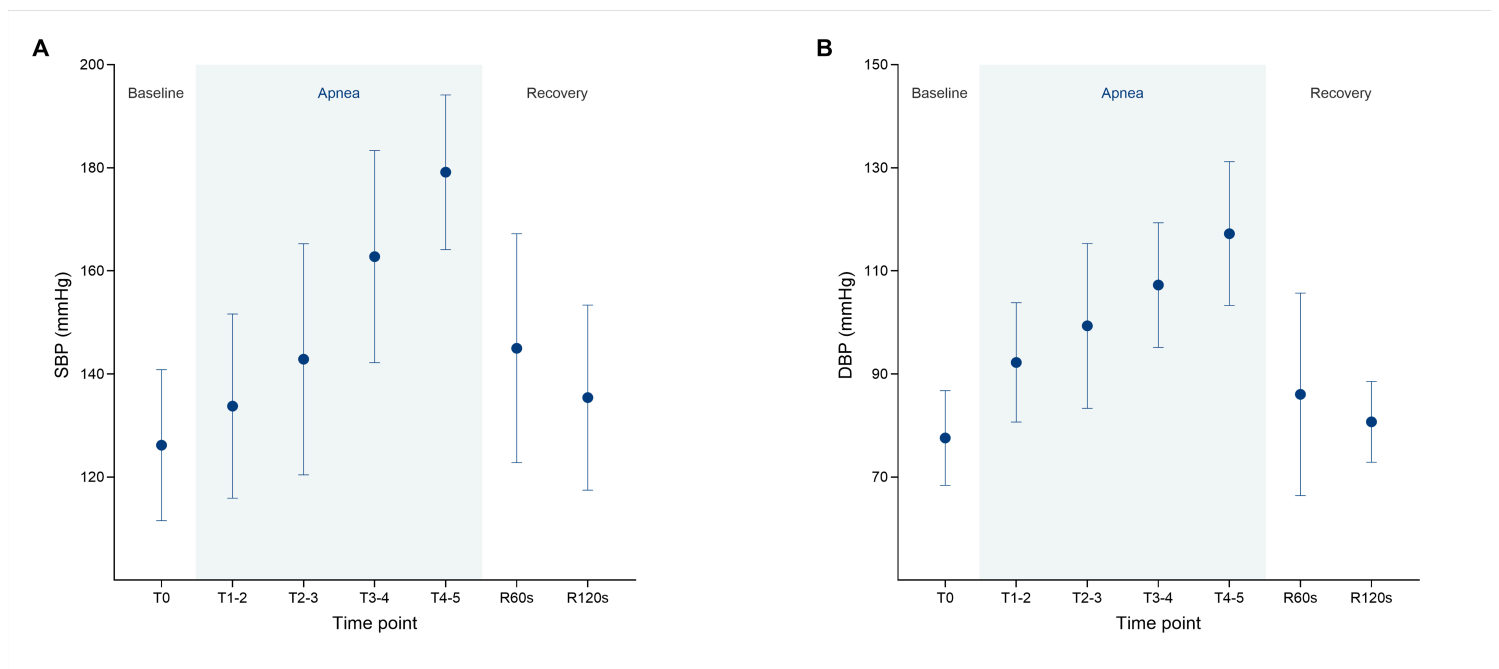


Figure 5

Changes in systolic blood pressure (SBP) (A) and diastolic blood pressure (DBP) (B) during dry static apnea (STA) in breath-hold divers. T0 represents baseline; T1–2, T2–3, T3–4, and T4–5 represent 1-min intervals during STA; R60s and R120s represent measurements 60 and 120 s after resumption of breathing, respectively. Values are presented as mean $\pm$ SD.

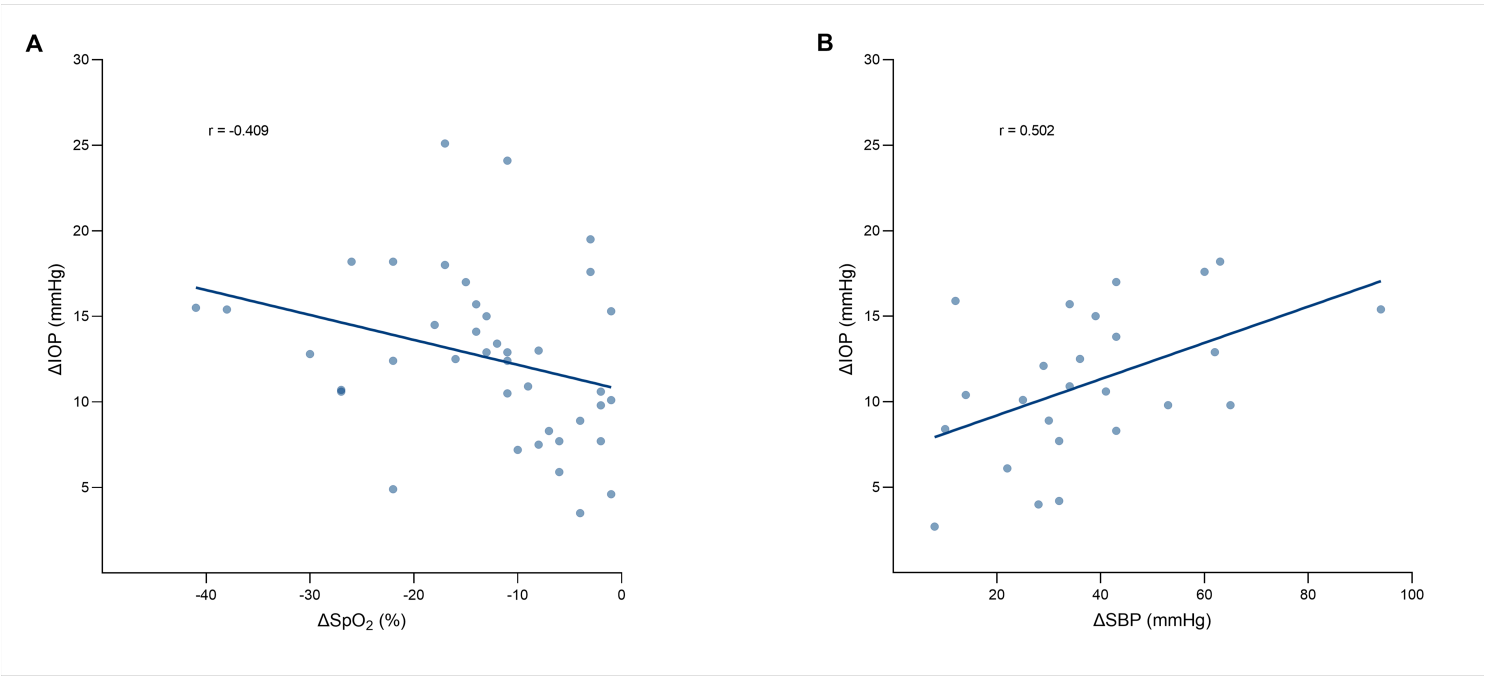


Figure 6

Correlation between changes in intraocular pressure (Δ IOP) and peripheral oxygen saturation (Δ SpO₂) at maximal apnea ($r = -0.409$, $P = 0.009$, $n = 40$) (A), and between changes in intraocular pressure (Δ IOP) and systolic blood pressure (Δ SBP) during the 3–4 min interval of apnea ($r = 0.502$, $P = 0.011$, $n = 25$) (B). Correlations were assessed using Spearman correlation analysis. Solid lines represent the fitted trend lines.

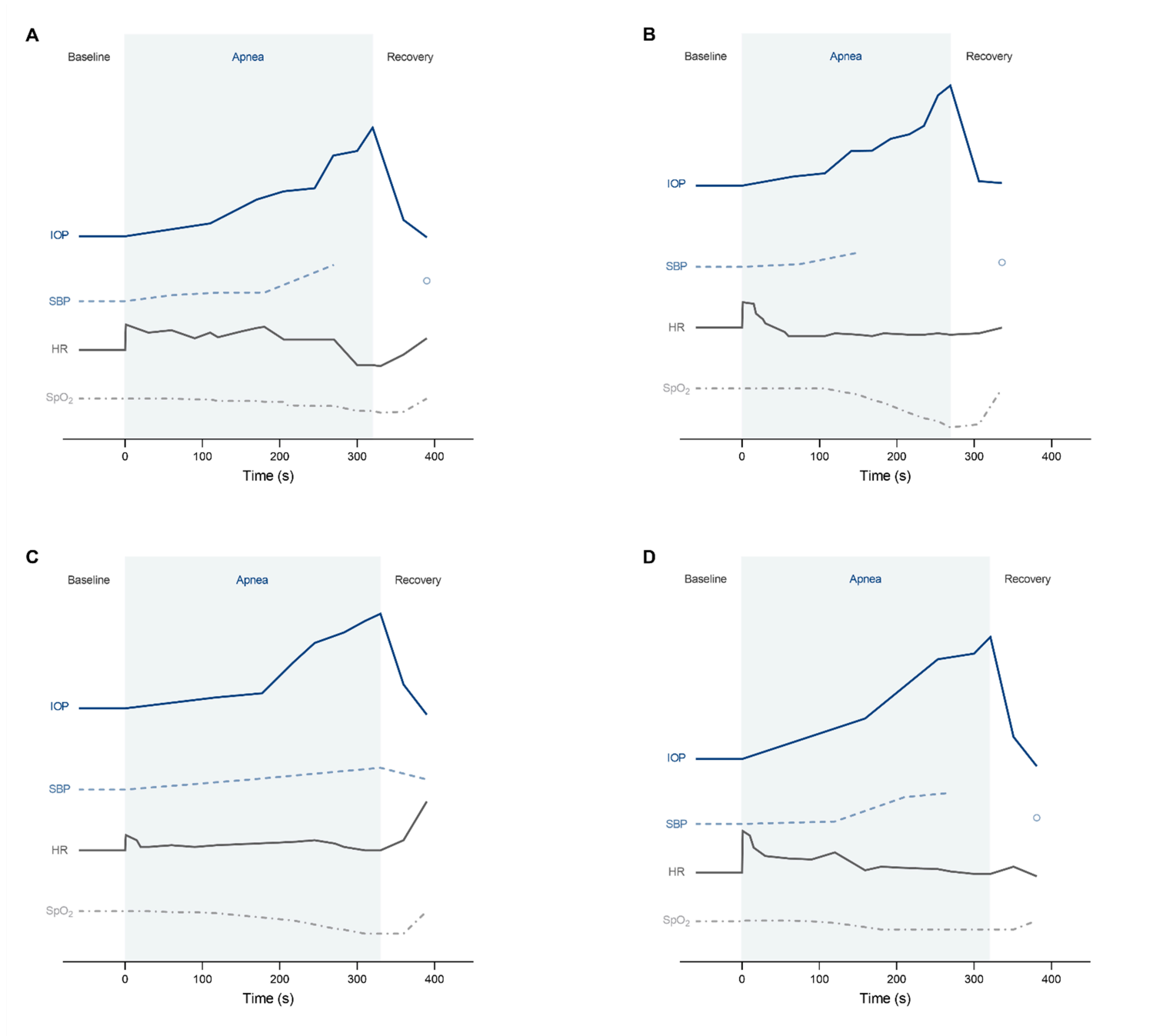


Figure 7

Representative recordings from 4 breath-hold divers during dry static apnea. Solid blue line: intraocular pressure (IOP); dashed blue line: systolic blood pressure (SBP); solid dark gray line: heart rate (HR); dash-dotted gray line: peripheral oxygen saturation (SpO₂). In cases where blood pressure could not be measured at the end of apnea, the dashed line is interrupted, and the open circle indicates the blood pressure value obtained during recovery.