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CASE STUDIES IN PHYSIOLOGY

Case Studies in Physiology: Breath-hold diving beyond 100 meters—cardiopulmonary responses in world-champion divers

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Abstract

In this case study, we evaluate the unique physiological profiles of two world-champion breath-hold divers. At close to current world-record depths, the extreme physiological responses to both exercise and asphyxia during progressive elevations in hydrostatic pressure are profound. As such, these professional athletes must be capable of managing such stress, to maintain performing at the forefront human capacity. In both divers, pulmonary function before and after deep dives to 102 m and 117 m in the open sea was assessed using noninvasive pulmonary gas exchange (indexed via the O_2 deficit, which is analogous to the traditional alveolar to arterial oxygen difference), ultrasound B-line scores, airway resistance, and airway reactance. Hydrostatic-induced lung compression was also quantified via spirometry. Both divers successfully performed their dives. Pulmonary gas exchange efficiency was impaired in both divers at 10 min but had mostly restored within a few hours. Mild hemoptysis was transiently evident immediately following the 117-m dive, whereas both divers experienced nitrogen narcosis. Although B-lines were only elevated in one diver postdive, reductions in airway resistance and reactance occurred in both divers, suggesting that the compressive strain on the structural characteristics of the airways can persist for up to 3.5 h. Marked echocardiographic dyssynchrony was evident in one diver after 10 m of descent, which persisted until resolving at ~77 m during ascent. In summary, despite the enormous hydrostatic and physiological stress to diving beyond 100 m on a single breath, these data provide valuable insight into the extraordinary capacity of those at the pinnacle of apneic performance.

NEW & NOTEWORTHY This study shows that world-champion breath-hold divers demonstrate incredible tolerability to extreme levels of hydrostatic-induced lung compression. Immediately following dives to >100 m, there were acute impairments in pulmonary gas exchange efficiency, mild accummulation of extravascular lung fluid, noticable intrathoracic discomfort, and evident nitrogen narcosis, however, within a few hours, these had all mostly resolved.

apnea; breath-holding; diving; lung compression; nitrogen

INTRODUCTION

Breath-hold diving is a ubiquitous activity for recreation, sustenance, military, and sport (1, 2); it involves highly integrative physiology and extreme responses to both exercise and asphyxia during progressive elevations in hydrostatic pressure (3, 4). However, since the famous anecdote of Giorgios Statti diving to 70 m in 1913 (3) to Herbert Nitsch reaching 214 m in 2007, the human capacity for deep breath-hold diving has continually refuted expectations of the physiological limits. Despite the majority of both male and female depth-records now at, or in excess of 100 m, only the most elite divers are actually capable of exceeding this depth—a threshold that was first established by Enzo Majorca in 1988 (3). However, we are unaware of any data inclusive of the physiological demands that occur during diving to such extreme depths. Herein, we evaluate the unique physiological profiles of two world-champion-level breath-hold divers who on a single breath dove to 102 m and 117 m in the open sea.

METHODS

Participants

Divers were male, nonsmokers, and free from cardiovascular or respiratory disease. Divers were recruited during a diving training camp in Croatia, which has been reported elsewhere (5); however, the comparisons herein are not otherwise duplicated. Both divers had ~15 years of breath-hold diving experience and are decorated world-champion-level divers in deep-diving disciplines. For both dives, the dive line (i.e., a static and weighted rope) was connected to a custom winch safety system to increase diver safety. A thermocline was evident at 12 ± 5 m (i.e., the decrease in sea

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temperature from 26°C ± 1°C to 16°C ± 0.5°C). The protocol was approved by the ethics committee at the School of Medicine, University of Split; both oral consent and written consent were obtained and conformed to the Declaration of Helsinki, except for registration in a database.

Diver 1

A world-class diver (32 yr, 180 cm, 85 kg, 26 kg/m^2) dove to 102 m using a discipline known as free immersion. This discipline consists of the diver propelling themself down the dive line using only their arms (i.e., the diver does not kick). The total dive time was 4:12 min.

Diver 2

A world-class diver (32 yr, 177 cm, 71 kg, 23 kg/m^2) dove to 117 m using the discipline known as constant weight with fins. This discipline stipulates that the diver propel themself down the dive line using a monofin or bi-fins, and then ascending under their own effort. Since this was a training dive, the diver partially utilized the winch safety system to aid ascent. The total dive time was 2:34 min.

EXPERIMENTAL DESIGN

Spirometry

Slow and forced vital capacity tests (SVC and FVC, respectively) were performed (HypAir, Medisoft, Belgium), according to standard guidelines (6). Residual volume (RV) was estimated using published RV estimations (7) and corrected to a mean adjustment of −15.35%, as described elsewhere (5). These calculated RVs were added to measured SVCs to provide an individualized estimation of total lung capacity (TLC). In addition, since both divers would perform lung packing (i.e., glossopharyngeal insufflation) before the dive to increase surface lung volume, a modified SVC test was conducted at baseline to approximate total packable volume. The RV reserve describes the volume difference between RV and the estimated lung volume at maximum depth [calculated in accordance with Boyle's law—as described by Patrician et al. (5)].

Cardiopulmonary Assessments

At baseline and again following the dive (at 5 min and 148 min in *diver 1*; at 10 min and 205 min in *diver 2*), pulmonary status was evaluated using the following methods: pulmonary gas exchange efficiency [indexed via the O_2 deficit, which was calculated from the difference between the end-tidal and the calculated arterial P_{O_2} (via pulse oximetry and corrected for the Bohr effect by using the end-tidal P_{CO_2})—a measure that has been validated in a variety of healthy and patient populations (8) and during exercise (9)]; ultrasound lung B-lines to quantify interstitial fluid accumulation (via Doppler ultrasound; Vivid-Q; bilateral parasternal to midaxillary imaging of hemithorax from the 2nd to 4th intercostals—totaling 28 zones) (10); and indexes of altered lung compliance [via airway resistance (i.e., an indicator of airway obstruction) and reactance (i.e., expresses the mechanical properties of the airways) using multifrequency (5–11–19 Hz) forced oscillation technique (MGC Diagnostics) (11) performed in the upright sitting position—each measurement of compliance consisted of ≥14 acceptable breaths, and ventilation between tests was 1.5%]. Electrocardiography (12-lead; custom waterproof unit) was measured throughout the dive.

RESULTS

Spirometry, lung volumes across the dive, and estimated lung compression for *divers 1* and *2* are presented in Table 1. At ~20 s predive, both divers performed glossopharyngeal insufflation (lung packing) to increase the volume of air (and thus oxygen) in the lungs.

Diver 1

The resulting pulmonary changes of the dive are presented in Fig. 1.

One minute before official top (OT; the start of dive), heart rate (HR) was 63 beats/min, which increased to 112 beats/min at OT, and was followed by substantial bradycardia within the first few meters (dropping HR to 57 beats/min within 4 s). HR remained steady (between 40 and 50 beats/min) across the dive (44 beats/min at 102 m). The electrocardiogram (ECG) revealed suspected sinus bradycardia with supraventricular premature and premature ventricular contractions that began promptly at 10 m until abruptly resolving at 77 m on the ascent (Fig. 2). Unfortunately, upon immersion, signal resolution became suboptimal and P waves were difficult to detect.

The resulting physiological changes of the dive are presented in Fig. 1. By 102 m, the hydrostatically induced lung compression was estimated to reduce lung volume to 0.94 L. Immediately following the dive, gas exchange was impaired [an almost 2.5-fold increase in O_2 deficit (alveolar to arterial partial pressure difference) to 54 mmHg], B-lines had increased from 8 to 27 (predominantly prevalent in the midaxillary and parasternal planes), and there was marked breathing discomfort (with mucus) upon exhalation and general fatigue. The diver reported mild nitrogen narcosis during the dive, which resolved quickly upon surfacing. At 148 min following the dive, gas exchange, B-lines, and subjective discomfort had mostly resolved back to normal. Despite airway resistance and reactance keeping within previously established normative ranges (11), airway resistance values in the small (1.39 ± 0.57 cmH_2O/[L/s]), medium (1.56 ± 0.59 cmH_2O/[L/s]), and large (1.89 ± 0.58

Table 1. *Spirometry, Lung Volumes Across the Dive, and Estimated Lung Compression*

	Diver 1	Diver 2
SVC, L	7.68	6.09
FEV1, L	5.45	4.31
FVC, L	7.39	5.99
FEV1/FVC, %	73.8	71.9
MEF, L/s	4.04	3.00
RV, L	1.42	1.37
TLC, L	9.10	7.46
Lung packing volume, L	1.47	1.16
Predive lung volume, L	10.57	8.62
Dive depth, m	102	117
Lung volume at max depth, L	0.94	0.68
RV reserve, L	−0.48	−0.69

FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; m, meters sea water; MEF, mean expiratory flow; RV, residual volume; SVC, slow vital capacity; TLC, total lung capacity.

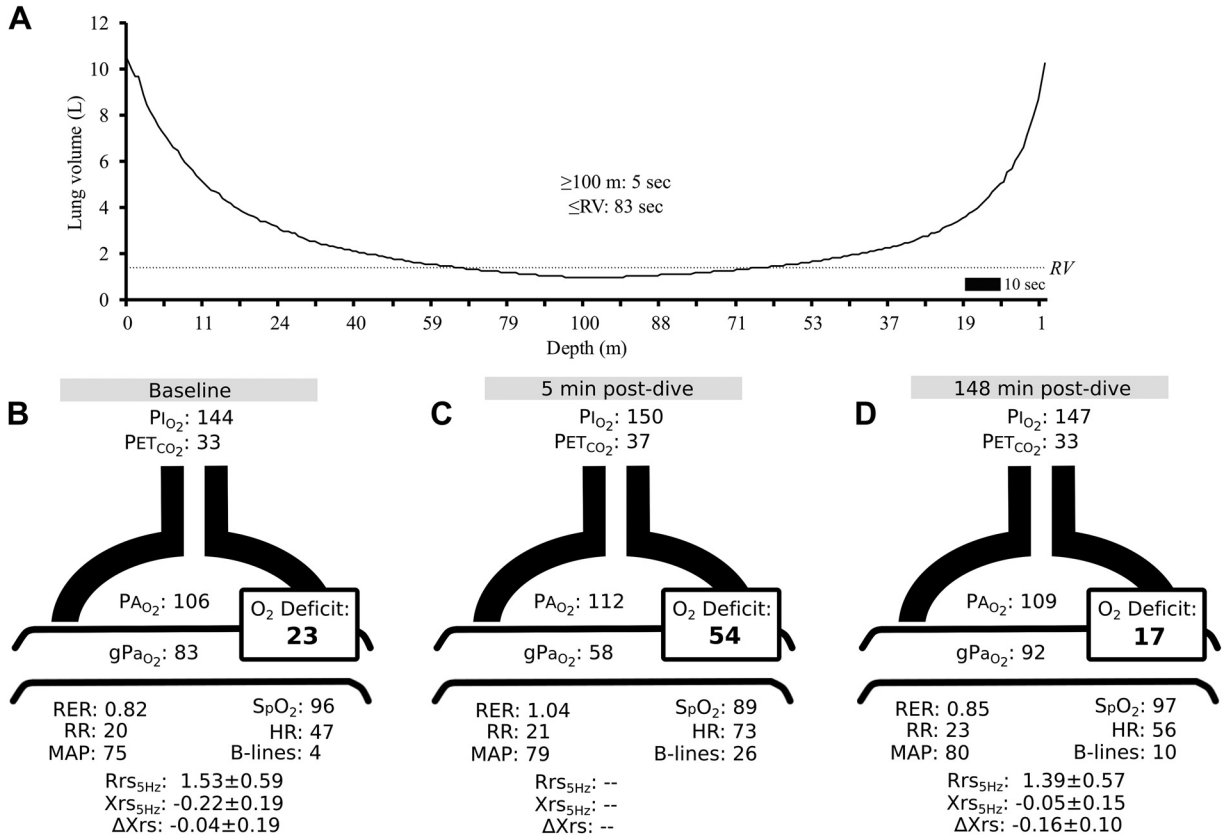

Figure 1. Schematic illustration of the dive profile to 102 m (*diver 1*), with the estimated changes in lung volume (*A*) and pulmonary gas exchange (at baseline, 5 min, and 148 min postdive, in *B*, *C*, and *D*, respectively). Total dive time was 4 min 12 s. HR, heart rate (beats/min); PA_{O_2}, alveolar partial pressure of oxygen (mmHg); gPa_{O_2}; calculated arterial partial pressure of oxygen (mmHg); P_B, barometric pressure (mmHg); $P_{ET_{CO_2}}$, end-tidal pressure of carbon dioxide (mmHg); PI_{O_2}, inspired partial pressure of oxygen (mmHg); m, meters of sea water; RER, respiratory exchange ratio; RR, respiratory rate (breaths/min); RV, residual volume (L); Rrs_{5Hz} small airway resistance (normal range: 1.60–3.81 cmH_2O L/s^{-1}); s, seconds; SpO_2, peripheral oxygen saturation (%); Xrs_{5Hz}, small airway reactance (normal range: −1.42 to −0.15 cmH_2O L/s^{-1}); ΔXrs, expiratory flow limitation (normal range: −1.0 to 2.8 cmH_2O L/s^{-1}).

cmH_2O/[L/s]) airways were all lower than baseline by −9%, −7%, and −9%, respectively. Airway reactance values in the medium (0.62 ± 0.30 cmH_2O/[L/s]) and large (1.43 ± 0.57 cmH_2O/[L/s]) airways were within −4% and −5% to baseline, but those in the small airways (−0.05 ± 0.15 cmH_2O/[L/s]) had decreased by 75% from baseline. Likewise, there was no evidence of expiratory flow limitation (ΔXrs; −0.16 ± 0.10 cmH_2O at 205 min vs. −0.04 ± 0.19 cmH_2O at baseline) (Fig. 1).

Diver 2

The resulting pulmonary changes of the dive are presented in Fig. 3.

One minute before OT, heart rate was 93 beats/min, which increased to 130 beats/min at OT, and was followed by substantial bradycardia within the first few meters (dropping HR to 54 beats/min within 3 s). HR remained steady across the dive (53 beats/min at 117 m). ECG across the dive was mostly unremarkable, aside from a few R-R interval irregularities during ascent, which could be extrasystole in nature, but due to issues in signal resolution (i.e., P waves undetectable), this was not possible to ascertain.

By 117 m, the hydrostatically induced lung compression was estimated to have reduced TLC to ∼0.68 L (i.e., a 12-fold reduction, and ∼1/2 of RV). Consequently, within 10 min following the dive, gas exchange was impaired, as reflected by an approximately fivefold increase in O_2 deficit from baseline (10 mmHg to 57 mmHg); there was some mild tightness/discomfort in the chest and an urge to cough that coincided with perceived restriction upon full inhalation and edema upon exhalation. Upon surfacing, the discomfort (i.e., tension) within the chest appeared to evolve from being perceptually more diffuse immediate postdive to being more localizable to the upper regions of the lung, which transitioned parasternally by 30 min postdive. Although a few red blood cell specks were visible in sputum, B-lines remained relatively unchanged from baseline (12 B-lines vs. 11 B-lines, respectively)—keeping mostly to the midaxillary planes. As requested by *diver 2*, B-lines immediately postdive were assessed in the upright position. The diver also reported significant nitrogen narcosis during the dive, and upon surfacing, was amnesiac—with respect to recalling dive details. However, the amnesia was transient in nature and therefore as it abated, the diver's recollection of their dive improved. Within ∼30 min of surfacing, the amnesiac symptoms had mostly resolved.

At 205 min postdive, gas exchange had improved but remained slightly elevated (18 mmHg), B-lines remained low (total of 6), and subjective discomfort (and sputum) had resolved back to normal. Airway resistance and reactance remained comparable with baseline (albeit, consistently

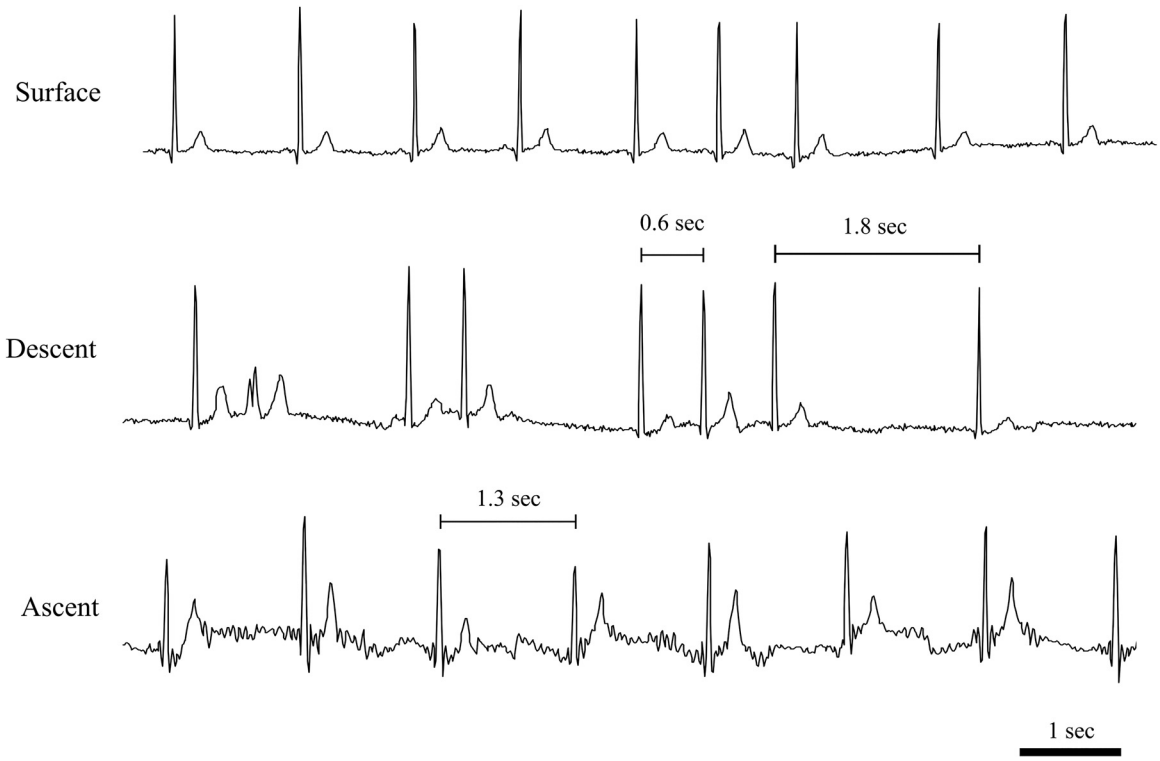

Figure 2. Electrocardiographic signals in *diver 1* (lead V5). Surface—sinus rhythm with respiratory fluctuations during the relaxed breathing preparation phase (3 min before dive). Descent—sinus bradycardia with regular supraventricular premature beats and six premature ventricular contractions occurring from 10 m on descent, continuing for ~2:16 min to 77 m on ascent. Ascent—sinus bradycardia, occurring during the last 77 m of ascent. Mean R-R interval durations during descent and ascent are included.

lower across all airway sizes) and within previously established normative ranges (11). Airway resistance values in the small (2.04 ± 0.55 cmH_2O/[L/s]), medium (2.15 ± 0.57 cmH_2O/[L/s]), and large (2.15 ± 0.57 cmH_2O/[L/s]) airways were all lower than baseline by −12.9%, −11.3%, and −14.6%, respectively. Airway reactance values in the small (−0.72 ± 0.17 cmH_2O/[L/s]) and large (0.86 ± 0.61 cmH_2O/[L/s]) airways were within +3.6% and −9.0% to baseline, but those in the medium (0.19 ± 0.33 cmH_2O/[L/s]) airways had decreased by 25.0% from baseline. Likewise, there was no evidence of expiratory flow limitation (ΔXrs; −0.15 ± 0.16 cmH_2O at 148 min vs. −0.19 ± 0.05 cmH_2O at baseline; Fig. 3).

DISCUSSION

This case study of two world-championship breath-hold divers diving beyond 100 m, on a single breath without any serious ill consequence, highlights the capacity for these athletes to endure enormous hydrostatic strain. Certainly, the evident transient impairment in pulmonary gas exchange efficiency, mild hemoptysis, and associated symptoms within 10 min of surfacing indicate the enormity of the physiological strain of diving to 102 m and 117 m. At such depths, total lung volumes were estimated to be reduced by 91% and 92%, respectively, and it would be estimated that between 50% and 60% of alveoli could be collapsed (12).

The mechanics of hydrostatically induced lung compression and tension within the thorax are complex (12, 13). The increase in B-lines in *diver 1* is consistent with their symptoms of mucus in the airways and discomfort upon exhalation, and thus, this provides mechanistic evidence for the origin to the impairment in gas exchange inefficiency (since any fluid within the alveoli or in the interstitial space would both reduce the surface area and increase the diffusion distance for O_2). Aside from the potential influence of body position immediately postdive, it is not clear why *diver 2*, in comparison, demonstrated no detectable change in the B-line score immediately following the dive. Even though there was no obvious obstruction or stiffening of the airways, the pattern of lower airway resistance and reactance postdive, across all airway sizes compared with baseline, in both divers, illustrates that the compressive strain of deep diving on the lungs is evident for at least up to 3.5 h. The long-term implications of these transient indexes of lung compliance remain to be determined.

The mechanisms driving these dyssynchronous ECG signals likely reflect marked autonomic conflict between parasympathetically mediated bradycardia (i.e., diving response) and sympathetically driven tachycardia (i.e., cold shock and/or exercise response) (14). Moreover, the potentially concomitant influences of hydrostatically induced centralization of blood volume from the legs to the thorax (15), arterial hypertension (23), and mechanical release of hyperinflation-induced compression of the heart from lung packing (16) could have all ensued within the first 10 m, and therefore affect baroreceptor activation and lead to altered chronotropic drive due to cardiac enlargement and activation of right atrial stretch receptors. Why it is only *diver 1* who displays marked dyssynchrony is not clear from this study; however, alike to autonomic conflict

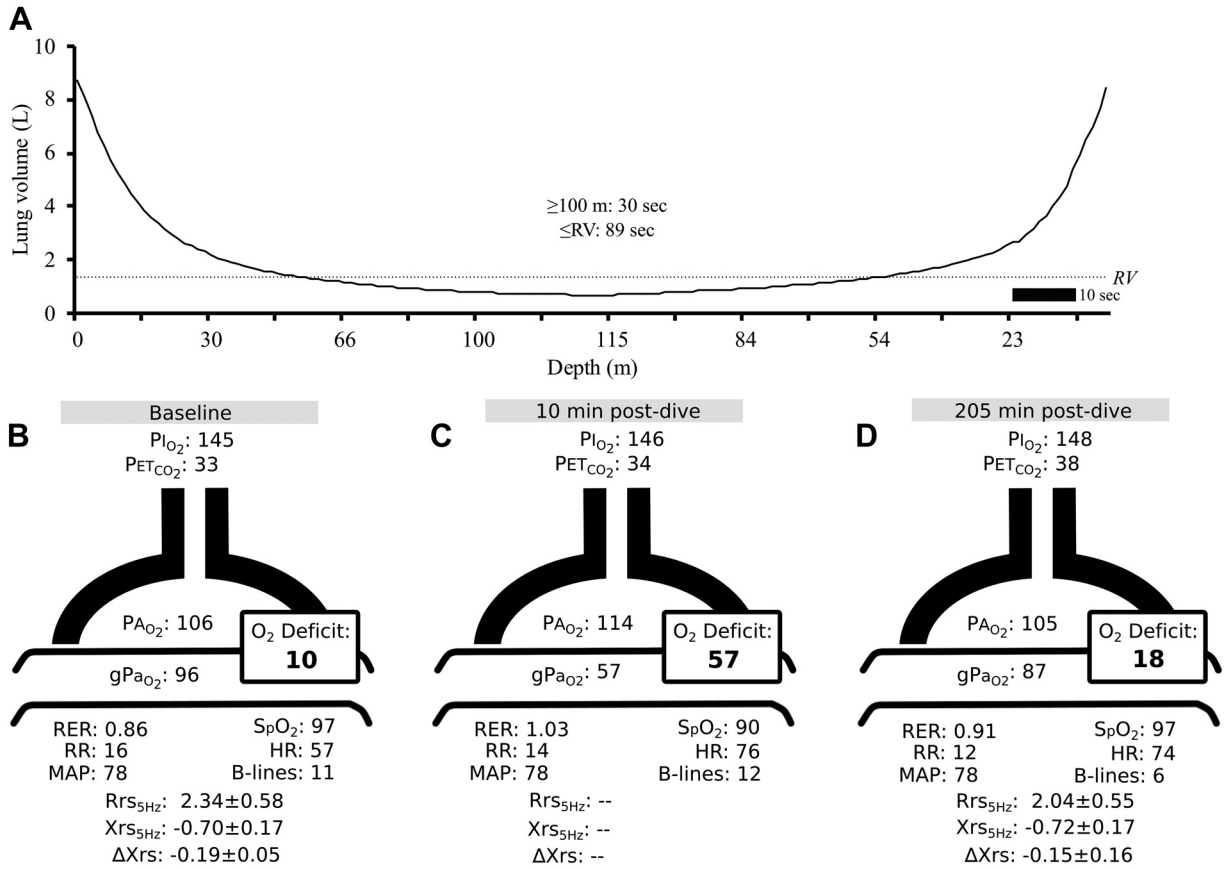

Figure 3. Schematic illustration of the dive profile to 117 m (*diver 2*), with the estimated changes in lung volume (*A*) and pulmonary gas exchange (at baseline, 10 min and 205 min postdive, in *B*, *C*, and *D*, respectively). Total dive time was 2 min 35 s. HR, heart rate (beats/min); PA_{O_2}, alveolar partial pressure of oxygen (mmHg); gPa_{O_2}; calculated arterial partial pressure of oxygen (mmHg); P_B, barometric pressure (mmHg); PET_{CO_2}, end-tidal pressure of carbon dioxide (mmHg); PI_{O_2}, inspired partial pressure of oxygen (mmHg); m, meters of sea water; RER, respiratory exchange ratio; RR, respiratory rate (breaths/min); RV, residual volume (L); Rrs_{5Hz} small airway resistance (normal range: 1.39–3.48 cmH_2O L/s^{-1}); s, seconds; SpO_2, peripheral oxygen saturation (%); Xrs_{5Hz}, small airway reactance (normal range: −1.42 to −0.27 cmH_2O L/s^{-1}); ΔXrs, expiratory flow limitation (normal range: −1.0 to 2.8 cmH_2O L/s^{-1}).

(14), some individuals may possess an inherent (benign or malignant) predisposition.

Early work in diving mammals (17) and humans (18) has demonstrated that the nitrogen tension in the blood rises during breath-hold diving. In fact, nitrogen narcosis is not an uncommon feature in breath-hold dives exceeding ~70–90 m (personal observations and diver communications). The narcosis described by both divers, and its apparent persistence in *diver 2*, are two of only a few examples in the literature (1). In these divers, there was no vertigo, tingling sensation, joint pain, loss of motor function, impairments to consciousness, paralysis, or difficulty speaking, which would insinuate some degree of decompression sickness, rather than primarily narcosis. Additionally, the risk of decompression at up to ~125 m is estimated to be <2% (19). In *diver 2*, transient amnesia was the most prevalent symptom which progressively resolved with longer time at the surface. The difference in narcosis severity between *diver 1* and *diver 2* likely hinges upon total exposure, as each diver spent 26 or 42 s, respectively, beyond 90 m. Furthermore, given *diver 2* spent 30 s ≥ 100 m, the alveolar partial pressure of nitrogen would be exponentially elevated (in accordance with Boyle's Gas Law), and therefore the uptake of nitrogen into the blood at this depth would be significant. The prolonged nature of the narcosis is interesting but aligns with reports of narcosis lasting up to 30 min following a 20-min no-decompression compressed gas dive to 33 m (20).

It is essential to note that majority of divers, due to a myriad of physiological and psychological reasons, will not be able to reach such extreme depths. For example, even diving to depths equivalent to RV has been shown to induce hemoptysis in trained divers (21), and overstraining the lungs has led to fatal accidents (22). Thus, we do not encourage this extreme diving, especially in view of the increased likelihood of nitrogen narcosis and/or decompression sickness. Rather, however, we wish to highlight the physiological capacity to endure such depths when highly adapted to deep diving. Such data provide novel findings of tolerability to extreme levels of lung compression in world-champion divers.

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DISCLOSURES

P.N.A. acts as an unpaid scientific advisor to MediPines. None of the other authors have any conflicts of interest, financial or otherwise, to disclose.

AUTHOR CONTRIBUTIONS

A.P., Z.D., and P.N.A. conceived and designed the research; A.P., C.G., B.S., H.G.C., and I.D. performed the experiments; A.P. and D.B.K. analyzed the data; A.P., Z.D., and P.N.A. and interpreted the data; A.P. prepared the figures; A.P. and P.N.A. drafted the manuscript; A.P., C.G., B.S., D.B.K., H.G.C., O.B., I.D., Z.D., and P.N.A. edited and revised the manuscript and approved the final version of the manuscript.

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