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The physiology and pathophysiology of human breath-hold diving.

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ABSTRACT

This is a brief overview of physiological reactions, limitations and pathophysiological mechanisms associated with human breath-hold diving. Breath-hold duration and ability to withstand compression at depth are the two main challenges which have been overcome to an amazing degree as evidenced by the current world records in breath-hold duration at 10:12 min and depth of 214 m. The quest for even further performance enhancements continues among competitive breath-hold divers, even if absolute physiological limits are being approached as indicated by findings of pulmonary edema and alveolar haemorrhage post-dive. However, a remarkable, and so far poorly understood, variation in individual disposition for such problems exists. Mortality, connected with breath-hold diving is primarily concentrated to less well trained recreational divers and competitive spear fishermen who fall victim to hypoxia. Particularly vulnerable are probably also individuals with pre existing cardiac problems and possibly, essentially healthy divers who may have suffered severe alternobaric vertigo as a complication to inadequate pressure equilibration of the middle ears.

The specific topics discussed include the diving response and its expression by the cardiovascular system which exhibits hypertension, bradycardia, oxygen conservation, arrhythmias and contraction of the spleen. The respiratory system is challenged by compression of the lungs with barotrauma of descent, intrapulmonary hemorrhage, edema and the effects of glossopharyngeal insufflation and exsufflation. Various mechanisms associated with hypoxia and loss of consciousness are discussed, including hyperventilation, ascent blackout, fasting and excessive post exercise oxygen consumption. The potential for high nitrogen pressure in the lungs to cause decompression sickness and nitrogen narcosis is also illuminated.

Key words: apnea, diving response, hypoxia, glossopharyngeal insufflation, pulmonary edema

INTRODUCTION

Breath-hold diving, primarily for food gathering, is still practiced in Japan and Korea much the same way as documented in up to 2000 year old Japanese art and literature (65).

While the performance of the aforementioned diving women and men (a.k.a. Amas) is impressive in terms of number of dives per workday, the maximal depth (~20 meters) and duration (~one minute) are less so, compared to the records set by male and female competitive divers of recent days. The current maximal breath-hold duration of a person resting face down in a swimming pool ("Static Apnea") is 10:12 minutes (www.aida-international.org) and the depth record of a person being pulled down to depth by a weight and returned to surface by an inflatable "lift bag" ("No Limits"), in the course of 4:24 minutes is 214 meters (www.aida-international.org).

How is it that, without any obvious specific evolutionary pressure, physiological coping mechanisms have developed that allow these diver-athletes to endure extreme hypoxia and an up to 22-fold compression of the lung gas volume? These mechanisms and what limits them are the topics of this brief review. For earlier full-length reviews see references: (29, 30, 42, 63).

The two main challenges in breath-hold diving are duration and its connection with hypoxia and depth causing mechanical strain on air containing body cavities by compression. A third challenge is the exposure to high gas pressures with potential pharmacological/toxic effects.

The Diving Response

The diving response, exhibited by all air breathing vertebrates is elicited by apnea and consists of peripheral vasoconstriction due to sympathetic activity, connected with initial hypertension, and a vagally induced bradycardia with reduction of the cardiac output (cf. Fig 1). These circulatory changes are further strengthened by cooling of the facial area and/or hypoxia. In particularly responsive subjects, apnea has been noted to elevate peripheral circulatory resistance up 4-5 times concomitant with correspondingly intense bradycardia and reduced cardiac output (28, 47, 53). The bradycardia may be part of a reflex response to apnea but there are observations that the blood pressure increase precedes the slowing of the heart frequency (cf Fig1) suggesting that baroreflex activation plays a role in the development of the bradycardia as may chemoreceptor stimulation from hypoxia during the later part of the breath-hold (44, 45). It is generally held that the diving response causes blood and lung oxygen stores to preferentially be distributed to the heart and the brain. Another sympathetic reflex which is part of the multifaceted diving response (cf. Fig 2) has attracted attention in recent years, namely the effect of breath-hold dives to enhance the hemoglobin concentration of circulating blood by splenic contraction (7, 26, 34, 77), which occurs early during the diving-response cascade, actually preceding the bradycardia (7). Healthy, splenectomized persons do not exhibit the hemoglobin increase when breath-holding (77). Higher hemoglobin levels in breath-hold divers than in non-divers were found in one study (21), which suggested long-term enhancement of hemoglobin levels in breath-hold divers may be a consequence of an observed 24% increase in erythropoietin levels (22). These findings contrast with another study in which elite breath-hold divers had normal hemoglobin concentrations and total hemoglobin mass (73) . To the extent that breath-hold divers do have increased hemoglobin levels it may be of the same nature as in persons suffering from obstructive sleep apnea in whom the blood changes reportedly correlate with the severity of hypoxia during sleep (12)

although, between the two groups, there are obvious quantitative differences in the exposure to hypoxia.

Cooling of the face, in particular the forehead and the eye region is particularly effective in eliciting bradycardia (6, 80). However, due to a marked increase in metabolism, breath-holding time during whole body immersion in cold water (20ºC) was 55 % shorter than in the thermoneutral non-immersed condition even if the heart rate was reduced by 26% in the water (82).

The diving response is highly variable among humans during both rest (6, 76) and exercise (47, 53). Systematic differences also exist depending on age and presence or lack of diving experience. The diving bradycardia is quite pronounced in children 4-12 months of age (33) and may have survival value during hypoxic episodes proximal to birth. The diving response weakens with advancing age and is more marked among habitual breath-hold divers than non-divers (76). The diving response is relatively more pronounced during exercise than during rest (11, 84). The oxygen sparing effect appears to be proportional to the degree of the bradycardia as exemplified in Fig 3. and statistically confirmed in several studies during exercise (3, 4, 47, 51, 55, 90).

During rest, the potential to conserve oxygen is less pronounced but a reduction in pulmonary oxygen uptake has been shown in breath-holding resting humans and more so in experienced divers than in non-diving controls (2, 27) which might help to explain the earlier mentioned extremely long-lasting breath-holds during “Static Apnea “competitions.

Arrhythmia:

A remarkable feature of the human diving response is the combination of bradycardia with cardiac arrhythmias, due to vagal inhibition of atrio-ventricular conduction combined with sympathetically induced enhancement of automaticity in other latent pacemakers being conducive to ectopic beats. Other arrhythmogenic factors likely to operate during breath-hold diving are face immersion in cold water, distension of the heart due to large intra-thoracic blood volume, and a large afterload. Given that the diving response is thought to favor myocardial (and brain) perfusion (10) it is noteworthy that one study has found ECG changes indicative of sub-endocardial ischemia immediately post-dive (68). These changes, which were absent in non-submersed breath-holds, included (but were not limited to) ST depression, heightened T wave and slowed repolarization with addition of a positive U-wave to the QRS complex. Cardiac rhythm disturbances were first recorded in pearl divers by Scholander et al in 1962 (79). In a later study, submersed wet dives to 55 m in a pressure chamber were performed by three experienced breath-hold divers (all from the same family); after an initial tachycardia, their heart rates fell to 20-30 beats/min near the "bottom". The longest R-R intervals corresponded to instantaneous heart rates of 8, 13 and 24 beats/min. Furthermore, a very high frequency of premature and inhibitory arrhythmias were recorded during their dives in cool (25ºC) water while such disturbances were much less prevalent in dives in thermoneutral (35ºC) water (28). Marked but asymptomatic bradycardia (temporarily 5.6 beats/min) induced by apnea with cold water face immersion has also been reported (6) (for a review of these influences see Ferrigno and Lundgren pp. 161-165 (29)).

Hypoxic loss of consciousness

Obviously the breath-hold diver must surface before hypoxia causes loss of consciousness. Unfortunately, breath-hold divers drown every year, frequently falling victims to now largely well understood and preventable patho-physiological mechanisms.

Hyperventilation:

Hyperventilation (a respiratory exchange ratio higher than the respiratory quotient (RQ)) before the dive reduces CO_2 stores in blood and tissues so that the breath-hold dive begins in a state of relative hypocapnia while the oxygen stores, mostly in the lungs, may have increased by a modest 250-300 ml, i.e enough for an additional 10-60 seconds of breath-holding, depending on physical activity. Thus the CO_2 drive to return to the surface to breathe is delayed and loss of consciousness ensues without forewarning since the week respiratory stimulus from hypoxia is easily voluntarily overridden (17, 18). To exemplify: a study of diving fatalities in South Africa reported a fatality rate in 24 scuba accidents at 29% while out of 14 breath-hold diving accidents half were fatal causing the author to call the latter type of diving ”the most dangerous diving activity” (39). Nonetheless, athletes competing for duration in immersed breath-holding (Static Apnea) typically hyperventilate extensively before performances, yet only about 10% surface with symptoms of severe hypoxia such as loss of motor control or, rarely, loss of consciousness (46). They apparently determine the duration of their breath-hold by means other than the hypercapnic ventilatory drive. Some may react to hypoxia via the albeit weak urge to breathe, while others use faltering vision (“grey-out”) as a clue and even read the wrist watch to break the breath-hold in time. End-tidal gas tensions have been monitored in competitive breath-hold divers’ first expiration after maximal Static Apneas. In four divers the pre breath-hold PCO_2 was about 2.7 kPa (20mmHg) and, immediately post breath-hold it was normocapnic at 5.1kPa (38 mmHg) while PO2 ranged from 2.6 to 3.1 kPa (19.6 - 23.6 mmHg), (52). In another study, the breath-holds ended with PO_2 levels at 3.5±0.8 kPa (26±6 mmHg) and PCO_2 at 6.5±0.5 kPa (49±4 mmHg). The same divers ended swimming breath-holds with essentially the same oxygen tensions, i.e. a PO_2 3.3±0.8 kPa (25±6 mmHg) and a slightly hypercapnic PCO_2 of 7.5±0.9 kPa (56±7

mmHg), (69). Thus these divers were well below a P_AO_2 of 4 kPa (30 mmHg) which, based on observations of high altitude hypoxia, is usually considered borderline for maintaining consciousness (25, 59), cited without reference to original laboratory studies.

Ascent blackout (hypoxia of ascent):

Hypoxia of ascent is caused by the reduction of the water pressure acting on the chest and hence the lung gas pressure. To appreciate the quantitative aspects of this mechanism it is useful to recall the relationship between gas pressure and volume described by Boyle's law. At the surface the pressure is 1.0ATA (atmospheres absolute) while each 10 m of depth adds another 1.0 ATA to the pressure on the chest and therefore to the gas pressure in the lungs. Thus, at 40m the alveolar gas pressure is 5 ATA and, disregarding minor volume changes due to O_2 and CO_2 exchange, the lung gas volume is 1/5 of the initial volume at the surface as long as the diver remains at that depth. Accepting, for the sake of illustration, an alveolar PO_2 of 8 kPa (60 mmHg) as adequate for reasonable mental function this oxygen partial pressure will be reached when the oxygen fraction in the alveolar air has been reduced by metabolism to 1.6% (41, 57). Assuming that an alveolar/arterial PO_2 of 2.7- 3.3 kPa (20-25 mmHg) (52, 69) will cause loss of consciousness (LOC) this situation will occur when the diver, during ascent, reaches between 7 and 11 m of depth. This is arrived at by the following simple calculation, in which water vapor pressure in the alveoli is 47 mmHg; ([alveolar gas pressure at LOC] x 760 - 47)x0.016 = 20 or 25; .this yields an alveolar (i.e. total pressure) on the chest of 1.7 or 2.1 atm corresponding to 7 and 11 m of depth, respectively. In reality the LOC is likely to happen at somewhat lesser depth because of the circulation time between the lungs and the brain. Nonetheless it is not surprising that drowning incidents with apparent connection to hypoxia of ascent are a relatively common among competitive spearfishermen

who are known to operate at relatively deep depths and practice extended breath-holds in pursuit of their game (38) Landsberg P. G. Fin Diver, 33:20,1974) quoted by (39).

Carbohydrate depletion:

Prolonged periods of physical work deplete the carbohydrate stores (glycogen) in the body, which forces the body to compensate by increasing the rate of lipid (fat) metabolism. When the human body burns fat to produce energy, it uses 8% more oxygen than when metabolizing carbohydrates. Also, 30% less CO_2 is produced by fat metabolism. Thus, a breath-hold hold diver who has depleted the glycogen stores will become hypoxic faster and, making the situation worse, the CO_2 driven stimulus to breathe will be delayed. A dive that could safely be performed in a rested and well-fed state may be dangerous after a long day of exertion from diving or land-based activities (50). Carbohydrate intake has been shown to reduce breath-hold durations, due to more rapid CO_2 generation (because of a higher RQ) in subjects who had fasted for 18 hours, suggesting that that the risk could be reduced by proper carbohydrate intake and that breath-hold diving on an empty stomach may be dangerous (48).

Barotrauma of Descent (lung squeeze)

If a diver inhales to total lung capacity which, for a realistic exemplification is 10L, and descends to 200 m (21 ATA of pressure) that gas volume is, according to Boyles law, compressed to 0.48L - for an easily envisioned analogy this compression ratio of 21/1 corresponds to a large beach ball (diameter 39 cm) being reduced to the size of a grapefruit (14 cm) and that does not even account for the unknown amount of gas being dissolved in the blood. Clearly, there must be physical limits to the deformation that the chest can endure and the expansion of blood containing structures within the chest can withstand. A schematic of the dimensional events at play is shown in Fig 4.

There are three possible outcomes of an excessive ambient pressure on the chest-lung complex during apnea: collapse of parts of the lung with atelectasis formation, fluid filtration into the airways and alveolar space, and alveolo-capillary membrane rupture with bleeding into the void spaces. Indeed, among competitive breath-hold divers there are reports of symptoms suggestive of pulmonary edema after deep dives (13, 31, 56), with some cases needing in-hospital oxygen treatment (personal observations by author PL). There are also reports of haemoptysis after breath-hold diving (9, 36) and in one study, pulmonary edema was confirmed with chest x-ray after dives to 30 m in the sea (72). Pulmonary edema has also been reported in connection with both surface swimming (1) and SCUBA diving (37). Simple head-out immersion has been found to induce a 0.7L redistribution of blood from the periphery into the chest (5). It is highly likely that the potentially much greater pressure differences between the thoracic space and the rest of the body during breath-hold diving cause larger blood redistribution into the chest, thus increasing the capillary engorgement. Extravasation of fluid has been confirmed in conjunction with surface swimming and scuba diving which, in all likelihood, impart lesser trans-vascular stresses. A sufficient pressure difference between the blood pressure in the pulmonary capillaries and the intra-alveolar gas pressure may cause stress failure (91) with leakage of fluid and blood into the lungs, similar to hydrostatic or cardiogenic pulmonary edema (89). A transpulmonary capillary pressure as low as 24 mmHg may cause capillary leakage, and is likely to be marked at pressures of 40 mmHg (91). Simple head-out immersion in subjects sitting upright has been shown to cause a redistribution of blood from the periphery into the chest of about 0.7 L and a more than four-fold increase in mean pulmonary arterial pressure (from 5 to 22 mmHg, n=3). Thus it seems reasonable to assume a considerable transfer of blood (75) and/or increase in pulmonary vascular pressure due to the extraordinary hydrostatic pressure differences across

the chest which are predictable in deep breath-hold diving. On the assumption of the blood volume in the pulmonary vessels increasing by 1.0L in a deep breath-hold dive and applying published estimates of pulmonary vascular compliance which range from 0.42 ml (60) to 1.2 $ml \cdot mmHg^{-1} \cdot kg$ body weight (3.15 to 9.02 $ml \cdot kPa^{-1} \cdot kg$) (24) it has been calculated that, in a 75kg person, this would yield increases in pressure in the pulmonary vascular bed in the range of 11- 32 mmHg (1.47- 4.27 kPa) (29). If there is a corresponding capillary pressure increase it might well be conducive to pulmonary edema. It is important in this context that even the shallow immersion of the chest during swimming at the surface, given enough time, has the potential of causing severe pulmonary edema (1, 37). That symptom-producing pulmonary edema is not frequently observed in breath-hold diving is probably due to the short duration of abnormal blood distribution compared to surface swimming and scuba diving.

To simulate the thorax/lung-squeeze effects of deep diving in the ocean, competitive breath-hold divers have invented a more convenient training method: they dive in shallow water, often in a swimming pool after complete exhalation to residual volume (49). In an experimental study on this diving method it was shown that dynamic spirometry readings were reduced post diving and slight haemoptysis was noted in two subjects (49). In that study, dives to 5 m depth after exhalation to residual volume was calculated to simulate a compression of the chest equivalent to a depth of 91m.

In a recent field study at a breath-hold diving competition in cold water it was reported that some breath-hold divers had reduced forced expiratory volumes and reduced arterial oxygen saturation after deep diving (56). These measurements should however be interpreted with some caution because finger probe pulse oximetry is sensitive to cold, with reduced

saturation due to reduced blood flow so that it is not representative of central blood oxygen saturation (14, 15). Furthermore, cold stimulation has been shown to induce bronchoconstriction (62).

It is still unclear whether the blood streaks and minor bleedings, that many divers report, are alveolar in origin or from other areas of the respiratory system. The laryngoscopic study by Lindholm et al (49) showed that the blood originated below the vocal cords. Mechanical shear caused by voluntary diaphragmatic contractions to counteract urge to breathe has also been suggested as a cause of haemoptysis by Kiyan et al. (36) which may be explained by the decrease in intrathoracic pressure that accompanies a contraction (43), causing further strain on the alveolo-capillary barrier. The long term complications or adaptations to repeated bleeding and pulmonary edema are not known.

It should also be noted that the blood pressure rises during breath-holding, and more so in some subjects, when the heart rate and cardiac output is not reduced in proportion to the increase in peripheral resistance (28, 53, 85). The hypertension could possibly contribute to edema.

Glossopharyngeal insufflation and exsufflation

During the last couple of year's breath-hold diving records have been set using a special respiratory maneuver, called glossopharyngeal insufflation (GI), to add air to the lungs on top of a full inspiration. Popularly, divers often refer to this technique as "lung packing". The purpose of this maneuver is to start the dive with a very large air volume in the lungs so as to reach the deepest possible depth before the chest and lungs are compressed to the limit of what is subjectively tolerable or mechanically safe. In addition, a larger volume of gas in the

lungs adds to the main oxygen store of the body and adds space for CO_2 storage. The technique consists in using the glossopharyngeal muscles to ”pump” air into the lungs. It is akin to glossopharyngeal breathing first described by Dail et al. in the 1950s in patients with poliomyelitic paralysis (16, 19). The reversed method, i.e. drawing air out of the lungs, starting at residual volume, is called glossopharyngeal exsufflation (GE)(54, 58). The latter method is employed by the diver whose lungs by compression are at such a small volume that the expiratory muscles are unable to generate sufficient air-pressure in the lungs and upper air ways by the Valsalva maneuver commonly employed for pressure equilibration of the middle ears and sinuses. In that situation GE allows the diver to draw the small amount of air from the lungs into the mouth and naso-pharynx which is needed for pressure equilibration by the Frenzel maneuver. As an alternative method some divers equalize the middle ear and sinus pressures with air but by allowing seawater to flow into these spaces via the nose. In one subject, able to instill water through a nostril (before diving) magnetic resonance tomography showed water in the middle ear and sinuses (32).

Divers also use GI and GE on dry land to improve the flexibility of the chest and stretchability of the diaphragm (Lindholm, personal communication 2002 and 2005 with divers in paper 52 and 56). Many competitive breath-hold divers have large lung volumes (54, 81, 86), but it is not known whether this is solely a result of the selection of individuals with a genetic advantage or whether training with GI increases the compliance of the ribcage and/or the lungs. Some divers are able to insufflate large volumes and expand the chest significantly, giving them a barrel chest appearance (54). It is possible that they have increased their articular mobility and stretched their respiratory muscles so that the chest volume can increase like what is seen in patients with emphysema. Yet the divers still maintain a normal lung compliance (86). From a study of GI in 16 healthy women (non divers) it was reported that

vital capacity (without GI) had increased by 3% after six weeks (67). GI was performed cautiously with volumes corresponding to 10-25% of VC. Remarkably, after each session of GI, VC was increased, indicating a 'warm-up' effect. This warm-up effect has also been shown by Seccombe et al. (81). It may be ascribed to an increase in static lung compliance (86). Tezlaff et al found that static lung compliance was normal in a group of competitive breath-hold divers, but after performing GI they exhibited a transient increase in lung compliance that lasted for almost 3 minutes (86).

GI has been reported to increase the volume of air which, if not compressed by chest and lung recoil would correspond to as much as 50% of the vital capacity. To exemplify: if a person with a vital capacity of 8L and a residual volume of 2L insufflates the lungs with 4L of air (measured at one ATA) the pressure in the lungs will rise by 10 kPa (75 mm Hg), as reported by Loring et al. (58). This causes the total volume of the gas at TLC (10L) and the volume (4L) drawn in by GI to be compressed to about 12.7L. Thus, the natural total lung capacity of 10L is exceeded by 2.7L (27%). This extra volume is accommodated by depression of the diaphragm and an increase of the chest circumference (66, 67) as well as by the compression of the gas just mentioned (58, 81). The pressure will also reduce the amount of blood in the chest which will free up more space for air (54). The high pressure is, however, not without risk as the pressure will reduce venous return and therefore cardiac preload and consequently diminish cardiac output potentially resulting in syncope (64, 71).

There are also observations suggesting pulmonary barotrauma induced by GI (35) causing transpulmonary pressures (i.e. pressure across the pulmonary pleura) as high as 8kPa (60mmHg) (58). Yet, this maneuver is routinely practiced by many divers with only few reports of major acute complications.

Decompression sickness and nitrogen narcosis

The effects of high nitrogen pressures in deep breath-hold dives are potentially similar to what scuba divers may experience. Theoretical calculations indicate that repeated deep breath-hold dives, separated by short intervals at the surface, would lead to accumulation of enough N_2 to cause decompression sickness (40, 87). Indeed, the repetitive dive pattern of the Korean Amas has been found to generate hypernitrogenemia. (74). Neurological decompression sickness in breath-hold divers has been reported (78), the diagnosis in some cases being confirmed by successful recompression treatment.

Despite the fact that breath-hold diving recently has reached depths at which an air-breathing scuba diver would be completely incapacitated by nitrogen narcosis, reports of this condition in breath-hold divers are all but absent in the literature. It is possible that episodes of narcosis are forgotten by the divers since it is likely to induce amnesia (8). It is also possible that it does not develop even during deep dives because of the short duration of the exposure as well as diminished gas exchange area due to lung compression and edema formation which might slow down the rate of nitrogen uptake in the blood. Moreover, a generally “macho” attitude among divers might make some of them reluctant to confessing to have been narcotized. However, there is one self-report by a world-record-holding diver in whom marked narcosis developed during descent to 160 meters of depth and was strongest on the first half of the ascent (83). The diagnosis of nitrogen narcosis is in this case supported by video footage showing the diver having difficulties with some simple, well rehearsed manipulations of the valve used for inflation of the lift-bag to initiate the ascent.

Fatalities

A common problem in explaining fatal accidents during breath-hold diving is that the official cause of death is frequently just listed as “drowning”(70). Sometimes more enlightening information is available from eyewitnesses having observed the victim hyperventilate intensely before the dive or loosing consciousness during ascent near the surface after a deep and/or long lasting dive. Drowning due to loss of consciousness secondary to hypoxia after excessive hyperventilation still is relatively common among “amateur” breath-hold divers (17, 18) and fatal hypoxia of ascent appears to be particularly frequently among competitive spear fishermen (38). Although, relative to the recent marked increase in the popularity of competitive breath-hold diving, spear fishing may be less widely practiced it accounted for over 30% of the fatalities recorded in the 2004 accident registry of “Divers Alert Network” (20). By contrast, the mortality among experienced competitive breath-hold divers which make the deepest dives has so far remained remarkably low judging from how few are reported in the lay press. Both of the two cases widely publicized in the last several years were due to technical mishaps; one was ascribed to entanglement in a line, the other to a malfunctioning lift bag. Still there are unexplained cases particularly among recreational snorkelers/ breath-hold divers. Some of these cases are undoubtedly of cardiac origin which, when due to arrhythmia, may not be explainable on the basis of autopsy findings and will simply be recorded as ”drowning”. A condition also not diagnosable post mortem is alternobaric vertigo (61, 88) which has been well studied in scuba divers but may be particularly dangerous for breath-hold divers. This condition (a.k.a. Lundgren’s syndrome (23)) consists of rotational vertigo, primarily during ascent and may, in severe cases, cause disorientation and vomiting. It is elicited by asymmetric pressure equilibration between the middle ears. A predisposing factor can be middle-ear barotrauma and asymmetrical swelling of the Eustachian tubes during a day of diving. In some instances scuba divers have been able to cope with even a severe case by stopping the ascent until the vertigo has subsided. By contrast, a breath-hold diver may be in a

much more dangerous situation if he/she is unable to swim in the right direction due to disorientation and floating passively to the surface is impossible because of reduced buoyancy due to lung compression.

Perspectives and future directions

Given the increasing popularity of various forms of breath-hold diving competition, breath-hold divers' quests for greater depths and longer lasting apneas is, without doubt, bringing the participants ever closer to absolute physiological/anatomical limits and life threatening injuries may become more common.. It is not known to what extent genetics and/or training allow some individuals to dive to well over 100 m without clinically apparent ill effects while others suffer pulmonary hemorrhage and signs of pulmonary edema at depths as modest as 20-30m. Some potentially critical factors in the etiology of swimmers'/divers' pulmonary edema such as cold exposure, physical exertion, and predisposing physiological traits (incipient hypertension and excessive sympathetic activation as parts of the diving response) are open to study. Is intense parasympathetic suppression of heart frequency and rhythmicity conducive to dangerous arrhythmia in susceptible individuals? Is CNS tolerance to hypoxia trainable? Are there long-term unfavorable effects of frequent exposures to extreme hypoxia and micro damage to the circulatory system? Some aspects of the reactions to breath-hold diving may extrapolate to clinical conditions unrelated to diving. Are there parallels between the suppression of respiratory drive in breath-hold divers and sufferers of sleep-apnea? Is the mechanism by which a ascending diver re-expands his/her alveoli, which most likely have been atelectatic at depth, applicable to the practice of pulmonary medicine? What is the explanation of the short term increase in lung compliance after GI. And, again, is the extreme stretching of the chest and lungs deleterious in the longer time perspective?

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LEGENDS

Fig 1. Electrocardiogram(ECG),blood pressure (BP, mmHg) by radial catheter and depth (meters) recorded vs. time in two expert divers performing breath-hold dives in water at a cool 25°C in a hyperbaric chamber. The relatively high heart rates and BP were most likely due anticipatory stress. Note that the marked increase in BP after start of the dive preceded the bradycardia. Reproduced by permission (28).

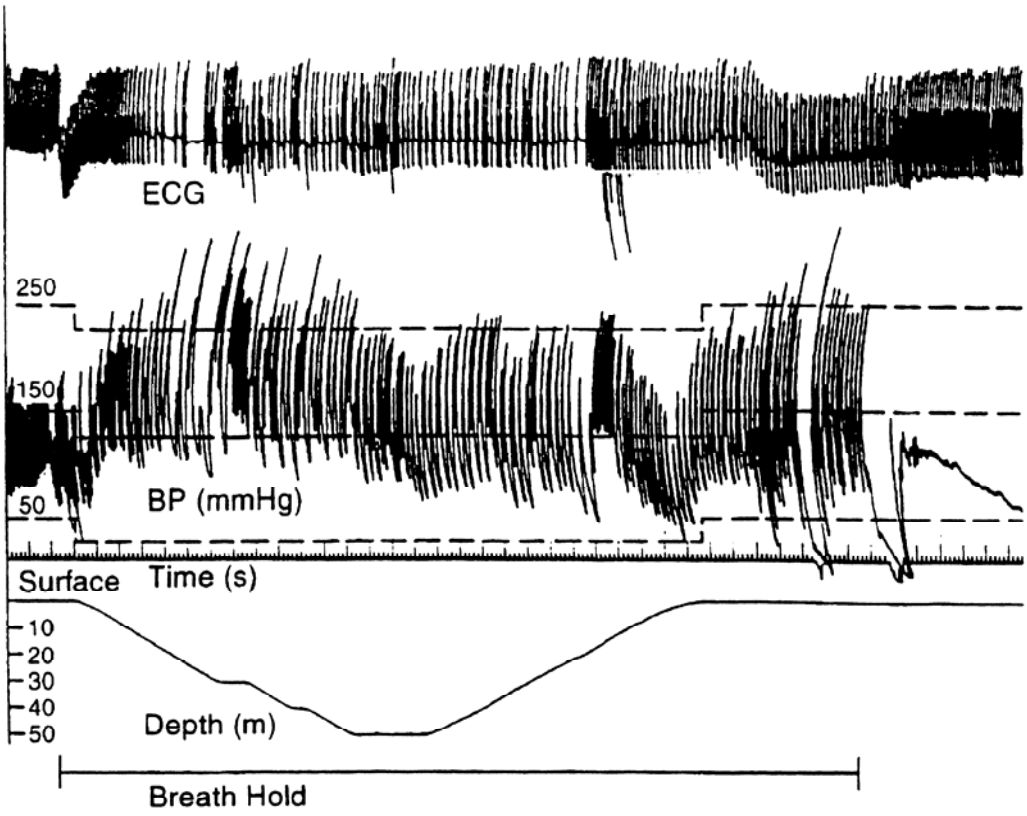
Fig 2. Schematic of the, often mutually opposing, physiologic reactions to submersed breath-holding Full line arrows: at head of arrow; positive influence, i.e. change in one direction at tail of arrow promotes change in the same direction; interrupted line arrow: negative influence, i.e. change in one direction at tail of arrow promotes change in opposite direction at head of arrow. The net effects, known as the diving response, are expressed in the diagram which is framed. The major components of the diving response are: apnea, vasoconstriction in muscles, viscera and skin, blood pressure increase, bradycardia, lowered cardiac output, elevated brain and myocardial blood flow, increased hematocrit, increased anaerobic metabolism, preserved O_2 delivery and CO_2 removal in the brain and heart. These changes enhance breath-hold endurance. Reproduced by permission (29).

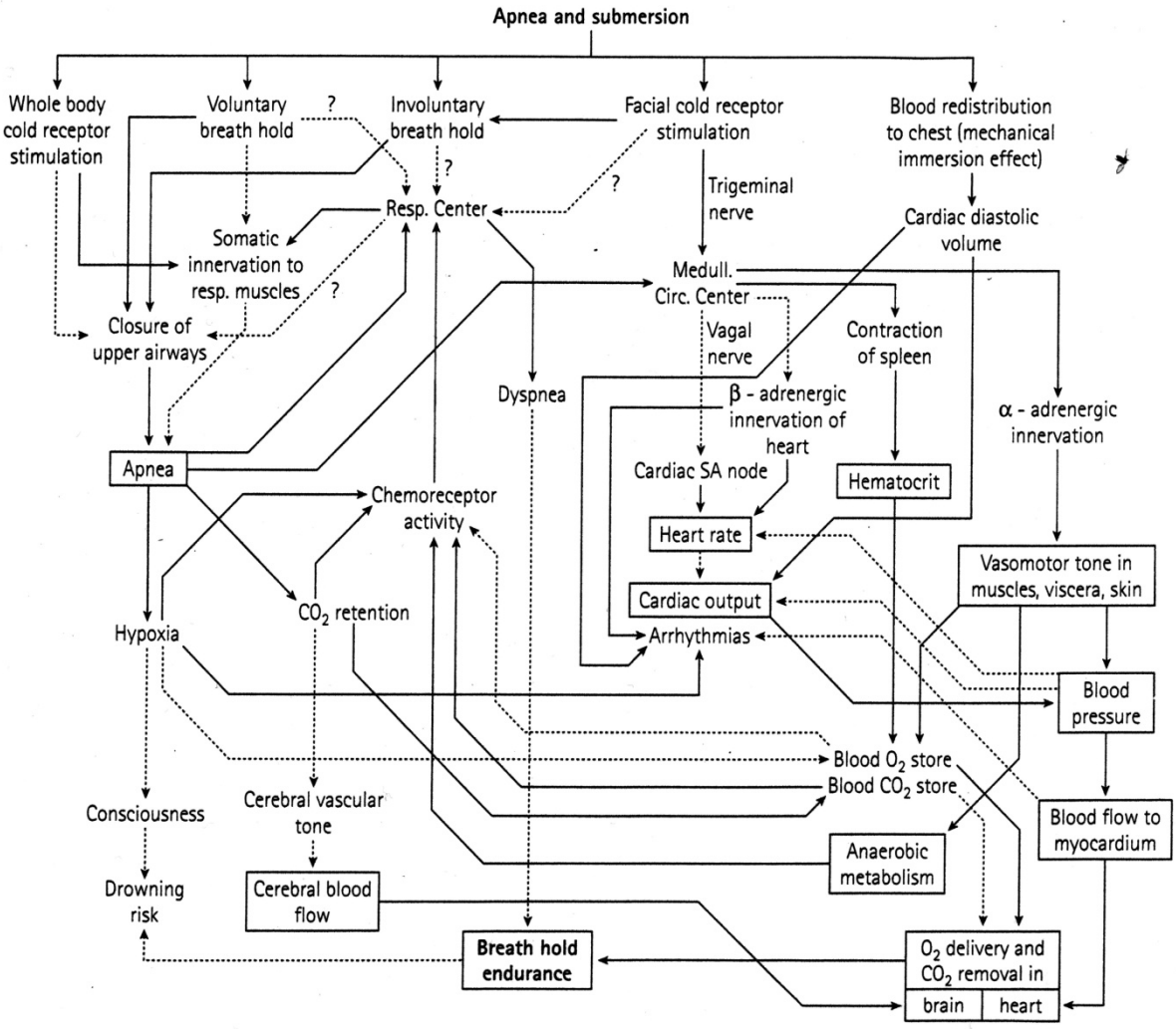
Fig 3.

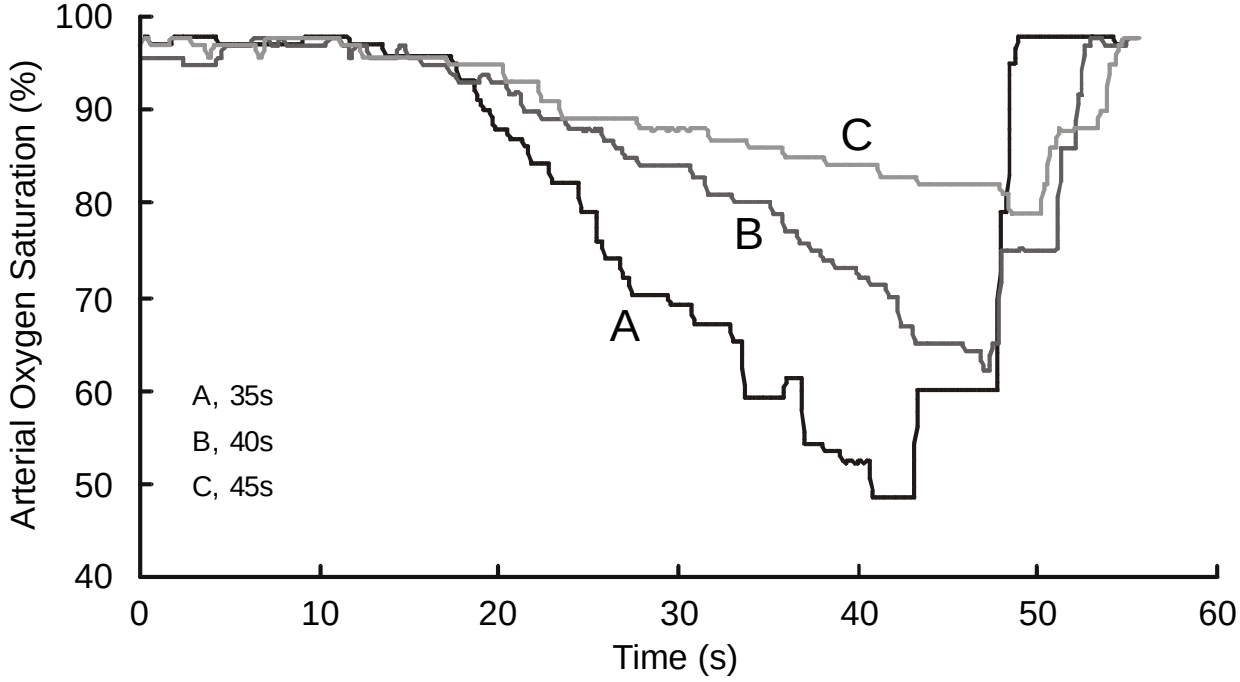
Upper panel: Arterial O_2 saturation as a function of time during combined apnea and leg exercise (120W). Three subjects are shown, who have different rates of arterial desaturation. Apnea starts at time zero and durations are shown to the left of the diagram.

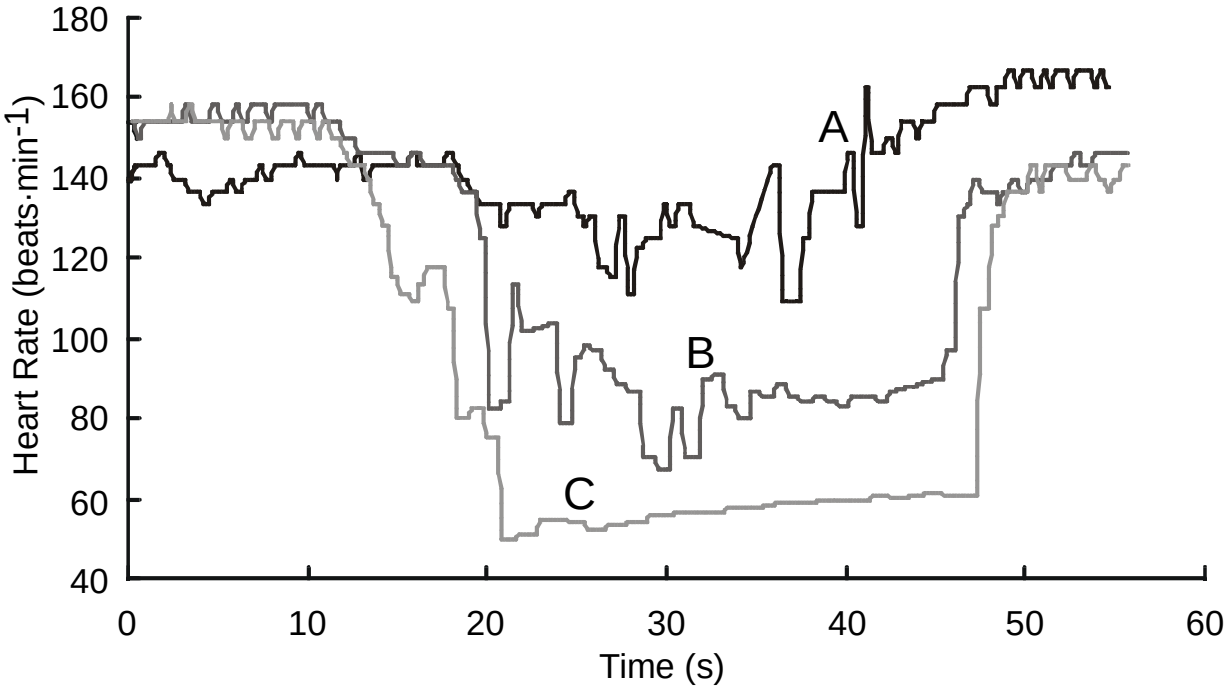
Lower panel: Beat-by-beat heart rate in the same subjects as in the upper panel. The reciprocal relationship between the degree of bradycardia and the rate of desaturation is clearly visible (modified from (47, 51, 55)).

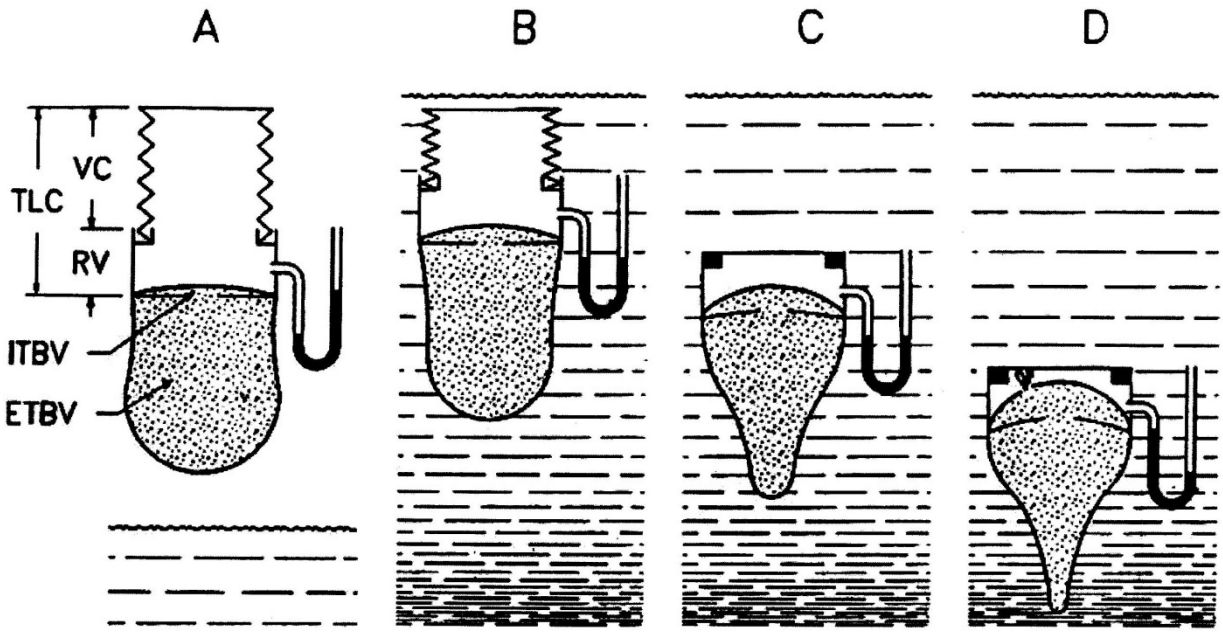
Fig 4. Schematic of how pressure equilibrium between the chest cavity/lung air and water surrounding the body is achieved (Panels A-C) and lost (Panel D). (A) The dive starts by inhalation to total lung capacity (TLC) before submersion; vital capacity (VC) represents the portion of TLC that can be used for pressure equilibration. The manometer indicates overpressure in lung air relative to the ambient atmosphere due to inward recoil of chest and lungs; the residual volume (RV) represents the "non-collapsible" fraction of TLC; the normal intrathoracic blood volume (ITBV) (in the vascular bed and heart) is modest in size; the extrathoracic blood volume is labeled (ETBV). (B) Beginning of descent with pressure equilibrium relative to ambient water maintained, as indicated by manometer, by compression of chest and some translocation of blood from ETBV to ITBV. (C) at greater depth the limits of mechanical compression of chest wall and stretching of diaphragm have been reached but further compression of lung air and maintenance of pressure equilibrium is achieved by redistribution of large volume of blood from ETBV to ITBV. (D) With further descent, the distensibility limit of the blood-containing structures in the chest may be reached, an underpressure develops in the lung relative to the ambient water and therefore to the ITBV with possible extravasation of fluid (pulmonary edema – not shown) and bleeding due to capillary rupture. Reproduced with permission (30) .


ECG
250
150
50
BP (mmHg)
Surface
Time (s)
10
20
30
40
50
Depth (m)
Breath Hold


Apnea and submersion
Whole body cold receptor stimulation
Voluntary breath hold
?
Involuntary breath hold
Facial cold receptor stimulation
Blood redistribution to chest (mechanical immersion effect)
?
?
Trigeminal nerve
Resp. Center
Cardiac diastolic volume
Somatic innervation to resp. muscles
?
Medull. Circ. Center
Closure of upper airways
Vagal nerve
Contraction of spleen
Dyspnea
β - adrenergic innervation of heart
α - adrenergic innervation
Apnea
Cardiac SA node
Hematocrit
Chemoreceptor activity
Heart rate
Vasomotor tone in muscles, viscera, skin
Cardiac output
CO_2 retention
Hypoxia
Arrhythmias
Blood pressure
Blood O_2 store
Blood CO_2 store
Consciousness
Cerebral vascular tone
Blood flow to myocardium
Anaerobic metabolism
Drowning risk
Cerebral blood flow
Breath hold endurance
O_2 delivery and CO_2 removal in
brain
heart


Arterial Oxygen Saturation (%)
100
90
80
70
60
50
40
0
10
20
30
40
50
60
Time (s)
A
B
C
A, 35s
B, 40s
C, 45s


Heart Rate (beats·min-1)
180
160
140
120
100
80
60
40
0
10
20
30
40
50
60
Time (s)
A
B
C


A
B
C
D
TLC
VC
RV
ITBV
ETBV