Tetzlaff K[1], *Muth CM*[2]

Physiological Challenges and Adaptations in Competitive Freediving

Physiologische Herausforderungen und Anpassungen im Wettkampfapnoetauchen

REVIEW

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1. UNIVERSITY HOSPITAL TÜBINGEN, *Department of Sports Medicine, Tübingen, Germany*
2. ULM UNIVERSITY MEDICAL CENTER, *Department of Anesthesiology and Intensive Care, Ulm, Germany*

Summary

› **Competitive freediving** is associated with extreme physiologic challenges due to the effects of apnea, submersion in water, and increased ambient pressure. There has nevertheless been a continued progression of world records across freediving disciplines in recent years, with breath-hold times exceeding 10 minutes, and reaching diving depths beyond 200 meters of seawater. The main physiological determinants of breath-hold endurance are the available oxygen stores, i.e., intrapulmonary gas volume, inspired gas concentration, and rate of oxygen consumption. Other factors such as psychological and inherent factors contribute to the individual tolerance to an increasing respiratory stimulus with prolonged breath-hold duration.

› **A freediving athlete** must be able to tolerate apnea to stay underwater long enough to reach great distance or depth and resurface safely. Elite freedivers exhibit somewhat remarkable physiological adaptations such as a more pronounced diving-response and hypercapnia tolerance compared to non-divers, as well as metabolic and cerebrovascular adaptations. To reach great depths, elite freedivers employ a breathing technique called glossophyryngeal insufflation (GI) that is enlarging the ratio of total lung capacity to residual volume, thus, mitigating risk of lung squeeze and increasing intrapulmonary gas volume, and thereby enhancing available oxygen stores. Pulmonary anatomy and physiology could not be accounted to restrict the breath-hold diving depths achieved by competitive athletes.

› **However,** the acutely increased intrapulmonary pressure during GI is transmitted to the pulmonary vasculature, thereby eliciting a right ventricular pressure overload, and left ventricular dysfunction that may augment hypotension and syncope during glossopharyngeal insufflation. Due to extreme diffusion gradients between alveolar pN_2 and N_2 pressures in the body tissues at great depth, N_2 will be forced into blood and body tissues during descent, elevating risk of N_2 narcosis and decompression illness during deep dives. Breath-hold training and preparation have been shown to enhance breath-hold performance.

› **This review highlights** recent data on the profound cardiovascular, respiratory, and gas exchange effects that are observed in competitive freedivers, and discusses possible adaptations and risks for humans.

KEY WORDS:
Breath-Holding, Apnea, Hypoxia, Hypercapnia, Glossopharyngeal Breathing, Decompression Stress, Diving

Introduction

The term freediving (apnea-diving, breath-hold diving) refers to diving without the aid of external air supply, i.e., diving with a single deep breath while maintaining airways below the surface of the water. Freediving is the ancestral form of diving and has been practiced by humans since ancient times. Freediving populations such as the Ama divers of Japan and Haenyo of Korea for hundreds of years have employed special freediving techniques to harvest crops from the sea. Early scientific research on these freediving populations fostered our understanding of the human physiology and pathophysiology of breath-hold diving (55). However, this understanding has more recently been challenged by the performances achieved by competitive freedivers.

Competitive freediving as a leisure activity has gained popularity in the last 50 years only when single freedivers took up the challenge to explore the maximum depths humans could go to on a single breath. Formal freediving competitions were sanctioned by the international Confédération Mondiale des Activités Subaquatiques (CMAS), founded in 1959 with celebrated documentary filmmaker Jaques Cousteau. Italian Enzo Maiorca, first freediver to break the 50m barrier in 1961, and French Jacques Mayol vied with each other for the world record in a rivalry that inspired French director Luc Besson to make his 1988 film "The Big Blue", which in turn has inspired the current generation of competitive freedivers. The scientific community took note of these achievements when Mayol reached 62m and US freediver Robert Croft surpassed 64m in 1966, since these depths had previously been considered incompatible with human physiology (7, 58).

Today, competitive freediving has become a popular water sport where athletes compete for breath-hold duration, distance, or depth. Both CMAS and the Association Internationale pour le Développement de l'Apnée (AIDA), a non-profit organization created in 1992 to help develop freediving as a sport, hold national and international competitions in various disciplines both in pool and open water. Both organizations have created a stable set of rules and guidelines for freediving competitions and record attempts (table 1). The purpose of this review is to delineate the physiological changes and challenges as well as human adaptations associated with competitive breath-hold diving. ›

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CORRESPONDING ADDRESS:
Prof. Dr. Kay Tetzlaff
Department of Sports Medicine
University Hospital Tübingen
Hoppe-Seyler-Str. 6
72076 Tübingen, Germany
✉: kay.tetzlaff@klinikum.uni-tuebingen.de

Table 1

Competitive freediving disciplines as approved by CMAS (International Confédération Mondiale des Activités Subaquatiques) and AIDA (Association Internationale pour le Développement de l'Apnée). *Abbreviation used by AIDA, #discipline only approved by CMAS).

	DISCIPLINE	EXPLANATION
Open water	CWT	Constant weight: athlete must cover the vertical distance in apnea to the declared depth without any change in his weight during the whole attempt with fins.
	CWT-BF (CWTB*)	Constant weight with bi-fins: athlete must cover the vertical distance in apnea to the declared depth without any change in his weight during the whole attempt with the use of bi-fins only
	CNF	Constant weight no fins: athlete must cover the vertical distance in apnea to the declared depth without any change in his weight during the whole attempt without fins.
	FIM	Free immersion: athlete must cover the vertical distance in apnea to a declared depth without fins, allowed to use arms along the rope
	VWT	Variable weight: athlete must cover the vertical distance in apnea to the declared depth with a guided ballast device and return with his own power, leaving the ballast at depth
	Skandalopetra#	Athlete dives with the aid of a stone (usually a marble slab) attached to a rope
Pool	STA	Static apnea: athlete aims to perform a maximum breath-hold duration with body floating at surface and face submerged
	DYN	Dynamic apnea: athlete aims to cover the maximum horizontal distance while keeping the body below the surface
	DYNBF	Dynamic apnea with bi-fins: athlete aims to cover the maximum horizontal distance while keeping the body below the surface with bi-fins
	DNF	Dynamic apnea without fins: athlete aims to cover the maximum horizontal distance while keeping the body below the surface with no fins
	Speed apnea#	Athlete aims to cover a fixed distance in the shortest possible time, free style

Physiology of Breath-Holding at Depth

The Diving Response

An important reflex mechanism enabling humans to withstand asphyxia for prolonged dive duration is the mammalian diving response. It can be elicited by suspension of the respiratory excursions with high lung volumes as well as the stimulation of the facial branches of the trigeminal nerve upon contact with water (21). The diving response will be enhanced with cold stimulus (2) and has been found to be pronounced with face immersion (61). The response involves synergistic sympathetic and parasympathetic activation, catecholamine increase, and redistribution of blood flow to maintain adequate oxygen supply to hypoxia sensible organs (10, 25, 29). Heart rate in trained freedivers usually will drop to 40-60 beats per minute, however, bradycardia of 20-24 beats per minute has been reported in elite freedivers (16). Bradycardia is usually not accompanied by a similarly significant drop in cardiac output, so that peripheral vasoconstriction results in an increase in blood pressure. Dramatic increases in arterial blood pressure to values as high as 280/200 mmHg (systolic/diastolic) have been reported (17). An oxygen-conserving effect of bradycardia could be shown by an inverse relationship between heart rate and pulse oximetry, particularly during exercise (37).

Breath-Hold Endurance

Maximum breath-hold time is highly variable between and within subjects; healthy untrained subjects are normally able to achieve 1-2-minute breath-holds. Breath-hold training, however, has been shown to extend breath-hold duration through physiological factors such as increased lung volume and attenuation of oxygen consumption, and relaxation techniques as well as dietary restrictions. Trained freedivers achieve average submersed breath-hold times of about 5 min under resting conditions (11, 24), and elite freedivers have set records beyond 10 minutes; the current world record in static apnea approved by AIDA was set in 2009 by French freediver Stéphane Mifsud at 11.35 minutes (76). In healthy untrained subjects the breath-hold breaking point is governed by physiological stimuli such as hypoxia- and hypercapnia-induced chemosensation and removal of inhibitory pulmonary afferent nerve activity. Trained freedivers, however, can overcome the urge to breathe and maintain a closed glottis and mouth throughout increasing involuntary breathing movements of the diaphragm called the struggle phase of breath-holding (59). The determining physiological factor of a maximal breath-hold breaking point in elite freedivers therefore rests upon a critical level of hypoxemia before loss of consciousness (40).

Blood Gas Changes

The partial pressures of oxygen (O_2) and carbon dioxide (CO_2) are subject to pronounced changes during freediving. When breath-holding above the water surface the arterial or end-tidal partial pressure of oxygen pa/etO_2 falls below its normal level of about 100 mmHg and that of carbon dioxide pa/etCO_2 rises above its normal level of about 40 mmHg, following a linear time course. During freediving, however, the time course of the change in blood gases is largely affected by face immersion inducing a diving response, and compression of the diver's lungs during descent as well as decompression during the ascent from depth. Alveolar partial pressures of nitrogen (N_2), O_2 and CO_2 will rise with increasing depth according to the linear increase in total ambient pressure (table 2). Since mixed-venous partial pressures do not change at the same time, all three gases diffuse from the alveolar space into blood. For example, the arterial pO_2 will amount to 250 mmHg when reaching a depth of 20 m where total pressure is three times the atmospheric pressure at surface. While alveolar pCO_2 ($pACO_2$) will increase accordingly during descent, there is a paradoxical diffusion of carbon dioxide into blood due to its high diffusibility and high solubility in blood, leading to redistribution of CO_2 into well-perfused tissues of the organism in the sense of an acutely increased CO_2 storage capacity. This effect is enhanced by immersion-related redistribution of blood with an increase of intrathoracic blood of about 700-1000mL (3, 58). Accordingly, $pACO_2$ values of about 50 mmHg were measured at a simulated depth of 20m, although the alveolar $pACO_2$ should have risen to about 100 mmHg. During simulated hyperbaric chamber dives $paCO_2$ levels of 40-45 mm Hg were measured in two elite

Table 2

Relationship between depth, ambient pressure, corresponding oxygen and nitrogen partial pressures, and relative reduction of a given gas volume (m=meter sea water, ata=atmospheres absolute).

DEPTH	AMBIENT PRESSURE	OXYGEN PARTIAL PRESSURE	NITROGEN PARTIAL PRESSURE	GAS VOLUME
(M)	(ATA)	(ATA)	(ATA)	
0	1	0.21	0.79	1
10	2	0.42	1.58	1/2
20	3	0.63	2.37	1/3
30	4	0.84	3.16	1/4
40	5	1.05	3.95	1/5

freedivers during dives of 4-5 minutes at 20m (46). However, $paCO_2$ values before the dive were low at 30mm Hg because of hyperventilation (figure 1). During ascent from depth, i.e. decompression of the lungs, alveolar partial pressures of N_2, O_2, and CO_2 will decrease again. While for CO_2 the normal direction of flow is restored, pAO_2 may reach the mixed-venous value causing paradoxical diffusion of oxygen from the blood into the alveolar space. A diminished solubility of carbon dioxide in venous de-oxygenated blood during ascent (Haldane effect) will blunt a further rise of $paCO_2$ to extremely high levels. At the breaking-point of breath-holding in air, $petO_2$ is typically 62 mmHg and petCO2 is typically 54 mmHg; these changes may worsen the longer the breath is held. During deep breath-hold dives, however, hyperoxia at depth, rapidly declining paO_2 with decompression, reversed O_2 diffusion when pAO_2 falls below paO_2, and continued O_2 consumption will all contribute to risk of critical hypoxemia. At the end of deep freedives, arterial pO_2 values as low as 26 mm Hg have been measured after 42 m freshwater pool dives conducted with a sled for descent (50), with even further reduction if subjects exercised (fin diving). Measured paCO2 values, however, were more variable and closer to normal values after those experimental freedives and were higher after fin dives, according to an enhanced metabolic rate due to exercise (50). While the breath-hold breaking point normally would be reached by paO_2 falling below or $paCO_2$ rising above a certain threshold partial pressure, competitive freedivers have developed techniques and adaptations to keep $paCO_2$ below such a threshold, thus, relying on hypoxia as the main stimulus to break the breath-hold. Since hyperventilation before the dive will lower carbon dioxide stores and tension, competitive breath-hold dives usually start from a lower $paCO_2$ level, hence prolonging breath-hold endurance. Measured $petCO_2$ and $paCO_2$ as low as 18 mmHg and 21 mmHg, respectively, have been reported in competitive freedivers after prediving hyperventilation routine before submersed breath-holds or deep dives (50). Extreme hyperventilation before breath-holding entails an increased risk of reaching critically low hypoxemia without triggering a respiratory stimulus from rising $paCO_2$; this risk may be enhanced further when paO_2 decline is accelerated during ascent from depth.

Physiological Challenges During Deep Diving

Deep diving in apnea is associated with extreme physical challenges, and reaching great depths is only possible with special adaptations to these challenges. First, individual breath-hold endurance must allow the freediver to stay underwater long enough to reach these depths and resurface safely. Second, pressure equalization of the air-filled middle ear and the paranasal sinuses during descent must operate flawlessly and require only minimal additional air.

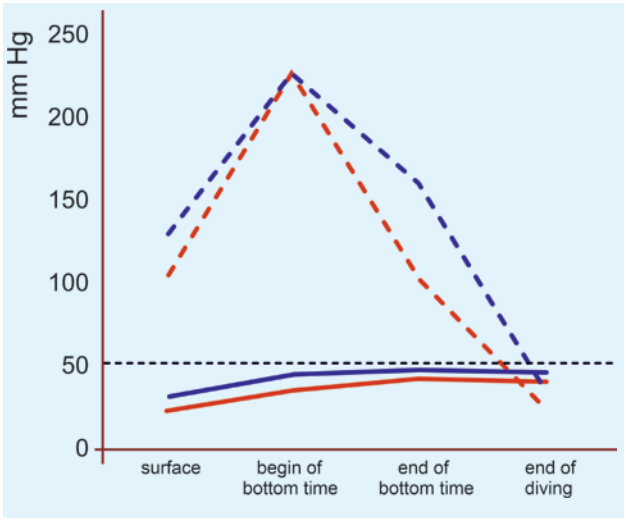

Figure 1

Measured arterial oxygen (dashed lines) and carbon dioxide (solid lines) partial pressures of two elite freedivers (diver 1=blue, diver 2=red) during a breath-hold dive to a depth of 20 meters.

Elite freedivers have partly developed special techniques to keep the amount of air required for active tube ventilation as low as possible (22). Third, the lungs and airways must be able to tolerate huge changes in volume and pressure during descent and prevent their lungs from suffering pulmonary negative pressure barotrauma (lung squeeze). It was previously believed that the ratio of total lung capacity (TLC) to residual volume of the lungs (RV) would determine the maximum depth divers would be able to reach safely (1, 7), however, more recent research revealed techniques used by elite freedivers to overcome physiological limits.

Lung Compression

According to Boyle's law, TLC decreases with reduction in ambient pressure and halves already at 10 m water depth. Theoretically, further reduction in TLC will only be possible until the RV is reached, as negative intrapulmonary pressure below RV would lead to the influx of blood and fluid into the alveolar space (7). For example, with an assumed TLC of 6 L and an RV of 1.5 L, the TLC/RV ratio of 4 would translate to a depth of 30m where total lung volume would be reduced to 1.5 L. Due to the non-linear relationship between lung volume reduction and chest wall pressure at depths beyond 20m, larger pressure changes are necessary to lead to further volume reduction. When compression of the thoracic gas volume is reaching RV, the increase in chest wall pressure at depth does not become abruptly critical, however, will lead to negative pressure barotrauma when the RV continues to decrease. Clinical consequences include collapse of lung areas (atelectasis), exsudation of tissue fluid into the alveolar space and/or rupture of alveolo-capillary membranes (39).

An important mechanism to prevent negative pressure pulmonary barotrauma is immersion mediated thoracic blood shift. Due to the influx of 750-1200 mL of blood into the pulmonary pathway, compression of the lungs to a much lower lung volume than the residual volume is possible (7, 58). In case of the above example of a given TLC of 6 L and an RV of 1.5 L, considering an assumed additional intrathoracic blood volume of 1 L, this would result in an equivalent reduction of the residual volume to 0.5 L. Thus, a compression ratio of 12 instead of 4 (without consideration of blood shift) become possible, corresponding to a maximum depth of 110 meters of seawater. The TLC/RV ratio can be enlarged even further by inhaling additional amounts of air through a complex swallow-breathing maneuver called glossopharyngeal respiration ›

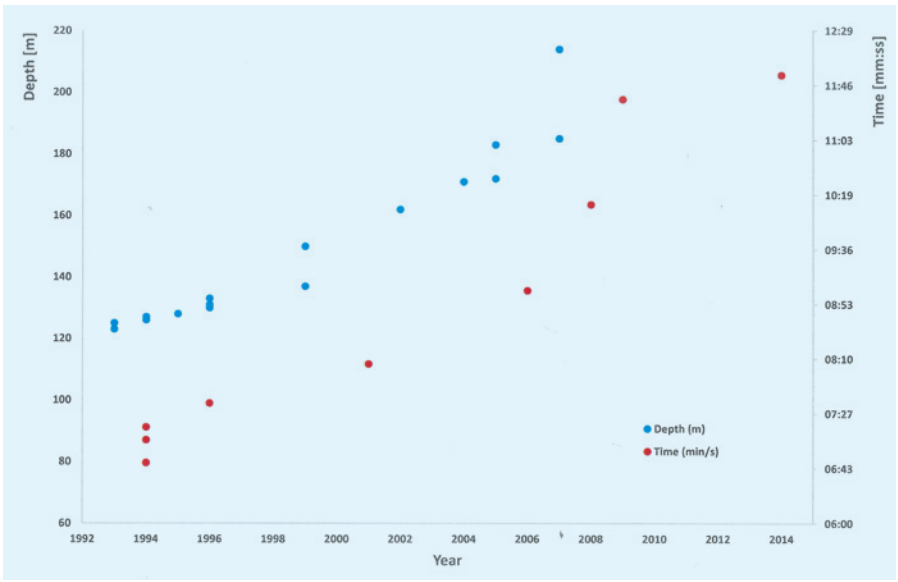

Figure 2

World records as documented by AIDA for static breath-hold duration in minutes (red dots) and depth (blue dots).

(GI). GI has been adapted from a technique previously used to keep patients with neuromuscular diseases with impairment of the inspiratory respiratory muscles off ventilators for as long as possible. In post-polio patients, employment of GI led to average increases in vital capacity of 700 mL (6). More recently, the use of GI was reported among swimmers and elite freedivers. In swimmers, GI could improve buoyancy and maximum inspiratory capacity, especially in short-distance swimming (48). In breath-hold diving, GI is primarily intended to improve the TLC/RV ratio to penetrate to even greater depths and prevent negative pressure barotrauma of the lungs (38, 47). However, GI will also increase available oxygen stores and will allow to compensate for air needed to equalize cranial air cavities (ears, sinuses) during descent.

The breathing maneuver comprises a sequence of air pumping cycles that will be performed with the soft palate closed against the nose to minimize air escaping. Each cycle entails maximum inhalation, filling of the mouth-throat with air and closing the mouth while the vocal folds are closed. The compressed air in the oral cavity is subsequently pressed into the lungs (swallowed) by means of the buccal muscles, with the vocal folds now open. The increase in intrapulmonary pressure is maintained by vocal fold occlusion. By applying this technique, elite breath-hold divers may be able to increase their TLC by up to 47% with wide interindividual variability of the volume increase and number of buccal pumping cycles employed to reach this volume (24, 41, 49, 67). Imaging studies showed that the increase in lung volume is mainly achieved by widening of the thorax, flattening of the diaphragm, opening of the costodiaphragmal recessus and herniation of the lung below the xiphoid (12, 38). Thus, TLC is not equivalent to maximum lung volume, and due to its enormous distensibility the lungs can stretch to above normal volumes. However, GI exerts considerable mechanical stress on lung elastic properties. Increases in intrapulmonary and transpulmonary pressures up to 109 cmH_2O and 80 cmH_2O, respectively, have been measured during GI maneuvers (41), indicating that some individuals can withstand transpulmonary pressures and volumes far greater than those to which lungs would normally be exposed to. Quantitative analyses of pulmonary and central blood volume changes revealed an almost 50% decrease of central blood volume after lung hyperinflation in the non-immersed state (43). The lung hyperinflation results in translocation of blood from the central blood volume into abdominal, pelvic, and peripheral veins (44). Some athletes also use another maneuver called glossopharyngeal exsufflation to suck air into the mouth at great depth, as respiratory muscles will not be able to provide the force needed against the pressure difference. This air will be needed to equalize middle ear pressure when going deeper. Elite freedivers have meanwhile reached depths beyond 200 meters of seawater with the record set by Austrian freediver Herbert Nitsch at 214 m in 2007 (figure 2).

Lung Atelectasis and Hypoxemia at Depth

Changes in blood gases at depth will be affected not only by changes in ambient pressure, but also alterations of the pulmonary alveolo-vascular interface. More recently it could be demonstrated that relative hypoxemia can occur at maximum depth, presumably due to pulmonary gas exchange abnormalities caused by lung compression. Brachial artery pO_2 measured in a freshwater tank at 40 m depth was decreased in two out of six trained freedivers while the other divers showed a mean increase of 181% in their arterial paO_2 (5). Hypoxemia occurred in one freediver whose paO_2 declined from 97 mmHg to 61 mmHg at depth and further decreased to 53 mmHg with an oxygen saturation of 88% when surfacing (5). In another study analyzing arterial blood gases and investigating lung ultrasound immediately before, at depth, and immediately after breath-hold dives to 15m and 42m, hypoxemia accompanied by alterations of lung parenchyma were detected in some divers, specifically signs of a small area of re-expanded parenchymal atelectasis (50). These findings indicate that the traditionally predicted hyperoxemia is not consistently present at maximum depth in all freedivers, and that lung volume reduction is not necessarily uniform and could instead result in regional atelectasis, providing a mechanism for intrapulmonary right-to-left shunt and hence explain the lower than predicted paO_2.

Decompression Stress

The observation that marine mammals dive routinely and repeatedly to impressive depths without obvious decompression injury reinforced scientific belief that human breath-hold diving would not entail any risk of decompression sickness. However, recent evidence from postmortem studies in stranded whales (28, 45) suggested that decompression sickness may occur because of irregular ascent from depth (15). It was hypothesized that behavioral adaptations and physiological peculiarities such as the reduction in blood flow to nonessential tissues and a progressive collapse of alveoli would prevent marine mammals from nitrogen uptake and thereby minimize decompression stress. However, tissue N_2 measurements have demonstrated that these mammals have obviously adopted strategies to manage diving with increased N_2 load (15, 26).

In humans, risk of decompression sickness arises from exposure to rapid ambient pressure reduction when body tissues are saturated with N_2, an inert gas constituting 78% of ambient air. During dives with self-contained underwater breathing apparatus, according to Henry's law, N_2 will dissolve in body tissues due to the pressure gradient between ambient pressure and lower tissue N_2 partial pressures (pN_2). Gas transfer into tissues will continue with time until equilibrium between tissues and ambient N_2 inert gas pressure. Washout of N_2 from tissues follows similar kinetics when

ambient pressure is reduced; however, according to Haldane's laws N_2 elimination is in principle slower than absorption, in particular from tissues with slow gas elimination kinetics. Consecutive tissue N_2 accumulation during ascent may cause supersaturation and, eventually, gas phase formation. In breath-hold diving, however, N_2 uptake from alveolar air is restricted to the available amount of gas in the lungs before the breath-hold. Eventually alveolar pN_2 will increase during descent and drive N_2 into the tissues. During ascent from depth, N_2 will be coming out of solution and diffuse back into the alveolar space. As N_2 release from tissues is slower than uptake, repeated breath-hold dives with a short surface interval may lead to the accumulation of N_2 in body tissues. There is accumulating evidence that repetitive freediving to even shallow water depths of 15-25 meters seawater as practiced still by few professional freedivers is associated with risk of decompression sickness (32, 65).

Seafood gatherers such as the Ama divers of Japan make up to 50 breath-hold dives per working shift with durations of about 1 minute and surface times of 1-2 minutes, going to a maximum depth of 25 meters. Measurements in Haenyo freedivers of Korea showed elevated pN_2 in brachial vein at the end of diving shifts with subsequent decline according to Haldanian desaturation kinetics (54). There has been anecdotal evidence from freedivers suffering neurological symptoms after extreme repeated free-dives: a neurological syndrome called "taravana" had been described in breath-hold divers of the Tuamotu Archipelago (8). These divers performed repetitive dives to depths of 30-40 m with only short surface intervals and occasionally developed post-dive neurological events such as vertigo, nausea, paralysis, and unconsciousness, some of which were fatal. A Danish physiologist reported symptoms such as nausea, dizziness, progressive visual disturbances, and unilateral paresis after repetitive freedives to 20m depth, and modelling of N_2 tissue saturation supported the idea of reaching critical tissue nitrogen concentrations after frequent repetitive freedives with short surface intervals (51). Intravascular gas bubbles have been detected by Doppler measurements in Ama divers after a series of repetitive dives to depths of 20m (35), and tear film bubble formation has been reported in submarine escape tank divers after repeated breath-hold dives to 30m (69). Cases of stroke-like incidents after repetitive shallow-water dives have been reported from Ama divers (31). These divers had neither vascular diseases nor risk factors for stroke, and the magnetic resonance imaging findings were consistent with a vascular pathogenesis of the lesions, i.e. occlusion of cerebral arteries. In a population survey 6.9% of Ama divers reported stroke-like neurological events during or immediately after repetitive breath-hold diving (71).

Anecdotal evidence and theoretical considerations support the hypothesis that adverse consequences of elevated N_2 may also apply to single deep breath-hold dives. Accordingly, signs of nitrogen narcosis have been reported frequently after ascent from deep breath-hold dives (36, 74), confirming N_2 diffusion into tissues at depth. Time of ascent from great depth may be insufficient to allow elimination of evolving N_2 gas bubbles. Theoretical considerations predict risk of decompression sickness negligible until reaching depths of about 100m, beyond which it will increase nonlinearly (20). Since human lungs do not collapse at shallow water depths as is the case with diving mammals, gas diffusion will continue until minimum compressed lung volume is reached at a depth around 230 meters (19). Beyond this depth, complete pulmonary shunt will cease N_2 equilibration between alveoli and arteries, see end of paragraph. Since peripheral tissues will be protected from N_2 uptake due to vasoconstriction, evolving gas bubbles may primarily embolize cranial vessels that continue to be well perfused during prolonged breath-holds. Hence cases of decompression sickness in freedivers exclusively involve neurological symptoms such as stroke-like syndrome and have been reported after breath-hold dives to depths beyond 100m in otherwise healthy young subjects with no history of cardiovascular disease or other pertinent risk factors (20, 62, 73). An obvious hypothesis is that due to atelectasis formation of the lungs, intrapulmonary arterio-venous anastomoses open at depth, through which gas bubbles located in the venous circulation radiate into the arterial system and cause cerebral arterial gas embolism (66).

Compromised Cardiac Function

Prolonged breath-hold, depth, and lung hyperinflation through GI will all contribute to hemodynamic stress on the cardiovascular system during deep freedives. Prolonged apnea alone causes massive left ventricular dilatation and an increase in norepinephrine levels (11). A significant reduction in cardiac output (due to a decrease in both heart rate and stroke volume) and in left ventricular diastolic and systolic volumes, consistent with a preload reduction could be demonstrated by echocardiography at 5 m depth (42). Lung hyperinflation through GI is augmenting cardiac function impairment with acute biventricular systolic dysfunction consistent with an acute pressure overload (52). Studies using magnetic resonance imaging techniques demonstrated a substantial decrease in pulmonary hemodynamic parameters during GI associated with an increase in lung volume (13). The hemodynamic changes mimicked the changes seen in pulmonary arterial hypertension, however, were mostly reversible after the cessation of voluntary lung hyperinflation. Another study confirmed that submaximal glossopharyngeal insufflation decreased ventricular volumes and cardiac output further, albeit by a small amount, and right ventricular end-diastolic volume remained reduced under these conditions (4).

Importantly, during breath-holding at TLC, changes in cerebral blood flow are observed only transiently during the early stages of breath-holding while blood flow and oxygen delivery are maintained at extreme apnea (70). It is postulated that whilst cardiac output is important in regulating cerebral hemodynamics, the role of mean arterial pressure in restoring cerebral perfusion pressure is of greater significance to the regulation of cerebral blood flow.

Human Adaptations to Competitive Breath-Hold Diving

As outlined above competitive freedivers are exposed to extreme physiological challenges at depth. Anecdotal reports from elite freedivers that they observed individual breath-hold times of several minutes before starting their career do indicate a possible genetic predisposition to breath-hold performance (Tom Sietas, personal communication). However, to improve performance, freedivers use adaptive techniques that increase their lung capacity, reduce metabolic rates, and improve their breath-hold tolerance.

Two weeks of daily breath-hold training have been shown to improve breath-hold time with earlier onset of the diving response. Apneas with identical durations resulted in less arterial oxygen desaturation after training, indicating prolonged O_2 conservation by the enhanced diving response (14). More experienced competitive freedivers in general have a stronger diving response characterized by a more pronounced bradycardia and larger increase in blood pressure when compared with untrained control subjects. Elite competitive freedivers also exhibit greater increases in cerebral blood flow, higher cerebrovascular reactivity (30) and can better tolerate hypoxemia and hypercapnia. A blunted ventilatory chemosensitivity to hypercapnia at rest and post-exercise that is distinct from scuba divers and controls has been reported (23, 57, 72). This diminished chemosensitivity to CO_2 has also been reported in other diving groups, such as synchronized swimmers, underwater hockey players, and trained young competitive swimmers, indicating that sub-aquatic training with repetitive breath-holding may induce peripheral chemoreflex desensitization. ›

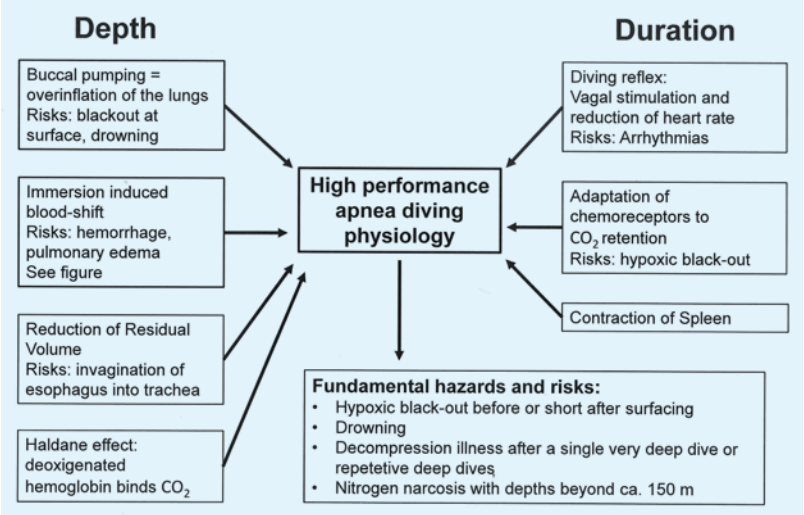

Figure 3

Overview of depth and breath-hold duration affecting breath-hold performance and associated risks and hazards.

It has been suggested that breath-hold training can accentuate splenic contraction during a breath-hold and increase baseline spleen size. In fact, breath-hold performance has been correlated with spleen size and trained freedivers have been shown to have larger spleens than matched control subjects (63), indicating a possibility to enhance oxygen stores and the ensuing increase in hematocrit, as has been reported in diving mammals. Modest increases in hematocrit have been reported in Ama divers and experimental laboratory settings with inconsistent results (27, 53, 60), thus, questioning the clinical importance. However, a wide range of the amplitude of the spleen volume reduction after repeated apneas with or without face immersion has been observed. Hence a greater splenic contraction with subsequently greater emptying of oxygenated blood and thereby an increase in oxygen transport capacity in the blood could contribute to prolonged breath-hold endurance in trained freedivers. It has been postulated that the rapid onset of the splenic contraction after simulated apneas suggests a centrally mediated feed-forward mechanism rather than the influence of slower peripheral triggers (34).

Greater lung volume will increase the amount of oxygen available for a breath-hold dive. Accordingly, a 15% higher vital capacity was measured in professional Ama divers compared to non-diving Japanese women (18) which was attributed to an increase in force by the inspiratory muscles. Measured lung volumes in competitive freedivers are either greater than non-diving control subjects or exceed significantly the calculated predicted values (38 ,49, 72). Similar respiratory adaptations have previously been confirmed for competitive swimmers (9) suggesting that years of swimming training and/or the earlier age at which training begins may have a significant influence on subsequent forced expiratory volume in one second (FEV1) and swimming performance (56). It remains unclear, however, to what extent genetic factors and inherited pulmonary traits may modify the improvement in lung volumes in aquatic sports. In a 3-year lung function follow-up of four competitive freedivers, an increase was seen in ventilatory volumes that was attributed to repetitive GI training (75); one case report showed an increase in lung volumes over eight years (68). Acute improvements in vital capacity were reported after 6-11 weeks of GI training (64). However, breath-hold training alone may improve lung volumes and competitive performance. FEV1 could be increased throughout a 3-month apnea training program, with concomitant increases in maximal oxygen uptake and arterial oxygen saturation during an incremental exercise testing (33). It has been postulated that apnea training improves effectiveness at both peak exercise and submaximal exercise and can also improve swimming technique by promoting greater propulsive continuity (34). Thus, training-induced adaptations may provide a partial explanation of the greater breath-hold performance seen in competitive freedivers compared with less trained freedivers and non-divers.

Evidence for thermal adaptation came from freediving populations such as the Ama of Japan who traditionally dived wearing cotton suits leaving their body in direct contact with water. It was reported that the Ama's critical water temperature threshold, i.e. the minimal temperature at which a human can stay immersed into water for 3 h without shivering, was lower when compared to non-diving controls. It was postulated that the higher shivering threshold was indicative of either stronger peripheral vasoconstriction or more effective countercurrent heat exchange (18). This adaptation to cold is independent of freediving and, interestingly, was lost after the introduction of neoprene wetsuits in the 1980's.

Finally, a variety of additional measures have been reported that may improve maximal breath-hold duration such as preconditioning (preparatory breath-holds) and relaxation techniques to attenuate oxygen consumption as well as dietary restrictions and supplementation.

Conclusions

This review has provided an overview of the pertinent physiological challenges that competitive freedivers are being exposed to during both training and competitions and outlined mechanisms and adaptations to improve breath-holding performance (figure 3). The article also delineates the stress responses associated with deep freediving, which are less well understood. While glossopharyngeal insufflation allows athletes to dive longer and deeper than previously believed physiologically possible, narcosis and decompression stress may put individuals at serious risk. Future studies need to fully elucidate (patho-)physiological thresholds that should not be surpassed by athletes and evaluate possible long-term consequences of continued breath-hold training.

Conflict of Interest

The authors have no conflict of interest.

Summary Box

- This article provides an overview of the pertinent physiological challenges during freediving training and competitions and delineates the pathophysiological stress responses associated with deep freediving, which are less well understood.
- Human adaptations to freediving such as enlarged spleen and lung size, a pronounced diving response and diminished chemoreceptor sensitivity help to increase freediving performance. However, athletes need to be aware of physiologic thresholds and risks of possible maladaptations.

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