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Gas exchange and pulmonary stress variations during SCUBA and breath-hold diving in open seawater

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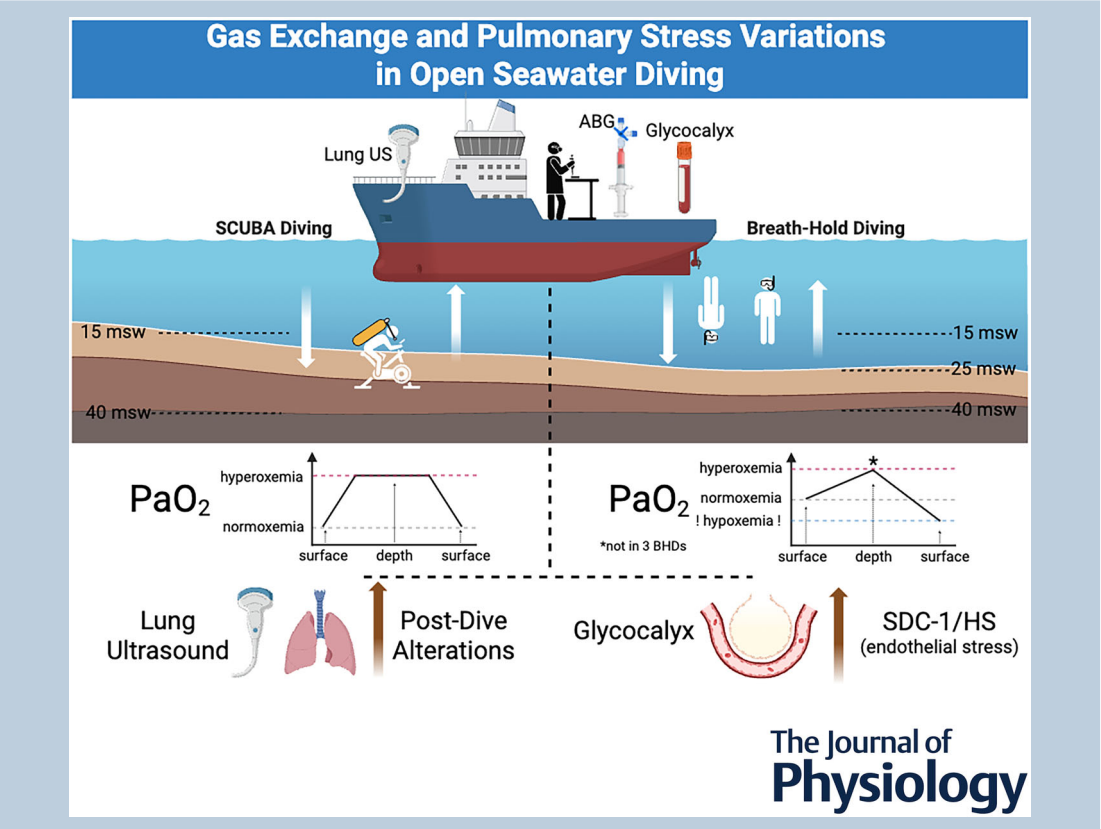

Abstract figure legend Healthy, trained divers were studied before, during and after diving in open seawater with different techniques. SCUBA divers (diving to 15 or 40 m with air; cycling at depth) and breath-hold divers (BHDs; sled-assisted dives to 15, 25 or 40 m) underwent underwater and surface arterial blood gas (ABG) sampling. In SCUBA divers, arterial partial pressure of oxygen (PaO_2) rose markedly at depth and returned to baseline on resurfacing.

Generative artificial intelligence: No generative artificial intelligence tool was used in the preparation of the present manuscript.

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In BHDs, PaO_2 increased at depth but fell to hypoxaemic values on resurfacing before breathing; a subset (three BHDs) did not display bottom hyperoxaemia. Lung ultrasound (US) showed post-dive B-lines and pleural irregularities, more pronounced after deeper dives and in breath-hold divers. Plasma syndecan-1 (SDC-1) and heparan sulfate (HS) increased post-dive, showing endothelial stress.

Abstract Understanding of pulmonary gas exchange measurements in divers at sea is incomplete. In this study, arterial blood gases (ABGs) were measured in SCUBA divers breathing compressed air and pedalling at depths of 15 or 40 m in seawater (msw). In breath-hold divers (BHDs), ABGs were obtained before, at 15, 25 or 40 msw, and at the surface before breathing. Lung ultrasound was also performed in both groups before, at 15 msw, and after all the dives. Blood syndecan-1 (SDC-1) and heparan sulfate (HS) were also measured. Among 10 SCUBA divers (one female; ages 32–57), PaO_2 increased at depth as predicted. Among 12 BHDs (three female, ages 33–62), PaO_2 rose at depth and decreased on surfacing; two participants at 15 msw and one at 25 msw did not develop bottom hyperoxaemia. Lung ultrasound was normal at 15 msw, while interstitial oedema or pleural irregularities were found after surfacing in most SCUBA divers and BHDs. In SCUBA divers, significant post-dive increases occurred in SDC-1 and HS; in BHDs, a significant increase was found in HS after the 15 and 25 msw dives, while SDC-1 increased after all depths. Compared with warm-freshwater experiments, ABG values in SCUBA divers were similar, while in BHDs relative hypoxaemia at depth was less common. Elevated levels of glycocalyx markers were consistent with endothelial stress, possibly providing a mechanism for fluid to accumulate in the pulmonary interstitium and explaining the ultrasound abnormalities.

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Key points

- The understanding of lung–environment interactions during open-sea diving remains limited.
- We integrated underwater and surface arterial blood gases, lung ultrasound and endothelial glycocalyx markers (syndecan-1, heparan sulfate) to quantify gas-exchange perturbations and lung stress in SCUBA and breath-hold divers (BHDs).
- SCUBA: arterial oxygen (PaO_2) increased at depth and returned to baseline at the surface; BHDs: PaO_2 increased at depth (except in three participants), then values fell to hypoxaemia on surfacing.
- Post-dive lung ultrasound showed subclinical interstitial oedema – from focal B-lines to diffuse patterns – and pleural irregularities more marked after deeper dives and in BHDs than in SCUBA.
- Circulating glycocalyx markers increased post-dive, consistent with endothelial stress.

Background

The lung is one of the most finely tuned organs in the human body. Its development traces back to the Devonian Period (419–358 million years ago) when aquatic ancestors ventured on land and the lungs evolved into a more efficient paired configuration to increase the surface area available for gas exchange (Cupello et al.,

Matteo Paganini is an emergency medicine physician and post-doctoral researcher, clinically active in both prehospital and hospital settings. His research focuses on carbon monoxide poisoning and water-related medicine – including environmental physiology, diving and hyperbaric medicine, with a specific interest in lung ultrasound applied to different clinical contexts – envisioning a wider application in water-related context. The rest of his research activity is devoted to improving emergency care efficiency and investigating the application of disaster medicine to mitigate climate change consequences. His aspiration is to help emergency care understand and improve its resilience, translating academic and frontline clinical experience into practical, evidence-based solutions.

2022). From the Eocene Epoch (56–34 million years ago), several groups of mammals returned to the water, with different degrees of time spent in it (Uhen, 2007): from fully aquatic cetaceans (e.g. whales, dolphins, porpoises) and sirenians (e.g. manatees and dugongs), to polar bears and otters who maintained a land-based life. Humans, part of the latter group, initially returned to water for food harvesting and the retrieval of specific goods, then for military purposes, and ultimately for sport activities, practising breath-hold diving (BHD).

Although terrestrial and marine mammals evolved different adaptations to voluntary breath-holding and immersion, these mechanisms ultimately serve the same purpose: prolonging underwater activity. In particular, gas management is optimized by redistributing blood to the thoracic and cerebral circulations, specifically to counteract oxygen (O_2) reserve exhaustion and carbon dioxide (CO_2) accumulation. In human breath-hold divers, arterial partial pressures (Pa) of O_2 and CO_2 increase during descent as the lungs progressively collapse. Conversely, during ascent, PaO_2 declines abruptly due to ongoing metabolic consumption and the reduction in ambient pressure, and $PaCO_2$ decreases as the lungs expand (Barković et al., 2023; Bosco, Rizzato, Martani et al., 2018; Bosco, Rizzato, Moon et al., 2018; Bosco et al., 2020; Fitz-Clarke, 2018; Lindholm & Lundgren, 2009; Scott et al., 2021). To overcome such limitations, humans invented the self-contained underwater breathing apparatus (SCUBA), thereby prolonging submersion time. SCUBA delivers breathing gas at the same pressure as the surrounding water, counteracting external thoracic compression and preventing lung collapse. Increased PaO_2 and $PaCO_2$ have been observed in SCUBA divers exercising underwater (Cherry et al., 2009; Fraser et al., 2011; Peacher et al., 2010). A more recent study confirmed the rise in PaO_2 at depth but found a decrease in $PaCO_2$, likely due to a lower work rate than in previous studies (Paganini et al., 2024).

The peculiar conditions encountered while diving – cold, increased pressure and impaired breathing – put significant stress on the human body, particularly the lungs, potentially altering arterial gas profiles described previously. For example, some breath-hold divers do not develop the predicted hyperoxaemia but a relative hypoxaemia at depth (Bosco, Rizzato, Martani et al., 2018), probably as a consequence of pulmonary shunt. Current hypotheses account for the accumulation of lung stress after cycles of compression–decompression, given the repetitive nature of BHD. Potential explanations include lung atelectasis or lung oedema, as confirmed by two recent studies that performed lung ultrasound scans immediately after resurfacing (Paganini, Moon et al., 2023; Yu et al., 2024). Also, inflammation and oxidative stress have been confirmed to increase in both BHD and SCUBA diving (Mrakic-Sposta, 2019; Vezzoli et al., 2024), but specific markers of pulmonary stress have not yet been investigated. Syndecan-1 (SDC-1) and heparan sulfate (HS) are key pulmonary glycocalyx markers associated with endothelial lung capillary damage and alveolar epithelium injury. Current literature shows that increases in plasma SDC-1 and HS fragments in different physiopathological models correlate with glycocalyx degradation and increased microvascular permeability (Torres Filho et al., 2016), lung oedema and lung injury (Collins et al., 2013), even suggesting a role in acute mountain sickness (Swenson et al., 2020), or with higher antioxidant defences and endothelial repair capacities in well-trained athletes (Kröpfl et al., 2021). These markers could help further explain the extent of lung stress in SCUBA or breath-hold divers, where environmental pressure and temperature changes, along with cardiovascular adaptive responses, may cause acute glycocalyx degradation.

The present study represents the first integrated approach – including arterial blood gas (ABG) analyses, lung ultrasound and markers of lung parenchymal stress – exploring pulmonary stress deriving from different diving techniques in open seawater and its consequences for gas exchange.

Methods

Participants

Well-trained, healthy SCUBA divers and breath-hold divers were considered eligible for the study and recruited through existing advertising networks in diving clubs and pools. Inclusion criteria were age $\geq$18 years and no history of orthopaedic, cardiovascular, liver, renal or metabolic disorders. Exclusion criteria were pregnancy, allergy to local anaesthetics, coagulation abnormalities, alterations in the arterial vascularization of upper limbs, or vasculopathy.

Experimental design and diving plan

The original experimental protocol and its addendum, covering all procedures, were approved by the Human Ethics Committee of the Department of Biomedical Science at the University of Padova (No. HEC-DSB/03-18; addendum: November 2022) and adhered to the principles of the *Declaration of Helsinki*.

Meetings with eligible participants were held approximately 30 days before each experimental session to explain the protocol, collect written informed consent and conduct preliminary medical screenings. Such meetings also helped the included subjects become familiar with the procedures and ensure their proper execution.

The experiments were conducted in three stages between June 2024 and June 2025 at two sites in open

seawater: the Gulf of Naples and the Tremiti Islands, Italy. Such locations were chosen based on: safety of the diving sites, stability of water temperature, availability of ground-based resources, availability of a diving boat large enough to set up a laboratory, and the presence of a support boat to stop maritime traffic in case fast transportation was needed. Ambient temperature ranged from 30°C to 34°C, with a sea state ranging from generally calm to slightly rough. Water temperature at target depths ranged from 17° to 25 ± 0.5°C across all stages and locations.

To ensure healthy conditions, participants were re-screened medically on the day of the experiment. Then, age, sex, height and weight were recorded. All participants could opt out or refuse any experimental procedure at any time. The safety of both participants and researchers was considered of utmost importance; therefore, emergency treatment and mitigation plans were established before running each experiment, and each participant was asked to report any symptoms after the end of experiments.

SCUBA divers. SCUBA divers used an open-circuit system, wore a standard buoyancy compensator device, a standard pair of fins, a face mask, a 5 mm neoprene wetsuit with one sleeve opened to allow placement of the arterial cannula, and carried a single 15 L steel tank filled with compressed air. After reaching the predefined depth in pairs – 15 m of seawater (msw) or 40 msw – in about 2 min, the divers were helped to remove their fins and pedalled on a submerged bike (OKEO Srl, Genova, Italy), set at 100 W (plus an estimated 50 W to move legs underwater = 150 W total effort) at a rate of 60 rpm for 10 min (Borg, 1982). The diving assistant checked the target depth and time on each diver's diving computer, adjusting the bike's saddle to accommodate height differences among the participants. The total bottom time was approximately 12 min for both dives, allowing for fin removal, positioning, pedalling and fin wearing. All the SCUBA divers ascended at max 3 m/min, to perform a final safety stop of 3 min at 5 msw. No decompression was performed in the 15 msw dive. For the 40 msw dive, SCUBA divers followed the indications of the diving computers, using a Buhlman 35/75 inbuilt algorithm, strictly monitoring the slow ascent and performing deco stops as needed. A schematic representation of the SCUBA dives profiles is provided in Fig. *A*1. Safety measures included two professional instructors accompanying the couples underwater, a physician underwater to perform procedures, and medical personnel onboard the experimental boat with oxygen and emergency equipment; a support vessel for emergency evacuation was also readily available. Some participants consented to perform both depths in two different days; others instead performed only one of the two depths.

Breath-hold divers. Breath-hold divers wore a 5 mm neoprene two-piece wetsuit, with one sleeve opened to allow placement of the arterial cannula, and a standard face mask. During baseline sampling and navigation to the diving spot, they were asked to refrain from any effort, rest in the shade and maintain adequate hydration. They performed a standardized warm-up consisting of surface exercises (static breath-hold alternated with relaxed and controlled breathing), then four breath-hold dives – each separated by 5–10 min. Before the experimental dive, the athletes performed a 5 ming controlled, non-forced ventilation at 8–9 breaths/min (inspiratory-to-expiratory ratio of 1:2 with normal tidal volumes as perceived by each diver); they were not allowed to perform lung packing. The experimental dives to 15, 25, or 40 msw were performed using the sled-assisted technique, descending with the head toward the bottom and ascending with the head toward the surface. Safety measures included a professional BHD instructor accompanying each diver, SCUBA divers at the bottom and 10 m below the surface, and medical personnel onboard the experimental boat with oxygen and emergency equipment; a support vessel for emergency evacuation was also readily available.

Arterial blood gases

As previously described (Bosco, Rizzato, Martani et al., 2018, Bosco et al., 2020), the radial artery of the non-dominant limb was cannulated with a 20 G (3 Fr) Teflon catheter under local anaesthesia after verifying radial and ulnar artery patency through an Allen test (Fuhrman et al., 1992) and using a 'catheter over the needle' technique. The cannula was fixed to the skin by an adhesive band and connected to a circuit with a Luer Lok-type fitting to avoid leakages, differently assembled for breath-hold divers (Bosco, Rizzato, Martani et al., 2018, Bosco et al., 2020; Paganini, Moon et al., 2023) or SCUBA divers (Paganini et al., 2024). A safety briefing before the dives trained participants to turn the main stopcock or to apply direct, firm pressure to the vascular access site to prevent critical bleeding in the event of disconnections or accidental removal.

As depicted in Fig. 1, a first arterial blood sample (baseline) was drawn from both groups a few minutes after arterial access placement, while at rest, sitting on a stretcher (step A), to avoid procedural stress and possible hyperventilation as confounding factors.

In the SCUBA diving group, arterial blood samples were obtained (Fig. 1): at 15 msw before (step B) and after (step C) pedalling, then at the surface, as soon as they got out of the water (step D); at 40 msw before (step E) and after (step F) pedalling, then at the surface, as soon as they got out of the water (step G).

In the breath-hold divers' group, arterial blood samples were obtained (Fig. 1): after the pre-dive standardized preparation and after putting the face underwater (step B); at 15 msw (step C); after the 15 msw dive, face still submerged, before breathing (step D); at 25 msw (step E); after the 25 msw dive, face still submerged, before breathing (step F); at 40 msw (step G); after the 40 msw dive, face still submerged, before breathing (step H).

Blood collection sampling underwater slightly differed between groups, following previous methods (Bosco, Rizzato, Martani et al., 2018, Bosco et al., 2020; Paganini, Moon et al., 2023, Paganini et al., 2024). Of note, bottom breath-hold divers' samples were detached immediately when the diver arrived at the surface to perform the surface sample. Instead, for the SCUBA divers, bottom samples were delivered through a water-filled bottle, lifted by an inflated surface marker buoy connected to a retrieval line; additional stopcocks between the main line and the syringes prevented contamination during detachment.

At the end of the experiments, the arterial access was removed aseptically, and a compression bandage was applied for 2 h. The insertion site was then monitored over the next 2 days for complications.

Assuming that ABGs from normal individuals are stable for more than half an hour at room temperature and that this time can be extended to several hours if preserved on ice (Knowles et al., 2006), the retrieved blood samples were stored on ice at arrival and processed within 5 min in the blood gas analyser present on site (Epoc Blood Analysis System / NXS Host, model PD470SH-B, Siemens Healthcare AG, Erlangen, Germany). The following values were tracked: pH; PaO_2; and $PaCO_2$.

Lung ultrasound

Thoracic ultrasound was performed on both groups of divers at baseline, underwater at 15 msw, and immediately after resurfacing from 15 msw, using two identical, commercially available ultrasound devices (Versana Active, GE Healthcare, United States) – one of which was marinized (Paganini, Cantarella et al., 2023) – equipped with a linear probe (8–13 MHz). Scans included the anterior and lateral aspects of each lung. Specifically, sonographers investigated intercostal spaces in a longitudinal plane, dividing the chest along four subsequent lines (medially to laterally: the parasternal, midclavicular, anterior axillary and posterior axillary) on the left and then on the right chest. Each 'line' was recorded separately, in the same order as specified above, to ensure data collection consistency. Scans at depth took 30 to 60 s, with diver assistants helping with wetsuit opening and subject positioning. Two expert sonographers performed and recorded ultrasound scans, which were then blindly reviewed by two other experts in the field to ensure the validity of the interpretation.

Pulmonary glycocalyx markers

Venous blood was drawn using the standard venipuncture technique from antecubital veins, both before and after the experimental dive, within 15 min of exiting the water. Plasma levels of pulmonary glycocalyx markers – SDC-1 and HS – were measured using human ELISA kits according to the manufacturer's instructions (Fine Test, Wuhan, China; items No. EH0278 and No. EH4010, respectively). The determinations were assessed in

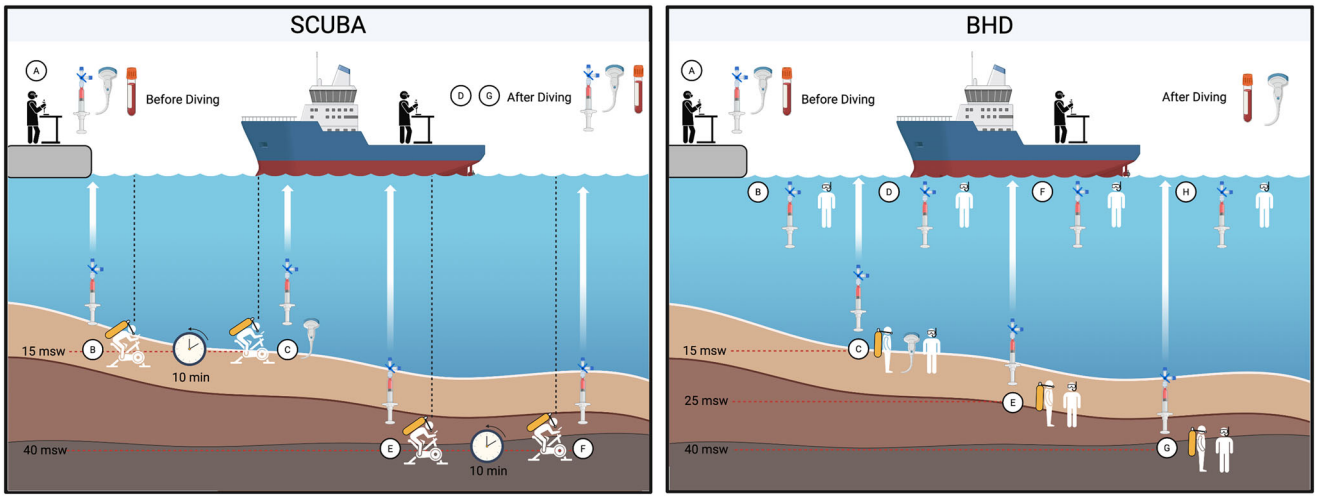

Figure 1.
Schematic representation of the experimental protocol for SCUBA (*A*) and breath-hold divers (*B*)

duplicate, ensuring the inter-assay coefficient of variation was in the range indicated by the manufacturer, and read by a microplate reader spectrophotometer (Infinite M200, Tecan Group Ltd., Männedorf, Switzerland).

Venous gas emboli detection

Venous gas emboli (VGE) monitoring was performed through 2D echocardiography with the same apparatus, equipped with a sector probe (1–4 MHz) in apical 4-chamber view, while on left-lateral decubitus once on board the vessel and within 10 min of resurfacing. VGE were graded according to the Eftedal-Brubakk scale as follows: 0 – No bubbles; 1 – Occasional bubbles; 2 – At least one bubble every four cardiac cycles; 3 – At least one bubble every cardiac cycle; 4 – At least one bubble per cm^2 in every image; 5 – White out, single bubbles cannot be discriminated (Eftedal & Brubakk, 1997).

Data analysis

All data obtained were coded onto a master sheet using a Microsoft Office Excel spreadsheet (Version 2016, Microsoft Corporation, Redmond, WA). Where appropriate, the Shapiro–Wilk test was used to determine the distribution of quantitative data. Then, means and standard deviations (SD) were used in normally distributed samples, whereas medians and interquartile ranges (IQR; 25−75%) were used in non-normal distributions. A paired *t* test was used to test for differences between normally distributed groups; the Wilcoxon test for dependent samples was used for non-normal distributions. Significance was set at $P < 0.05$. Due to high inaccuracy, the Wilcoxon test was not performed in the case of a non-normal distribution with less than six pairs available, and only trends were described. Pulmonary glycocalyx markers were analysed with absolute values and after transformation to relative values, taking the first measurement T0 as 100%, allowing an appreciation of the magnitude of change. All statistical analyses were performed using R Statistical Software (version 3.6; R Foundation for Statistical Computing, Vienna, Austria).

Results

Ten SCUBA divers (one female) and 12 breath-hold divers (three females) participated in the study without reporting complications (characteristics reported in Table *A*1). Basal assessments, namely ABGs (step A), venous blood samples (T0), and lung ultrasound scans were all within normal ranges in both groups. None of the participants reported any symptoms after the experiments.

SCUBA divers

In SCUBA divers, ABG and lung ultrasound were obtained in six dives to 15 msw and eight dives to 40 msw (one without ABGs). In bottom samples, PaO_2 significantly increased ($P < 0.001$) from 98 ± 7 mmHg in step A to 318 ± 20 and 317 ± 27 mmHg in steps B and C at 15 msw, and with a greater magnitude at 40 msw, reaching 497 ± 26 and 505 ± 32 mmHg in steps E and F (Fig. 2 – SCUBA; Table 1). When resurfacing from both depths, PaO_2 returned to values statistically comparable to baseline (D: 101 ± 7 mmHg; G: 101+11 mmHg), with a significant drop compared with the respective after-pedalling deep sample (both $P < 0.001$). No statistically significant differences in PaO_2 were observed between the samples obtained at depth before and after pedalling. pH and $PaCO_2$ values fell within normal ranges, with no significant variations compared with baseline, except for samples obtained after pedalling at 40 msw and when resurfacing from 40 msw, compared with those after pedalling (Table 1).

Lung ultrasound at 15 msw revealed normal lung sliding due to ongoing respiratory activity, without pulmonary alterations. At the surface, interstitial oedema was found in four of six participants after the 15 msw dives. After the 40 msw dive, five of eight participants showed pulmonary involvement: interstitial oedema isolated to one lung base in three, one with focal involvement of the upper anterior lung, and another with diffused oedema and pleural irregularities. None of the pulmonary alterations were correlated with surface hypoxaemia (Table 2).

Eight dives to 15 msw and another eight to 40 msw provided samples for SDC-1 and HS. From baseline to 15 and 40 msw, respectively, SDC-1 significantly increased from 22.40 ± 4.51 ng/mL to 27.74 ± 8.34 ng/mL and 38.62 ± 12.48 ng/mL and HS raised from 4.99 ± 1.01 μM/mL to 6.16 ± 1.18 μM/mL and 7.28 ± 1.75 μM/mL (Fig. 3*A*,*a*,*B*,*b*). Echocardiography was performed on a subset of participants, identifying one diver with VGE grade 1 after the 15 msw dive (out of six divers), and one diver with VGE grade 2 after the 40 msw dive (out of six divers).

Breath-hold divers

The 12 included breath-hold divers provided data for nine dives to 15 msw, eight dives to 25 msw (one without ABG and lung ultrasound), and four dives to 40 msw. Dive durations are reported in Table *A*2.

The PaO_2 was within normal limits at baseline (A; 94.4 ± 5 mmHg) and significantly rose to 101 ± 4 mmHg after the pre-dive preparation (B). Both these samples significantly differed from the others, showing an overall increase at depth (C: 171 (161–187) mmHg; E: 202

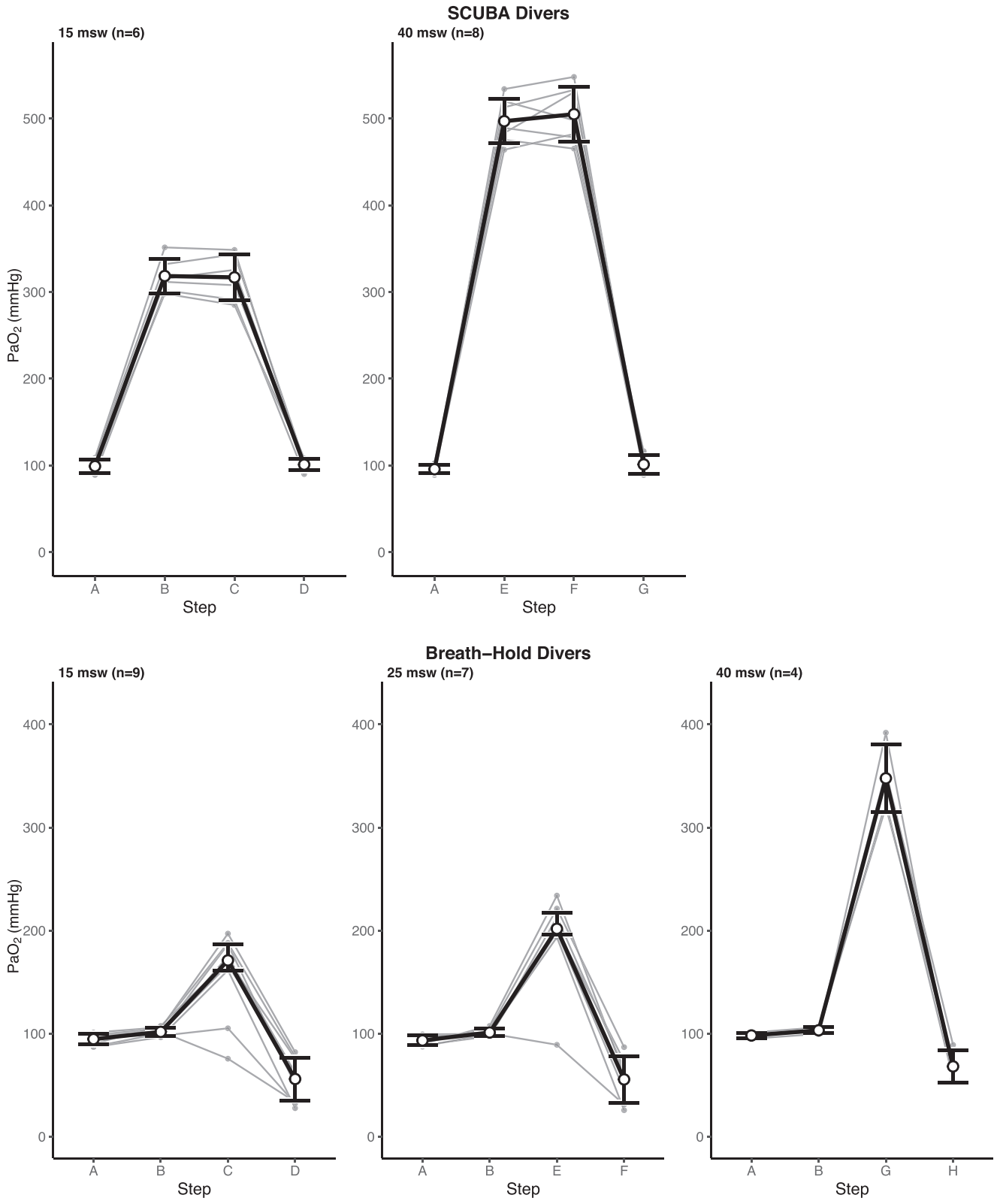

Figure 2. Arterial partial pressures of oxygen (PaO_2) variations in divers at different steps
In SCUBA divers: baseline (A); at 15 m of seawater (msw) before (B) and after (C) pedalling; at surface from 15 msw (D); at 40 msw before (E) and after (F) pedalling; at surface from 40 msw (G). In breath-hold divers: baseline (A); pre-dive, face underwater (B); at 15 msw (C); after the 15 msw dive, face underwater (D); at 25 msw (E); after the 25 msw dive, face underwater (F); at 40 msw (G); after the 40 msw dive, face underwater (H).

Table 1. SCUBA divers' arterial blood gases

	Step	SCUBA 1	SCUBA 2	SCUBA 3	SCUBA 4	SCUBA 5	SCUBA 6	SCUBA 7	SCUBA 8	SCUBA 9	SCUBA 10	Mean ± SD
pH	**A**	7.41	7.41	7.39	7.42	7.45	7.44	7.46	7.40	7.43	7.46	7.43 ± 0.02
	B	—	—	7.32	7.37	—	—	7.46	7.38	7.44	7.57	7.42 ± 0.09
	C	—	—	7.35	7.43	—	—	7.49	7.36	7.42	7.46	7.42 ± 0.06
	D	—	—	7.40	7.46	—	—	7.49	7.47	7.40	7.48	7.45 ± 0.04
	E	7.43	7.42	—	—	7.32	7.40	—	7.38	7.43	7.44	7.40 ± 0.04
	F	7.36	7.35	—	—	7.38	7.42	—	7.36	7.43	7.41	7.39 ± 0.03 *
	G	7.40	7.36	—	—	7.39	7.41	—	7.38	7.40	7.45	7.40 ± 0.03 *
PaO_2 (mmHg)	**A**	98	103	93	109	91	93	108	99	97	89	98 ± 7
	B	—	—	316	351	—	—	332	312	298	302	318 ± 20 *
	C	—	—	326	349	—	—	343	308	285	291	317 ± 27 *
	D	—	—	100	98	—	—	105	108	105	90	101 ± 7 † ‡
	E	475	483	—	—	464	489	—	520	513	534	497 ± 26 * † ‡ §
	F	465	530	—	—	482	478	—	498	533	548	505 ± 32 * † ‡ §
	G	116	95	—	—	115	94	—	98	101	89	101 ± 11 † ‡ ¶ •
$PaCO_2$ (mmHg)	**A**	41	39	41	39	37	35	39	42	39	40	39 ± 2
	B	—	—	48	40	—	—	41	42	36	30	39 ± 6
	C	—	—	45	38	—	—	38	44	38	42	41 ± 3
	D	—	—	40	36	—	—	37	37	40	38	38 ± 2
	E	38	35	—	—	44	40	—	44	39	43	40 ± 4 ‡
	F	40	43	—	—	44	43	—	43	40	45	43 ± 2 *
	G	38	41	—	—	37	38	—	40	37	40	39 ± 1 •

(*Continued*)

Table 1. (Continued)

	Step	SCUBA 1	SCUBA 2	SCUBA 3	SCUBA 4	SCUBA 5	SCUBA 6	SCUBA 7	SCUBA 8	SCUBA 9	SCUBA 10	Mean ± SD
pH *P* values												
	A	**B**	**C**	**D**	**E**	**F**	**G**					
A	—	0.928	0.416	0.210	0.244	**0.004**	**0.005**					
B		—	0.769	0.521	0.346	0.286	0.267					
C			—	0.186	0.690	0.463	0.822					
D				—	0.488	0.354	0.260					
E					—	0.382	0.776					
F						—	0.268					
G							—					
PaO_2 *P* values												
	A	**B**	**C**	**D**	**E**	**F**	**G**					
A	—	**<0.001**	**<0.001**	0.581	**<0.001**	**<0.001**	0.244					
B		——	0.724	**<0.001**	**0.001**	**0.007**	**<0.001**					
C			—	**<0.001**	**0.002**	**0.008**	**0.001**					
D				—	**<0.001**	**0.002**	0.191					
E					—	0.405	**<0.001**					
F						—	**<0.001**					
G							—					
$PaCO_2$ *P* values												
	A	**B**	**C**	**D**	**E**	**F**	**G**					
A	—	0.810	0.387	0.080	0.485	**0.042**	0.657					
B		—	0.588	0.590	0.250	0.276	0.509					
C			—	0.088	**0.046**	0.292	0.156					
D				—	0.293	0.215	0.705					
E					—	0.092	0.332					
F						—	**0.001**					
G							—					

Arterial blood gases obtained from SCUBA divers, showing values of pH, arterial partial pressures of oxygen (PaO_2) and of carbon dioxide ($PaCO_2$). A: At rest, out of water; at depth, 15 msw, before (B) and after (C) pedalling; D: after the 15 msw dive, out of water; at depth, 40 msw, before (E) and after (F) pedalling; G: after the 40 msw, out of water. Measures of distribution for each step are reported as means ± standard deviation. Statistically significant variations from: * A; † B; ‡ C; § D; ¶ E; • F. Exact *P* values are reported at the end of the table, with statistically significant variations in bold.
Exact *P* values. Statistically significant variations paired *t* test in bold.

Table 2. Arterial partial pressure of oxygen (PaO_2, in mmHg) from arterial blood gases and lung ultrasound findings at different observation points in SCUBA divers. Underwater lung ultrasound was performed at 15 msw, not at 40 msw.

	A: Baseline		B: At 15 msw, before pedalling	C: At 15 msw, after pedalling		D: Surface after 15 msw	
SCUBA diver #	ABG PaO_2	Lung US	ABG PaO_2	ABG PaO_2	Lung US	ABG PaO_2	Lung US
3	93	NTR	316	326	NTR	100	NTR
4	109	NTR	351	349	NTR	98	3 B-lines in both lung bases
7	108	NTR	332	343	NTR	105	3 B-lines in both lung bases
8	99	NTR	312	308	NTR	108	3–6 B-lines in both lung bases
9	97	NTR	298	285	NTR	105	NTR
10	89	NTR	302	291	NTR	90	3 B-lines in one lung base
SCUBA Diver #	**A: Baseline**		**E: At 40 msw, before pedalling**	**F: At 40 msw, after pedalling**		**G: Surface after 40 msw**	
	ABG PaO_2	**Lung US**	**ABG PaO_2**	**ABG PaO_2**		**ABG PaO_2**	**Lung US**
1	98	NTR	475	465		116	NTR
2	103	NTR	483	530		95	3 B-lines diffused + pleural irregularities in anterior bilateral
5	91	NTR	464	482		115	3 B-lines in one lung base
6	93	NTR	489	478		94	NTR
7	108	NTR	—	—		—	Focal confluent B-lines in one anterior segment
8	99	NTR	520	498		98	3 B-lines in one lung base
9	97	NTR	513	533		101	3–6 B-lines in one lung base
10	89	NTR	534	548		89	NTR

ABG: arterial blood gases; msw: metres of seawater; NTR: nothing to report; US: ultrasound.

(196–217) mmHg; G: 348 ± 33 mmHg) and hypoxaemia back at the surface (D: 56 ± 21 mmHg; F: 56 ± 23 mmHg; H: 68 ± 16 mmHg) (Fig. 2 Breath-hold divers; Table 3). The PaO_2 rapidly dropped when resurfacing for each bottom/surface couple comparison (C–D; E–F; G–H). Of note, three participants did not develop the predicted hyperoxaemia at depth: two at 15 msw (No. 4: 105.1 mmHg; No. 11: 76 mmHg) and one at 25 msw (No. 3: 89 mmHg). Statistically significant variations in pH were found between pre-diving (B: 7.45 ± 0.05 mmHg) and the baseline (A: 7.41 ± 0.02 mmHg), 15 msw (C: 7.40 ± 0.04 mmHg), surface after 15 msw (D: 7.40 ± 0.03 mmHg), and bottom 40 msw (G: 7.43 ± 0.03 mmHg) samples, but remained within normal ranges – thus physiologically insignificant (Table 3). $PaCO_2$ values did not exhibit significant variations across the steps, except for the sample obtained at the surface after 15 msw (D: 42 ± 4 mmHg), which was higher than those measured at A (37 ± 1 mmHg), B (36 ± 4 mmHg), C (38 ± 6 mmHg) and F (35 ± 8 mmHg). A trend can be noted when resurfacing from 25 msw and 40 msw, decreasing from the respective bottom sample, but not in the 15 msw dive.

Lung ultrasound did not show any alterations at 15 msw. Different degrees of lung parenchymal alterations were instead found after almost all the dives at the surface, as follows: eight out of nine divers after the 15 msw dive had interstitial oedema in lung bases, with two manifesting pleural irregularities; all the seven divers after the 25 msw dive presented with interstitial oedema progressing from the bases to the anterior and upper lung quadrants, associated with pleural irregularities in four of them; three out of four divers after the 40 msw dive showed interstitial oedema involving both anterior and basal quadrants with diffused pleural irregularities (Table 4).

From baseline to 15 msw, 25 msw or 40 msw, respectively, SDC-1 demonstrated a significant increase from 22.86 ± 4.89 ng/mL to 25.91 ± 5.46 ng/mL, 32.42 ± 4.28 ng/mL and 39.50 ± 8.97 ng/mL. HS significantly rose from 4.47 ± 0.91 μM/mL to 5.11 ± 0.95 μM/mL and 5.81 ± 1.34 μM/mL, showing a rising trend to 6.38 ± 2.41 μM/mL in the deeper dive despite not reaching statistical significance, likely due to the small sample (Fig. 3*C, c, D, d*). Echocardiography identified one diver with VGE grade 3 after the 25 msw dive (out of five divers), and one diver with VGE grade 3, respectively, after the 40 msw dive (performed on four divers in this campaign).

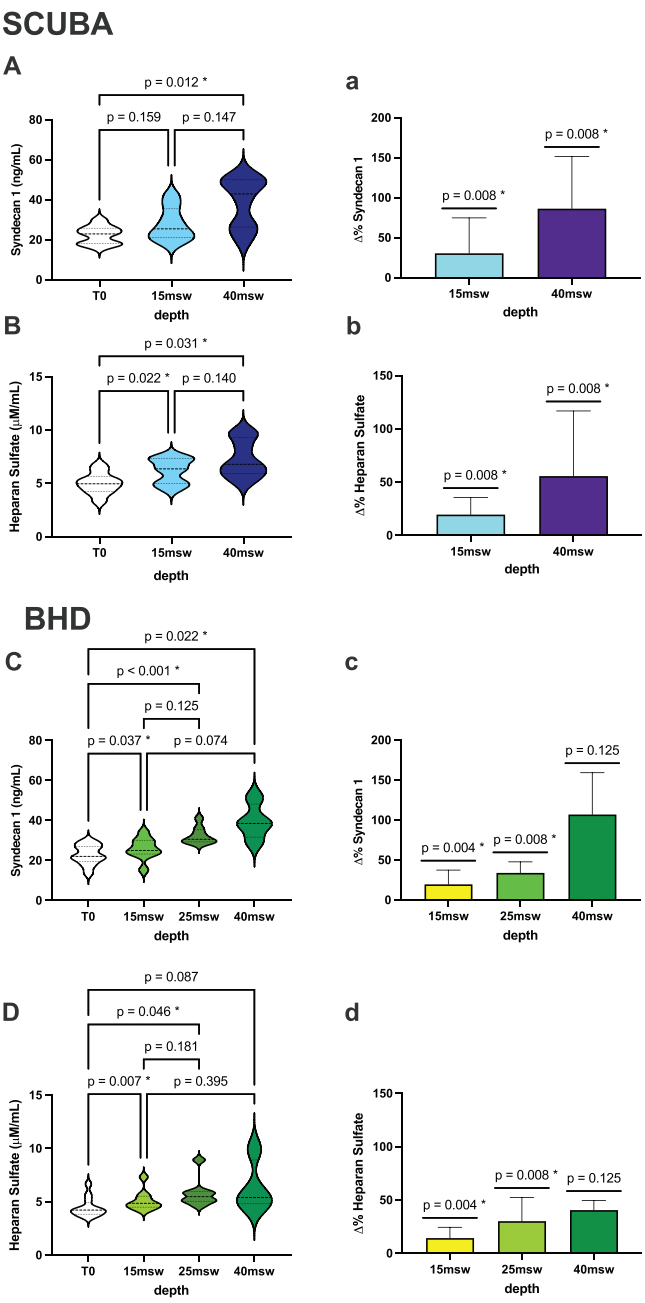

Figure 3.
Absolute values and relative-variation (Δ%), before (T0) and after diving to different depths (in metres of seawater (msw)), in: *Aa*, syndecan-1 and *Bb*, heparan sulfate in SCUBA divers (n = 8); *Cc*, syndecan-1 and *Dd*, heparan sulfate in breath-hold divers (BHD), (15 msw: n = 9; 25 msw: n = 8; 40 msw: n = 4). Statistically significant differences are identified with *

Discussion

Building on previous studies that only focused on ABGs in breath-hold divers in real underwater environments (Barković et al., 2023; Bosco, Rizzato, Martani et al., 2018, Bosco et al., 2020; Scott et al., 2021), lung ultrasound was recently added to improve the understanding of lung stress (Paganini, Moon et al., 2023); SCUBA divers were also investigated with ABGs (Paganini et al., 2024). By integrating ABGs, lung ultrasound and pulmonary stress

Table 3. Breath-hold divers' arterial blood gases

	Step	BHD 1	BHD 2	BHD 3	BHD 4	BHD 5	BHD 6	BHD 7	BHD 8	BHD 9	BHD 10	BHD 11	BHD 12	
pH	**A**	7.40	7.42	7.42	7.40	7.40	7.41	7.43	7.40	7.43	7.39	7.39	7.43	7.41 ± 0.02 †
	B	7.42	7.51	7.45	7.40	7.43	7.42	7.40	7.41	7.48	7.51	7.49	7.53	7.45 ± 0.05
	C	7.36	7.43	7.38	7.45	7.37	—	—	—	7.39	7.42	7.36	7.43	7.40 ± 0.04 † •
	D	7.36	7.42	7.38	7.42	7.44	—	—	—	7.37	7.44	7.35	7.43	7.40 ± 0.03 † •
	E	7.32	7.48	7.33	7.32	—	7.48	7.31	7.38	—	—	—	—	7.33 (7.32–7.43)
	F	7.38	7.49	7.42	7.49	—	7.47	7.34	7.35	—	—	—	—	7.42 ± 0.07
	G	—	—	—	—	—	—	—	—	7.45	7.45	7.38	7.42	7.43 ± 0.03 †
	H	—	—	—	—	—	—	—	—	7.48	7.51	7.37	7.39	7.44 ± 0.07
PaO_2 (mmHg)	**A**	90	97	87	98	87	89	99	93	101	99	98	95	94 ± 5
	B	107	101	100	98	96	97	100	104	106	105	102	100	101 ± 4 *
	C	171	167	187	105	161	—	—	—	188	197	76	175	171 (161–187) * †
	D	76	62	50	32	28	—	—	—	78	82	34	62	56 ± 21 * † ‡ §
	E	234	193	89	213	—	202	198	221	—	—	—	—	202 (196–217) * †
	F	61	26	31	66	—	73	45	87	—	—	—	—	56 ± 23 * † ¶
	G	—	—	—	—	—	—	—	—	354	320	325	392	348 ± 33 * †
	H	—	—	—	—	—	—	—	—	89	72	56	55	68 ± 16 * † §
$PaCO_2$ (mmHg)	**A**	37	39	37	37	38	39	37	38	35	38	37	36	37 ± 1 #
	B	35	30	38	36	40	41	39	37	31	32	32	39	36 ± 4 #
	C	43	31	42	30	34	—	—	—	40	44	45	39	39 ± 6 #
	D	44	37	49	38	38	—	—	—	43	45	40	45	42 ± 4
	E	54	26	53	49	—	25	47	45	—	—	—	—	47 (35–51)
	F	40	28	37	31	—	24	44	42	—	—	—	—	35 ± 8 #
	G	—	—	—	—	—	—	—	—	38	36	47	46	42 ± 6 †
	H	—	—	—	—	—	—	—	—	33	29	48	44	39 ± 9
pH *P* values														
	A	**B**	**C**	**D**	**E**	**F**	**G**	**H**						
A	—	**0.009**	0.421	0.588	0.156 **	0.791	0.406	0.527						
B		—	**0.004**	**0.005**	0.078 **	0.669	**0.028**	0.185						
C			—	0.796	***	**0.012**	0.122	0.301						
D				—	***	**0.026**	0.206	0.307						
E					—	0.219 **	NA	NA						

(*Continued*)

Table 3. (Continued)

	Step	BHD 1	BHD 2	BHD 3	BHD 4	BHD 5	BHD 6	BHD 7	BHD 8	BHD 9	BHD 10	BHD 11	BHD 12
F						—	NA	NA					
G							—	0.657					
H								—					
PaO_2 *P* values													
	A	**B**	**C**	**D**	**E**	**F**	**G**	**H**					
A	—	**<.001**	**0.012** **	**<.001**	**0.016** **	**0.005**	**<.001**	**0.022**					
B		—	**0.012** **	**<.001**	**0.031** **	**0.002**	**<.001**	**0.013**					
C			—	**0.004** **	***	***	***	***					
D				—	***	0.583	**<.001**	0.617					
E					—	**0.016** **	NA	NA					
F						—	NA	NA					
G							—	**<.001**					
H								—					
$PaCO_2$ *P* values													
	A	**B**	**C**	**D**	**E**	**F**	**G**	**H**					
A	—	0.168	0.505	**0.013**	0.469 **	0.436	0.184	0.720					
B		—	0.148	**0.002**	0.297 **	0.657	**0.042**	0.313					
C			—	**0.040**	***	0.162	0.948	0.506					
D				—	***	**0.022**	0.763	0.464					
E					—	0.051 **	NA	NA					
F						—	NA	NA					
G							—	0.158					
H								—					

Arterial blood gases obtained from breath-hold divers, showing values of pH, arterial partial pressures of oxygen (PaO_2) and of carbon dioxide ($PaCO_2$). A: At rest, out of water; B: after the pre-dive standardized preparation, face underwater; C: at 15 msw; D: after the 15 msw dive, face still submerged; E: at 25 msw; F: after the 25 msw dive, face still submerged; G: at 40 msw; H: after the 40 msw dive, face still submerged. Measures of distribution for each step are reported as means ± standard deviation or as medians (IQR 25–75%). Statistically significant variations from: * A; † B; ‡ C; # D; ¶ E; • F; § G. Exact *P* values are reported at the end of the table, with statistically significant variations in bold. Exact *P* values. Statistically significant variations in bold. Statistical tests details: **: Wilcoxon's test; ***: Wilcoxon's test not performed due to <6 pairs available; NA: paired comparisons not possible in E and F *versus* G and H due to unavailable data; paired *t* test if not noted.

Table 4. Arterial partial pressure of oxygen (PaO_2, in mmHg) from arterial blood gases and lung ultrasound findings at different observation points in breath-hold divers. Underwater lung ultrasound was performed at 15 msw, not at 25 and 40 msw. Subjects who did not develop the predicted hyperoxaemia at depth are noted with *.

BH diver #	A: Baseline		B: pre-dive standardized preparation, face underwater	C: At 15 msw		D: Resurfacing from 15 msw	
	ABG PaO_2	Lung US	ABG PaO_2	ABG PaO_2	Lung US	ABG PaO_2	Lung US
1	90	NTR	107	171	NTR	76	3 B-lines in both lung bases
2	97	NTR	101	167	NTR	62	3 B-lines in anterobasal right lung
3	87	NTR	100	187	NTR	50	NTR
4	98	NTR	98	105 *	NTR	32	3 B-lines in both lung bases
5	87	NTR	96	161	NTR	28	3 B-lines in both lung bases
9	101	NTR	106	188	NTR	78	3–6 B-lines in both lung bases + pleural irregularities in left lung base
10	99	NTR	105	197	NTR	82	3 B-lines in one lung base
11	98	NTR	102	76 *	NTR	34	3 B-lines in both lung bases + pleural irregularities in right lung base
12	95	NTR	100	175	NTR	62	3 B-lines in right lung base, 3–6 in left lung base
BH diver #	**A: Baseline**		**B: pre-dive standardized preparation, face underwater**	**E: At 25 msw**		**F: Resurfacing from 25 msw**	
	ABG PaO_2	**Lung US**	**ABG PaO_2**	**ABG PaO_2**		**ABG PaO_2**	**Lung US**
1	90	NTR	107	234		61	Diffused 3 B-lines + pleural irregularities in left lung base
2	97	NTR	101	193		26	3 B-lines in anterior right and left lung base
3	87	NTR	100	89 *		31	3 B-lines in both lung scans, confluent in anterior right and basal left

(*Continued*)

Table 4. (Continued)

BH diver #	A: Baseline		B: pre-dive standardized preparation, face underwater	C: At 15 msw		D: Resurfacing from 15 msw	
	ABG PaO_2	Lung US	ABG PaO_2	ABG PaO_2	Lung US	ABG PaO_2	Lung US
4	98	NTR	98	213		66	3 B-lines in all right lung
6	89	NTR	97	202		73	3 B-lines + pleural irregularities in both lung bases
7	99	NTR	100	198		45	3 B-lines in both lung bases + pleural irregularity with 3 B-lines in anterior right lung
8	93	NTR	104	221		87	3 B-lines + multiple pleural irregularities in both anterobasal lungs
BH diver #	**A: Baseline**		**B: pre-dive standardized preparation, face underwater**	**G: At 40 msw**		**H: Resurfacing from 40 msw**	
	ABG PaO_2	**Lung US**	**ABG PaO_2**	**ABG PaO_2**		**ABG PaO_2**	**Lung US**
9	101	NTR	106	354		89	NTR
10	99	NTR	105	320		72	3 B-lines + pleural irregularities diffused in both anterobasal lungs
11	98	NTR	102	325		56	3 B-lines + pleural irregularities in both lung bases
12	95	NTR	100	392		55	3 B-lines in both bases + pleural irregularities in right anterobasal lung

ABG: arterial blood gases; BH: breaht-hold; NTR: nothing to report; msw: metres of seawater; US: ultrasound.

biomarkers, we were able to better describe how the lungs interact with the underwater environment.

First, the gas exchange capacity of SCUBA divers' lungs did not seem to be impaired when diving at different depths. The PaO_2 values obtained in the present study behaved similarly to those of previous experiments performed with simulated wet dives in hyperbaric chambers (Cherry et al., 2009; Fraser et al., 2011; Freiberger et al., 2016; Peacher et al., 2010), and by our group in a deep pool in warm water showing hyperoxaemia at depth (Di Pumpo et al., 2023), stable before and after pedalling on a submerged bike (Paganini et al., 2024), and normal values back at the surface. In parallel, lung ultrasound scans obtained underwater at 15 msw showed that lung sliding is preserved without any parenchymal abnormalities. This is the first time that ultrasound lung images have been acquired in SCUBA divers at depth, and, although in clinical practice lung sliding only rules out pneumothorax, this extreme physiology model also confirms that air delivered by the SCUBA circuit at the same environmental pressure keeps the lungs inflated and allows inflation and deflation of the parenchyma. Instead, when resurfacing, SCUBA divers developed ultrasound findings that – despite the small number of participants – seemed slightly more pronounced after the 40 msw dive, but did not result in any measurable gas exchange impairment. The available literature shows that SCUBA diving can increase extravascular lung water. Susilovic-Grabovac et al. performed lung ultrasound 30 min after a 18 msw dive (bottom time: 47 min) and found that B-lines progressively – but not completely – resolved across scans repeated every 30 min (Susilovic-Grabovac et al., 2017). While Dujic et al. did not find a significant increase in B-lines measured within 120 min of a single dive with wetsuits and compressed air to 33 m (bottom time 20 min) (Dujic et al., 2011), on three immersions to 63–65 msw (one per day, 13.5–16.5 min bottom time) with dry suits and SCUBA circuits breathing trimix and nitrox mixtures, Ljubkovic et al. found that B-lines occurred 120–180 min after resurfacing, stayed stable on the first day, increased on the second day, and stayed similarly elevated on the third day (Ljubkovic et al., 2010). Marinovic et al. performed prolonged monitoring during a 6-day diving campaign with dry suits (one per day; depths increasing from 60 to 80 msw; bottom times of 9–13 min), finding an increase in B-lines after the dives on days 2, 4 and 5 (Marinovic et al., 2010). Signs of increased extravascular lung water were found as well, using semi-closed and closed circuit rebreathers on repeated dives on consecutive days (Gouin et al., 2022; Martinez-Villar et al., 2022), or after exercising at 1 m depth in a pool, especially when the apparatus was placed on the back, therefore generating negative transpulmonary pressure (Castagna et al., 2018). In our upright, low-workload, brief bottom phase (∼10 min) SCUBA diving experimental model, the absence of B-lines at 15 msw is consistent with the Starling model applied to the alveolar–capillary barrier. Given that alveolar pressure rises in parallel with environmental pressure thanks to SCUBA circuits, a modest negative static-lung load can be imposed by upright posture, which was maintained for a brief time after rapidly reaching the predefined depth. Other negative alveolar pressure contributors, such as increased ventilatory effort and work of breathing due to higher gas densities, were not expected, as the participants were not exercising at high intensity, thereby keeping the net hydrostatic gradient small. This assumption is also supported by $PaCO_2$ values, which remained within normal ranges at both depths. On the other side of the alveolar–capillary barrier, an immersion-related central blood shift was present, increasing pulmonary capillary pressures. However, low exertion and short duration probably did not suffice to perturb the permeability equilibrium at depth. Albumin was not among the studied markers, but previous studies demonstrated that diving increases post-dive albumin concentrations, thereby increasing oncotic pressure and acting as a probable protective countermeasure (Ljubkovic et al., 2010). The development of surface lung ultrasound alterations can be similarly explained by taking into account transmural capillary stress: (1) further time elapsed to reach the surface, allowing hormonal interactions with pulmonary capillaries (e.g. increases in pro-atrial natriuretic peptide (Ljubkovic et al., 2010), knowing that atrial natriuretic peptide promotes endothelial permeability (Curry, 2005)); (2) the progressive degradation of endothelial glycocalyx – as emerging from SDC-1 and HS increase after dive – ultimately converting hydrostatic filtration into protein-rich oedema. Moreover, performing lung ultrasound immediately after resurfacing, while still onboard the support vessel, could have improved the overall accuracy of the assessment.

Second, breath-hold divers' PaO_2 overall followed the physiological model predicted by theory at different depths: hyperoxaemia developed in divers at depth, then oxygen levels dramatically fell when resurfacing, all in a very short time and consistent with similar studies performed in open seawater (Barković et al., 2023; Scott et al., 2021), except in three participants. These breath-hold divers did not differ from the overall cohort in any distinguishing characteristic, including weight, height, BMI (Table *A*1), after-dive lung ultrasound findings (Table 4) or dive duration (Table *A*2). In previous analyses carried out by our group in a pool with warm water (31.5 ± 0.5°C), a higher prevalence of relative hypoxaemia at depth was noted: two out of six participants at 40 m of freshwater (mfw) (Bosco, Rizzato, Martani et al., 2018); five participants at 15 mfw and four at 42 mfw out of 14 participants (Paganini, Moon et al., 2023). A significant difference in water

temperature between the studies should be acknowledged – around 31.5 ± 0.5°C in the pool and 17° to 25 ± 0.5°C in the current experimental campaign – with potential differences in diving response magnitude. The diving response is known for selectively redistributing blood to vital organs and is elicited more by cold water (Bosco, Rizzato, Moon et al., 2018). Schagatay and Holmes measured heart rate variations caused by face immersion after 60 min in air, both at different combinations of temperatures: the immersion in cold water after exposure to air at high temperatures caused the most significant reduction in heart rate, as a surrogate measure of a more intense diving response (Schagatay & Holm, 1996). In the present study, during summer, participants were exposed to hot temperatures while approaching the experimental sea sites – comparable to those outside the deep pool – but then submerged into colder water. This thermal discrepancy could have enhanced the diving response, leading to more intense peripheral vasoconstriction and thereby improving oxygen-sparing by directing a relatively greater proportion of systemic blood toward the alveolar interface where gas exchange occurs. Conversely, in the pool experiments, the higher occurrence of relative hypoxaemia at depth might reflect a less pronounced diving response and weaker peripheral vasoconstriction, resulting in less blood being exposed to oxygen diffusing from the alveoli. However, this explanation remains uncertain since changes in the diving response, as well as possible sources of shunt from intrapulmonary arteriovenous anastomoses (IPAVA) and especially patent foramen ovale (PFO) – known to be more prevalent in elite divers (Kelly et al., 2022) – potentially elicited by the centralized blood, were not investigated. Although not statistically significant, $PaCO_2$ increased at 25 msw and 40 msw and declined after each resurfacing, consistent with recent findings from similar experiments (Barković et al., 2023; Bosco, Rizzato, Martani et al., 2018; Scott et al., 2021). The rise in $PaCO_2$ at depth likely reflects lung compression, whereas the subsequent decrease corresponds to lung re-expansion during ascent. An inverted trend was observed at 15 msw, likely due to the shallower depth. In the ultrasound scans at 15 msw, lung sliding was absent – as expected, since the diver was not breathing – with lung parenchyma showing lung pulse synchronous with cardiac activity without any focal anomaly, overall due to compression of aerated tissue at depth. When back at the surface, almost all breath-hold divers showed varying degrees of subclinical interstitial oedema and pleural irregularities, further supporting previous studies (Frassi et al., 2008; Lambrechts et al., 2011; Paganini, Moon et al., 2023; Patrician, Pernett et al., 2021; Patrician et al., 2021a; Patrician et al., 2021b; Yu et al., 2024).

Overall, our experiments indicate that ABG variations follow what is postulated by traditional physiological theory, with some exceptions in BHD. In SCUBA divers performing non-strenuous activities in warm or cold water, PaO_2 increases at depth, with higher values registered when going deeper, while pH and $PaCO_2$ do not vary significantly. In no-limit BHD (sled for descent – inflated lifting device for ascent), pH and $PaCO_2$ are subjected to minor variations; depth does not induce PaO_2 increase in all the divers, with colder water seeming to reduce the prevalence of relative hypoxaemia at depth, while a profound hypoxaemia is consistently registered when back at the surface, irrespective of water temperature. As demonstrated by ultrasound findings, the lungs were affected in both SCUBA and breath-hold divers, without translating to symptoms or gas exchange impairment. Also, lung ultrasound findings seemed to be more marked after deeper dives, and in freedivers than in SCUBA divers, potentially reflecting higher stress levels imposed by deeper dives and BHD (again subjected to repeated compression and re-expansion cycles). Unfortunately, no inference was possible, as the intent of lung ultrasound in this campaign was only descriptive; potential correlations should be quantitatively explored in future experiments. Finally, increases in SDC-1 and HS levels in both SCUBA and freediving indicate diving-related endothelial stress. Given their non-lung-specific nature, stress may have originated from multiple sources. The most plausible explanation aligns with previous evidence of elevated circulating pro-inflammatory mediators (e.g. reactive oxygen and nitrogen species) after diving activities (Vezzoli et al., 2024), potentially associated with diving-induced circulating microparticles in SCUBA (Thom et al., 2015) and breath-hold divers (Barak et al., 2020), rather than VGE (which were anyway limitedly detected in our sample, and without scanning all the participants). Reduced flow-mediated dilatation of the brachial artery has been demonstrated after both deep breath-hold dives (35 msw) with short surface intervals and repetitive breath-hold dives, interpreted as generalized endothelial dysfunction (Barak et al., 2020). Anyway, paired with the lung ultrasound findings, the hypothesis of enhanced lung glycocalyx shedding in response to underwater exposure cannot be excluded, as the pulmonary endothelium is subjected to direct stress on both the capillary side (acute pulmonary hypertension due to central blood pooling) and the alveolar side (higher pressures in SCUBA; repeated compressions and expansions in BHD), but it requires further lung-specific evidence in future studies. For example, adding surfactant integrity markers could help explore the alveolar side. Previous measurements obtained 30 min after an 18 msw SCUBA dive showed increased levels of the mature form of surfactant protein B, interpreted as preserved alveolar cell integrity despite extravascular lung water detected by ultrasound (Susilovic-Grabovac et al., 2017).

Limitations of the present study include a small sample size due to both the difficulty of recruiting participants and experimental logistics, which currently hampers the generalizability of the findings. Also, participants were not studied to search for PFO or IPAVA, which limits interpretation of potential sources of shunt affecting gas exchange results. Despite strictly adhering to the protocol, samples obtained at depth may have been affected by analytical errors, particularly PaO_2 values exceeding the upper calibration limit of a blood gas analyser. Lung ultrasound was not performed on the posterior aspects of the lungs, which affected accuracy; this aspect should be considered in future experiments to improve sensitivity for pulmonary lesions. SDC-1 and HS were investigated as markers of glycocalyx stress; however, they are not lung-specific, and other potential sources of inflammation may have influenced the results. Thus, future experiments should include a wider panel of both lung-specific and general inflammatory markers. VGE monitoring was limited to a single post-dive assessment within 10 min after resurfacing, rather than serial observations over 120 min as recommended (Møllerløkken et al., 2016), since VGE was a secondary outcome intended for comparison with endothelial stress markers at the same timepoint. Lastly, we acknowledge several limitations inherent to conducting the experiment underwater, including: the inability to adjust pedalling effort once the bike was submerged or the absence of heart rate monitoring, to standardize exercise for SCUBA's cardiometabolic fitness level; the absence of a surrogate measurement of diving response; and lack of core temperature measurements to correlate with diving response. These constraints stemmed from technical challenges that we accepted in order to perform the study in open seawater.

Conclusions

The combined assessment of ABGs, lung ultrasound and circulating glycocalyx markers suggests that both SCUBA diving and no-limit BHD, across different depths, induce subclinical pulmonary strain without necessarily impairing gas exchange. All divers remained asymptomatic, despite pleural and interstitial ultrasound artefacts present in almost all post-dive scans. Compared with previous warm-freshwater experiments, SCUBA diving produced similar ABG variations, whereas open-seawater BHD resulted in fewer episodes of relative hypoxaemia at depth – though no solid explanation for this difference is currently available. As previously suggested, the three breath-hold divers failing to show the expected rise in PaO_2 at depth may have experienced lung atelectasis, a phenomenon that lung ultrasound might not consistently detect. Future research should incorporate lung-specific biomarkers and monitoring of the diving response to better describe how far alveolar–capillary barrier stress and transient extravascular lung water can progress before crossing the threshold from physiological adaptation to acute pathology.

Appendix

Table A1. Participants' characteristics

SCUBA DIVERS					
Diver #	Sex	Age	Weight (kg)	Height (m)	Body mass index (kg/m^2)
1	M	38	65	1.72	25.4
2	M	57	74	1.80	22.8
3	M	32	93	1.86	26.9
4	M	35	95	1.75	31.0
5	M	53	75	1.80	23.1
6	M	52	97	1.79	30.3
7	M	37	78	1.79	24.3
8	F	32	60	1.63	22.6
9	M	53	75	1.80	23.2
10	M	46	115	1.91	31.5
Mean (SD)	—	44 (10)	82.7 (16.8)	1.78 (0.08)	26.1 (3.6)
BREATH-HOLD DIVERS					
Diver #	Sex	Age	Weight (kg)	Height (m)	Body mass index (kg/m^2)
1	F	43	60	1.73	20.1
2	M	34	70	1.82	21.1
3	F	53	65	1.71	22.2
4	M	60	79	1.80	24.4
5	M	48	82	1.71	28.0
6	M	52	83	1.72	28.1
7	M	61	69	1.84	20.4
8	F	43	61	1.64	22.7
9	M	46	75	1.77	23.9
10	M	62	82	1.91	22.5
11	M	33	85	1.80	26.2
12	M	61	82	1.71	28.0
Mean (SD)	—	50 (10)	74.4 (9.1)	1.76 (0.07)	24.0 (2.9)

Table A2. Breath-hold (BH) dive durations

BH diver #	Depth (m)	Descent time (s)	Bottom time (s)	Ascent time (s)	Total duration (s)
1	15	22	28	26	76
2	15	23	45	24	92
3	15	19	38	22	79
4	15	18	31	27	76
5	15	20	29	24	73
9	15	25	34	26	85
10	15	18	24	24	66
11	15	20	37	26	83
12	15	22	26	27	75
1	25	33	36	42	111
2	25	37	42	40	119
3	25	34	39	39	112
4	25	34	24	41	99
6	25	31	33	44	108
7	25	38	31	42	111
8	25	31	35	38	104
9	40	51	30	60	141
10	40	54	23	59	136
11	40	50	32	51	133
12	40	49	34	52	135

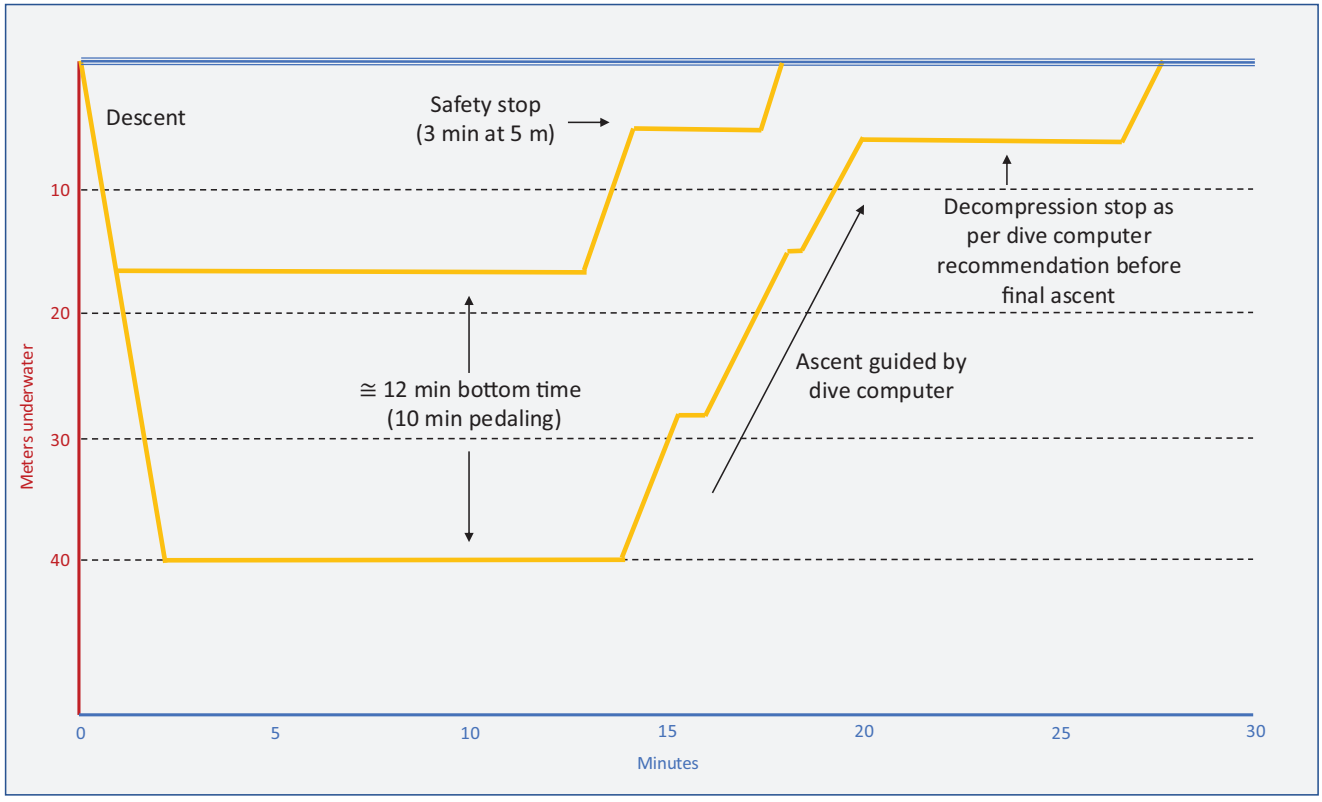

Figure A1.
Schematic representation of scuba diving profiles.

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Additional information

Data availability statement

All data supporting the findings of this study are available within the manuscript and its supplementary materials

Competing interests

The authors have declared no conflicts of interest.

Author contributions

The experiments were performed at sea. ABG and ultrasound results analyses were performed at the Department of Biomedical Sciences, University of Padova, Padova, Italy; pulmonary glycocalyx analysis was performed at the Institute of Clinical Physiology, National Research Council (IFC-CNR), Milano, Italy. Conceptualization: M.P., T.A.G., L.M., E.M.C., R.E.M., G.B. Formal analysis: M.P., S.M.-S. Funding acquisition: G.B. Investigation: M.P., T.A.G., S.M.-S., L.M., D.C., L.Z., V.M., R.C., M.M., G.B. Methodology: M.P., T.A.G., S.M.-S., L.M., D.C., E.M.C., R.E.M., G.B. Project administration: G.B. Resources: G.B. Supervision: G.B. Writing – original draft: M.P., T.A.G., S.M.-S., L.Z. Writing – review & editing: M.P., L.M., D.C., V.M., R.C., M.M., E.M.C., R.E.M., G.B. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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Keywords

arterial blood gas, breath-hold diving, diving physiology, environmental physiology, glycocalyx, lung ultrasound, oxygen, SCUBA diving

Supporting information

Additional supporting information can be found online in the Supporting Information section at the end of the HTML view of the article. Supporting information files available:

Peer Review History

Translational perspective

Diving exposes the lungs to physiological challenges, yet how gas exchange and lung tissue integrity are affected in open seawater remains poorly understood. We hypothesized that integrating arterial blood gas (ABG) analysis, lung ultrasound and circulating markers of endothelial glycocalyx damage (syndecan-1 and heparan sulfate) would reveal subclinical pulmonary strain in both SCUBA and breath-hold (BH) divers. In SCUBA divers breathing compressed air at 15 or 40 m underwater, arterial partial pressure of oxygen (PaO_2) increased at depth as predicted and returned to baseline upon surfacing. In BH divers reaching 15, 25 or 40 m underwater, PaO_2 rose at depth and fell to hypoxaemia on resurfacing, though a subset did not develop the expected hyperoxaemia at depth. All divers developed post-dive ultrasound signs of fluid accumulation in lung tissue and elevated glycocalyx markers indicating endothelial stress, without symptoms. For pulmonary and vascular biology, diving confirms its role as a human model of transient alveolar–capillary barrier stress and *in vivo* glycocalyx degradation. For sports medicine, these findings reinforce the hypothesis that subclinical lung changes are common even after relatively brief, moderate exposures to environmental pressure variations, warranting attention during repeated or prolonged activities. Clinically, the combination of lung ultrasound with blood biomarkers offers a portable, non-invasive toolkit adaptable to monitoring patients at risk of pulmonary oedema in other settings, such as high-altitude exposure, competitive diving events or heart failure. Future studies should incorporate lung-specific biomarkers and serial monitoring to determine when these reversible changes might cross the threshold toward clinically significant lung injury.