# Predicting performance in competitive apnoea diving. Part I: Static apnoea

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# Predicting performance in competitive apnoea diving. Part I: static apnoea

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**Key words**
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**Abstract**

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Ever since the first deep diving competitions were organized, there has been debate about when the ultimate limits of human apnoeic performance will be reached, and which factors will determine these limits. Divers have thus far surpassed all former predictions by physiologists in depth and time. The common factor for all competitive apnoea disciplines is apnoeic duration, which can be prolonged by any means that increase total gas storage or tolerance to asphyxia, or reduce metabolic rate. These main factors can be broken down further into several physiological or psychophysiological factors, which are identified in this review. Like in other sports, the main aim in competitive apnoea is to extend human performance beyond the known limits. While a beginner may extend apnoeic duration by getting closer to his or her personal limit, the elite diver can only extend the duration further by pushing the individual physiological limit further by training. In order to achieve this, it is essential to identify the performance predicting factors of apnoea sports and which factors can be affected by training, work that has only just begun. This is the first of two papers reviewing the main factors predicting performance in competitive apnoea diving, which focuses on static apnoea, while the following paper will review dynamic distance and depth disciplines. Great improvements have been made in all diving disciplines in recent years and the 10-minute barrier in resting 'static apnoea' has been broached. Despite this, current training methods and the strategies employed suggest that duration can be prolonged still further, and divers themselves suggest the ultimate limit will be 15 minutes, which appears physiologically possible, for example, with further development of techniques to reduce metabolic rate.

## Introduction

During the past decade the depth, distance and duration of competitive apnoeic diving have increased at an astonishing rate. Records are set at nearly every competition and there is no sign of this development tapering off. At the time of writing, the current male world record for swimming to depth in the 'constant weight' (CWT) category is 120 metres' sea water (msw), for horizontal underwater swimming (DYN) the distance is 250 m, and duration for static underwater immersion (STA) is 10 min 12 s. The female records are not far from these, with 96 msw in CWT, 214 m in DYN and 8 min in STA, which equal the male records set only a few years ago, and appear to be improving at the same rate. Part of the explanation for these improvements is that participation in the sport is increasing and more talents are being discovered. Training methods and diving strategies have also developed immensely during the past decade. The emergence of systematic training in apnoea diving schools and communication among divers worldwide are undoubtedly important aspects in the spreading of effective training methods. But what methods and strategies can be used to further increase performance?

## Different types of diving

There are two fundamentally different types of human diving: sustained, repetitive diving and single dives of maximum performance. Human apnoeic diving has likely existed since the emergence of mankind, and there are evolutionary theories that suggest swimming and diving activities among our earliest ancestors were responsible for some of the most unique human features.[1] The most natural human diving is probably making short, repeated dives within the aerobic dive limit spaced by short intervals. The goal is to spend as much time as possible per hour or day at the working depth to collect enough food. The excellent early work presented by Rahn and Yokoyama is still the best survey of typical human forage diving, which will not be discussed further.[2] Recent reviews have focused on pathology and safety issues concerning breath-hold diving.[3,4] Such issues will be largely excluded from this review, which focuses on predicting competitive apnoeic performance, where safety is provided by the organizers.

In competitive diving, where the aim is to perform one dive of maximal performance, the costs in terms of lactate accumulation and oxygen debt after the maximal dive are not important as the diver has unlimited time to recover, as long as consciousness is not compromised. Competitive diving is done for duration, distance or depth, including sub-disciplines, with or without fins.[4] These disciplines have varying key physiological features that determine individual performance. However, international competitions typically include all or several of these disciplines, and most athletes compete in all disciplines. This makes specialization difficult, requiring training for overall performance, rather than discipline-specific training. The expected influence of different physiological factors on the ultimate limits for duration, distance and depth, requires separate discussion of these disciplines. This first of two papers reviews the factors setting the limits for static apnoea duration, that is, how long can voluntarily be spent without breathing during rest without compromising consciousness? For competitive apnoea this feature is central, as disqualification results should signs of hypoxic loss of muscle control or syncope occur.

**Apnoeic duration – static apnoea**

Sufficient apnoeic duration is a prerequisite for performance in all apnoea sports, expressed in its most pure form in STA, where no variations in work or depth influence the performance (Figure 1). The current STA record of over 10 minutes far exceeds the duration for record dives in the other disciplines, suggesting that the major determinant of duration is the ability to restrict metabolic rate to below typical resting levels.

Three factors determine the limits of apnoeic duration:

- total body gas storage capacity in lungs, blood and tissues
- tolerance to asphyxia
- metabolic rate.

These can be broken down into several further factors (Figure 2).

GAS STORAGE

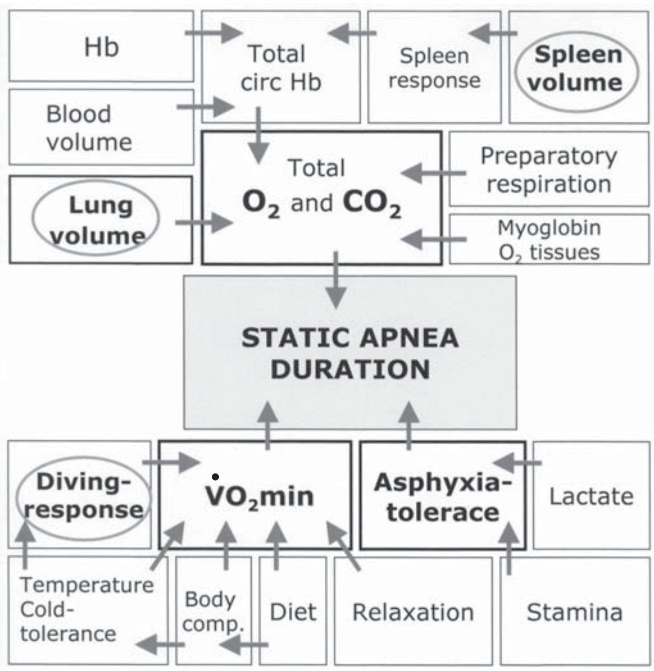
*Lungs*

Large lung volume has repeatedly been described as a factor distinguishing apnoea divers.[5] Benefits from having large lungs are obvious from both an apnoeic duration and depth perspective. With metabolic rate minimized, as in static apnoea, an extra litre of lung air could prolong an apnoea by up to one minute. We recently reported a mean (SD) vital capacity of 7.3 (0.9) L for 14 male elite divers, which was about two litres larger than in a control group matched for age and stature. The individual vital capacity of these divers correlated with their diving performance (total points from three disciplines) in the 2006 apnoea world championships.[6] Several divers had vital capacities of 8–9 L. This leads to the question whether expanded lung volume among divers is due to pre-selection or reflects training-induced changes.

Total lung capacity (TLC) is generally regarded as fixed in adults, but several studies suggest that specific training may increase it. Large lungs in elite divers could, aside from individual predisposition, be due to increased respiratory muscle strength, chest flexibility and or lung compliance, or, possibly training-induced lung growth. The classic study by Carey et al showed that lung volume can actually be increased reversibly by dive training.[7] Other longitudinal studies suggest an enlarging effect on the lungs by swimming and by high altitude exposure, but most likely not by other sports.[8] Stem cells are present in lung tissue, and after removal of lung lobes in children the lungs can regenerate to normal size within two years.[9,10] This suggests that lung growth may be induced in man, at least at an early age. Several elite divers, who have participated in our studies since 2003, state that their lung volume has increased since they started apnoea training (personal communications).

**Figure 1**
**Static apnoea performance – Herbert Nitsch setting a world record of 9:04 in 2006**

**Figure 2**
**The major factors influencing performance in competitive static apnoea (apnoeic duration at rest); some factors are grouped together for reasons explained in the text; circled factors have been shown to correlate with apnoea competition performance**



*Breathing techniques*

A frequently used method to increase lung volume just before diving is 'lung packing' or 'glossopharyngeal breathing' manoeuvres.[11] The breathing method was originally observed in paralyzed patients, and it does not involve normal respiratory muscles.[12] The normal TLC is determined by the maximal contraction of the inspiratory muscles, and the chest and lung recoil. By using the oral cavity and tongue as a pump to repeatedly press down small volumes of additional air into lungs already filled to TLC the diver can increase TLC by up to 4 L.[11,13] Apnoeic duration during rest will likely increase by the same number of minutes. One drawback of this manoeuvre is that the resulting increase in intrathoracic pressure will reduce venous return, resulting in syncope if the diver does not submerge in time.[14] A large inspired lung volume may also to some extent attenuate the development of the oxygen-conserving cardiovascular diving response discussed below.[15] Yet the likely net effect will be that the extra air volume will prolong apnoea, both by providing increased oxygen ($O_2$) storage, and by diluting the carbon dioxide ($CO_2$) received from the blood.

Lung packing can also be used as a training method to increase lung volume, and is often combined with specific stretching manoeuvres (personal communications from divers 2004−2008), but the resulting increase in vital capacity (VC) from a specifically-designed training programme of six weeks in non-divers was only 3%.[16] An effect of autoinflating the lungs is also that the alveolar surface will increase and the respiratory membrane will be thinner. Five minutes after lung packing, an increased TLC compared to before packing has been recorded suggesting a warm-up effect.[17] However, normal lung compliance in apnoea divers was recently reported, suggesting this may not be a major mechanism for long-term increases in TLC.[18]

*Blood*

Another major factor affecting total gas storage is the circulating volume of haemoglobin, which is a product of circulating blood volume and haematocrit or haemoglobin concentration [Hb]. The circulating haemoglobin will not only determine the available blood $O_2$ storage, but also increase the $CO_2$-buffering capacity, which is further enhanced when $S_aO_2$ is low (Haldane effect). Both factors will have a major influence on apnoeic duration.

*Blood volume*

Blood volume in diving mammals is greater than in other mammalian groups, accounting for 10–20% of body weight in seals and sea lions, compared to 7−8% in terrestrial mammals.[19,20] It further seems to correlate with the species' diving ability.[19] It would thus seem adaptive if human divers had greater blood volumes. Human blood volume can be increased through plasma volume expansion via heat adaptation or endurance training [21,22] Gas storage will only increase marginally unless this is accompanied by an increase in erythrocyte volume. In a study of moderately trained apnoea divers, scuba divers and triathletes, only triathletes were found to have higher Hb mass and increased blood volume.[23] Fat is much less vascular than lean tissue, and individual blood volume therefore varies with lean body mass.[24,25] There is no information regarding lean body mass in the study by Prommer et al.[23]

*Haematocrit and total haemoglobin*

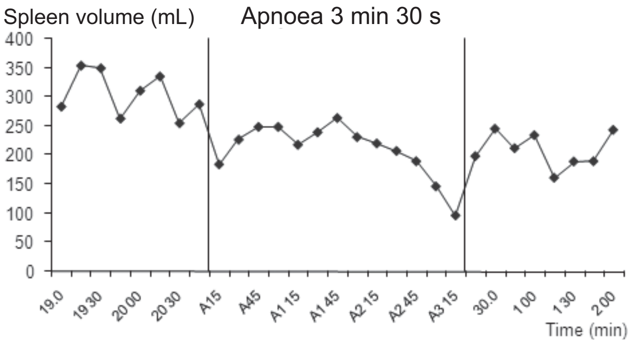
The circulating erythrocyte volume, normally slightly less than half of blood volume, can be increased by two separate mechanisms associated with apnoeic diving and altitude exposure. These two changes develop on different time scales: in the short term, by splenic contraction during apnoea or hypoxic breathing;[26,27] in the longer term, by enhanced erythropoietin production.[28–30]

*Splenic contraction*

The spleen of diving mammals is an extra storage site for erythrocytes when they are not needed for oxygen delivery. This supply can be ejected into the circulation during diving to temporarily enhance gas storage.[31,32] This effect is also present in mammals specialized in endurance running, e.g., horses and dogs, and also in humans during intense exercise.[33,34] The purpose of the storage is likely reduction of blood viscosity between these periods of activity. Splenic contraction in humans during apnoea diving was first observed in Ama divers.[35] The resulting Hb increase is associated with an increased apnoeic duration which is not present in splenectomized subjects.[26]

It was recently observed that the best performances in competitive apnoeic diving were associated with the largest spleens, with volumes of up to 600 ml.[36] The difference in splenic contraction between the smallest versus the largest spleens measured in the elite divers was equivalent to an increase of apnoeic duration by 30 s (unpublished observations). It is still unclear if this represents genetic diversity and pre-selection or training-induced changes, but the observed growth of a small accessory spleen after removal of the main spleen suggests a high regenerative ability.[37] The [Hb] elevation during apnoea is greater in divers than in untrained subjects and endurance athletes suggesting a training-induced promotion of the response.[38] Splenic contraction has been shown to be an active contractile process.[39] It is at least partly induced by hypoxia.[27,40] It was originally suggested to be part of the human diving response, but it now seems that it is not linked to the cardiovascular response, as the two responses are not induced by the same stimuli and occur on separate time scales.[26,41,42] Splenic contraction develops progressively across an apnoea and may need several apnoeas to develop fully (Figure 3).[41]

**Figure 3**
**Progressive development of spleen contraction in a subject performing an apnoea at rest**




*Total haemoglobin*

Higher baseline [Hb] in elite apnoea divers than in endurance skiers and untrained subjects suggests that either apnoea training or pre-selection favours this characteristic.[43] However, there are also reports of similar levels in divers and untrained subjects.[23] These differences could be related to the training status of the tested divers. The amount of erythrocytes in the blood is controlled via erythropoietin (EPO) produced by the hypoxic kidney.[28] While it is accepted that hypobaric hypoxia, i.e., through altitude exposure, enhances EPO production, it has only recently been shown that apnoeic episodes may also elevate EPO.[29,30] When non-divers performed 15 maximal duration apnoeas in sequence with short pauses, a mean increase in serum EPO of 24% was observed, equivalent to increases after six h at 1,800 m.[30] A follow-up study of 10 non-divers showed that 10 maximal effort apnoeas daily for two weeks also increased reticulocyte count, indicative of enhanced erythrocyte production (unpublished observations). However, [Hb] was not increased by this training, possibly due to its short duration or insufficient hypoxic exposure. When 15 apnoeas daily were performed rather than 10 for two weeks by three subjects, an increase in [Hb] of 3% resulted.

With more intense and specific apnoea training, and a diet sufficient in iron, it is likely that the Hb levels of apnoeists may increase further, leading to increased $O_2$ stores and increased $CO_2$ buffering capacity. The apnoea-related training of some elite divers reaches 20 hours per week, which by far exceeds these experimental protocols (personal communication with divers 2003–2008). The [Hb] in sleep apnoea patients appears to be correlated with the severity of the condition.[44] Apnoea diving, especially deep diving, may impose a more potent stimulus than altitude for EPO production, as there is a rapid reduction in tissue oxygen, after an initial phase of hyperoxia; it has been shown that the reduction is, in itself, an important stimulus and this occurs repeatedly during apnoea diving.[45]

*Tissues*

Local $O_2$ stores in the tissues are also important sources for aerobic metabolism. While there is a small amount of $O_2$ in solution in the tissues (2–3% of the total body storage), the main tissue stores are found in muscle myoglobin. In marine mammals, myoglobin concentration may be 10 times greater than in terrestrial species.[46,47] Myoglobin accounts for a considerable part of the total body $O_2$ stores in diving mammals and is correlated to maximum dive duration in toothed whales.[46,48] While potentially important in all apnoeic events, myoglobin content would be most important in dynamic apnoea disciplines (to be discussed in part II). It may seem less likely that myoglobin stores could be recruited for the central circulation during voluntary, conscious apnoea, if non-working areas are 'shut off' by the diving response, but depletion of myoglobin oxygen stores have been observed in sleeping elephant seal pups.[49] In addition to the ability of these hypoxia-adapted animals to withstand increased workload under hypoxic conditions, the enhanced level of myoglobin allows such animals to maintain lower critical $pO_2$ values, i.e., the lowest $O_2$ partial pressure where animals can sustain aerobic metabolism for prolonged periods of time. Thus, myoglobin is important for extending the aerobic dive limit, but the anaerobic capacity also influences diving ability in marine species.[50, 51]

Specific preparatory breathing techniques are used by divers to maximize total $O_2$ storage and to reduce lung, blood and slow-tissue $CO_2$. While $O_2$ is mainly stored in venous blood, $CO_2$ stores are predominantly in the tissues, especially in the muscles (about 10 L).[52] This muscle $CO_2$ storage can be mobilized with a time constant of 30 min, which is much slower than that of other tissue stores.[52] During prolonged yoga-breathing hyperventilation, often used by the divers, these $CO_2$ stores will be at least partially depleted, allowing a breath hold to continue for longer without excess $CO_2$ accumulation. The combination of increasing both the slow and major fast-equilibrating tissue storage sites may prolong apnoeic duration in divers.

## TOLERANCE TO ASPHYXIA

With the presence of this enhanced $O_2$ supply and maximal storage capacity made available for $CO_2$, the next factor determining individual apnoeic duration is the tolerance to asphyxia, that is, the lowest tolerable level of hypoxia primarily for the brain without compromising consciousness, and the highest acceptable level of hypercapnia and acidosis for body functions to remain intact.

*Respiratory drive and the phases of apnoea*

The respiratory drive during apnoea depends on both hypoxia and hypercapnia (and acidosis; asphyxia) and their interactions, i.e., greater hypercapnia is tolerated with higher $O_2$ levels.[53,54] Long-term exposure to hypercapnia will reduce the ventilatory response.[55] A blunted ventilatory response to $CO_2$ has been reported in submarine escape-training instructors, Ama divers and underwater hockey players.[56–58] While $CO_2$ is the dominant factor in inducing breathing in non-divers, the respiratory drive will be more dependent on the development of hypoxia in trained divers with a reduced ventilatory response to hypercapnia. At a given time into apnoea the $P_ACO_2$ is lower and the $P_AO_2$ higher in divers compared to non-divers, while at the end of maximal apnoeas the situation is reversed, showing that divers can tolerate higher levels of hypercapnia and more severe hypoxia.[59]

A dive or an apnoea is, according to the classical definition by Dejours, characterized by two phases, an initial "easy-going phase" without an urge to breathe, followed by a "struggle phase" where mainly the accumulating $CO_2$ will give rise to progressively more powerful involuntary breathing movements.[60] While the length of the first phase is determined mainly by the $P_aCO_2$, the duration of the second phase will also depend on psychological factors such as individual motivation and stamina. Long-term apnoea training has been shown to extend not only total apnoeic duration but also the duration of the "easy going phase".[61] The diving response is also increased after training, which may be involved in delaying the onset of the "struggle phase".[61] The 'breaking point' $P_ACO_2$ appears stable for a given individual at a given time despite different apnoeic conditions but can thus be varied through long-term training.[61,62]

While most inexperienced subjects interrupt the apnoea at the beginning of the struggle phase, elite apnoeists identify three phases of apnoea: the struggle phase will eventually lead to the "fighting phase", where the urge to breathe imposed by the combined stimuli of hypercapnia and hypoxia requires a strong effort not to resume breathing. During this phase the diver does not relax but uses muscle force to sustain the apnoea. The termination of apnoea often depends on learned sensory warning signs e.g., distortion of hearing or tunnel vision (personal communication with elite divers 2008). This enables the diver to interrupt the apnoea just in time to be able to perform the required 'surface protocol'. A developed psychological tolerance to the extreme discomfort during respiratory muscle contractions during the fighting phase is essential for performance in static apnoea. Part of the overall increase in apnoeic duration in divers is due to greater psychological tolerance built up through training.

Hyperventilation in various forms is used by divers to lower pre-apnoeic $P_aCO_2$ and extend the easy-going phase and the time before hypercapnia reaches apnoea-terminating levels.[63] However, this carries an increased risk of syncope as $O_2$ stores are not increased to the same extent. Some trained divers may terminate apnoea due to the hypoxic stimulus alone, while $PCO_2$ is still relatively low due to pre-apnoeic hyperventilation.[64] A strategy used by an increasing number of competitive divers, however, is to start the apnoea without prior hyperventilation or warm-up apnoeas, which is apparently successful and the underlying mechanisms deserve further study. Changes in lung volume and other stimuli derived from lung stretch receptors may also be involved in the integrated respiratory response to apnoeic diving, especially at depth.

*Hypoxia and brain function*

Several studies suggest that the minimum $S_aO_2$ level tolerable by the human brain may be lowered by apnoea training.[64,65] While an $S_aO_2$ of 50% is considered to threaten consciousness in untrained individuals, $S_aO_2$ levels of approximately 30 per cent have been recorded in apparently unaffected divers after maximal apnoeas, both in laboratory and field settings (unpublished observations). The diagram by Rahn and Fenn predicts how the combined effects of hypoxia and hypercapnia determine apnoeic duration,[53] and more recent data from several studies confirm that divers may resume breathing in a different range of $pO_2$ than non-divers.[59] During competition, divers frequently experience hypoxic loss of motor control (LMC) and sometimes syncope, but recover rapidly after assistance. While the short-term risk is obvious, the question arises as to whether such insults cause any long-term damage to brain function.

A study of extended apnoeas (mean 5 min 34 s) in divers without any signs of impaired consciousness revealed that brain damage markers increased by 37% within the first 10 min of recovery.[66] Although small compared with levels known to be associated with brain damage, this suggests there are effects on the brain which could potentially be harmful, considering the repetition of apnoeas in highly trained divers. Studies of neural function in apnoea divers, however, have revealed no long-term effects.[67] Hypoxia has been shown to prevent later ischaemic damage in experimental animal models.[68] This raises a counter-argument that apnoea training could be protective against later hypoxic events. It seems likely that frequent apnoea training involving periods of severe hypoxia may induce up-regulation of protective stress proteins.[69] Other potentially neuroprotective effects such as increased brain blood flow during apnoea have been attributed to hypercapnia and/or the diving response, as there appears to be an independent effect of face immersion.[70]

Thus, the threat of drowning due to syncope aside, there is currently no evidence that repeated syncopal events in voluntary apnoea are harmful to the human brain; it could likely be considered part of normal physiological responses to an imposed stress within the range allowed by human physiology. There is evidence that hypoxia is far less harmful than ischemia.[71] Many competitive divers probably already perform near their individual hypoxic limit, where improvements of results can only be made when training allows this limit to be moved forward. The tolerance to asphyxia was reported to be similar in amateur and expert divers, while inferior in non-divers, suggesting that the factor may already have been exploited among most elite divers.[59]

*Acidosis*

The accumulation of $CO_2$ and lactic acid during prolonged apnoea leads to progressive acidosis.[72] One of the effects of a more powerful diving response with face immersion during apnoeas with exercise was an increased blood lactate accumulation, suggesting an increased anaerobic metabolism during these apnoeas.[73] There is also lactate accumulation during resting apnoeas, although less pronounced.[42] The diving response is known to be more powerful in divers compared to untrained individuals suggesting that divers should accumulate more lactate in underperfused tissues.[74] Interestingly, Joulia and associates obtained lower blood lactate levels in divers compared to non-divers after resting or working apnoeas and also after eupneic exercise, suggesting the presence of specific adaptive mechanisms to reduce lactate accumulation.[75]

Whether such differences were induced by the diving training is not clear, but surprisingly the non-divers had lower lactate accumulation after hand grip exercise with apnoea than without apnoea.[75] This 'lactate paradox of apnoea' deserves further study. One of the essential features for enduring prolonged apnoea may be the blood-buffering capacity for hydrogen ions from accumulating $CO_2$ and lactic acid. The overall $CO_2$ storage in divers was estimated to be twice that of non-divers.[59] The increased total Hb resulting from apnoea training noted above may increase buffering capacity, and splenic contraction will contribute to this effect, and lung-volume expansion will have a diluting effect on $CO_2$.

METABOLIC RATE

The third component setting the limits for apnoeic duration is metabolic rate, determining how well the 'space' between the first two factors is managed. In resting apnoeas, the main factor limiting metabolic rate may be the cardiovascular diving response.[76] When no working muscles compete with the prioritized central circulation, the selective vasoconstriction may be most efficient. Other important factors include reaching a relaxed state before the apnoea, e.g., by meditation, and the influence of thermal factors and dietary status on metabolism.[77,78] While maximum $O_2$ uptake is a crucial factor for performance in aerobic endurance sports, during apnoea the individual minimum $O_2$ uptake ($\dot{V}O_{2\ min}$) is probably one of the most important features determining performance.

*The cardiovascular diving response*

The diving response was first observed in humans by Irving,[79] and has later been shown to have an oxygen-conserving and dive-prolonging effect.[74,80] The response is initiated by apnoea, and transmitted either via decrease in lung stretch receptor activity or by direct contact between respiratory and cardiovascular control centres.[76] A full response is only developed with simultaneous facial chilling, mainly of the forehead and eye region.[81] The main effects are a selective vasoconstriction in areas tolerant to hypoxia, and a vagally-mediated bradycardia which develops during the initial 30 s of apnoea.[76,82] There is a corresponding reduction in cardiac output.[83] In divers, the response may involve a lowering of resting heart rate to half.[74] It does not appear to depend on hypertension as the response often develops during the transient hypotension associated with inspiration at apnoea onset (unpublished observations). It is essentially a hypoxia-prevention system, developing earlier than any signs of asphyxia.

Responsible for the oxygen-conserving effects of this 'central priority system' are the reduced oxygen consumption in underperfused areas, which may temporarily rely on anaerobic metabolism, local $O_2$ stores or possibly hypometabolism, and the heart rate reduction that reduces the $O_2$ demand of the cardiac muscle.[84] This leads to slower depletion of lung oxygen stores.[85] The diving response does not change across series of apnoeas but long-term apnoea training is known to enhance it.[61,86] Some other factors modifying the diving response development are lung volume, with a more pronounced response with low volumes such as at depth (Figure 4).[15] To some extent hypoxia will modify the response with a more pronounced bradycardia at low $O_2$ saturations.[87] As cold-receptors are involved in triggering the response, thermal factors during diving are important for the outcome. Diving bradycardia develops even in warm water when ambient air temperature is higher, i.e., when there is a gradient between air and water (Figure 4). However, the simultaneous chilling of other areas than the face, which normally leads to tachycardia, will not

**Figure 4**
**Heart rate of a diver going to 68 msw in 28°C water, demonstrating that bradycardia occurs in warm water (see text); dive duration approximately 2.5 min**

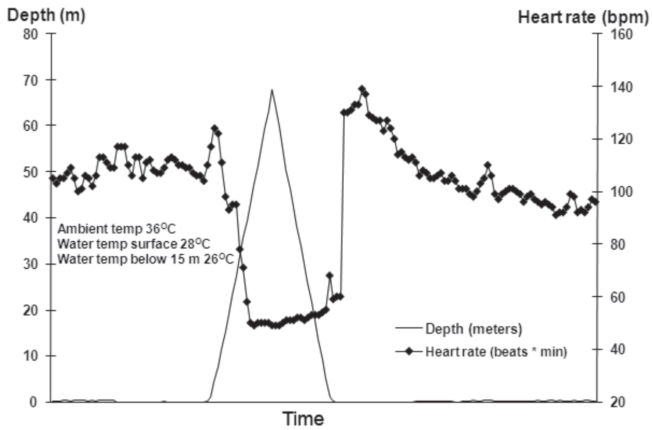
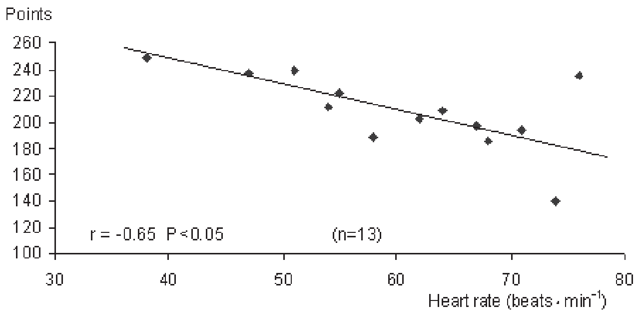


**Figure 5**
**Correlation between apnoeic heart rate during two min apnoeas and total competition points in the world championship in apnoea in 2006**




interfere with the response during apnoea.[88] Experimental two-minute apnoeas during rest in 13 male participants in the world apnoea championship in 2006 revealed an inverse correlation between apnoea heart rate and points in the competition (Figure 5).

*Temperature*

The diving response is dependent on both water and ambient air temperatures.[89] When apnoeas were performed in different ambient air temperatures, a standard water temperature for facial immersion evoked a diving response of varying magnitude, and within a certain temperature range, the change in temperature was the key stimulus.[89] This explains why tropical divers possess a powerful diving response despite diving in relatively warm water: as long as the air is even warmer the response will be initiated (Figure 4). When diving in cold water, the response would be expected to be attenuated in the immersed diver who is already cold before submerging fully. The peripheral vasoconstriction would be expected to be active even before diving, possibly compromising an $O_2$-conserving effect. However, when comparing dry-body simulated apnoeic diving with immersed dives, we recently found that the thermal stimulus during face immersion will generate a diving response powerful enough to conserve $O_2$ in divers even with the body immersed in cool water.[90]

The second effect of temperature on metabolism is the direct cooling effect on the body. While ‘cold-blooded’ animals have a metabolic rate and oxygen consumption correlated to body temperature, mammals generally respond to a threat to body temperature by increased metabolic rate, mainly through shivering thermogenesis. At water temperatures below approximately 33°C, the human body will either chill down or expend energy to keep core temperature constant.[91] The muscle work associated with shivering typically increases metabolism two- to three-fold, more in thin than in fatter individuals as fat provides better insulation due to its low perfusion.[91] This shortens the apnoeic duration and may also lead to an increased superficial blood flow. However, a reduced local temperature without inducing shivering would lower tissue $O_2$ consumption. The responses to environmental temperature are known to vary between different populations and there are also considerable inter-individual differences. Anyone tolerating skin and core temperature reductions without shivering would likely be able to perform longer apnoeas. Regional hypothermia is a strategy contributing to the minimization of energy expenditure in marine mammals.[51]

In the 1960s, Ama divers were described as some of the most cold-adapted humans, but more recent studies reveal that the use of wetsuits has led to deacclimatization.[92] Their $O_2$ consumption during diving without wetsuits was clearly higher in the winter.[93] Non-shivering thermogenesis (NST) may also influence $O_2$ consumption during immersion and this heat-generating system increases after training, e.g., cold-water swimming.[94,95] NST allows less peripheral circulation compared to shivering, which will likely increase the efficiency of the diving response. In winter swimmers during cold water (13°C) immersion, shivering started much later, heart rate was lower and the elevation of metabolic rate was slower.[95] Metabolic rate was increased by only half as much during 60 min immersion in the acclimatized swimmers, and their body temperature was lower compared to unacclimatized subjects.

Thus, better insulated individuals use less energy on shivering and cold-acclimatized individuals allow a lowering of body temperature instead of wasting energy on keeping it constant. Even repeated immersions in one day may produce some of these effects with a lowering of core temperature due to less shivering but with maintained peripheral vasoconstriction.[96] This means there could be a ‘cooling-down’ effect of using warm-up dives, which would likely lead to reduced metabolism and prolonged apnoeic duration. Such short- and long-term cold acclimatisation would be beneficial, and long-term changes could be one explanation as to why Scandinavian divers tend to perform well in championships, despite a shorter training season.

Thus, thermal input influences the cardiovascular responses relevant to apnoeic performance, and may have beneficial effects on metabolism during mild non-shivering cooling but with a negative effect once shivering is induced.

*Anthropometrics and body composition*

The surface area to mass ratio determines energy transfer between the body and the environment, and in cold water this is particularly relevant as the conductive capacity of water is approximately 25 times that of air. This means, that when large and small individuals with similar body shape are immersed, the smallest chill the fastest, and among subjects with the same weight, the one with the smaller surface area will sustain longer immersion before chilling.[97,98] With two individuals of the same weight and shape, the one with a thicker layer of subcutaneous fat will endure longer, and a smaller person will need more subcutaneous fat to be immersed the same time without chilling.[91,97,98] One would therefore expect the ideal diver to be a tall, reasonably muscled person, as this allows for a larger blood volume, with some subcutaneous fat and thereby increased insulative capacity, and female divers to have more subcutaneous fat to compensate for being smaller.

In certain groups of marine mammals, body mass in itself has been found to vary with maximum dive duration among species.[48] The enormous variation in body size in cetaceans make size-related differences in metabolic rate and surface-to-mass ratio important, but with the limited variation in size among humans this effect is probably relatively minor.

*Fasting and diet*

Fasting is a strategy often used by divers in order to enhance apnoeic performance, and fasting during extended periods is observed in marine mammals without any inhibiting effect on physiological performance.[99] During fasting, the body relies mainly on fat metabolism, which requires 8% more $O_2$ per unit energy produced than during purely carbohydrate metabolism. Therefore, if only taking this factor into account, a fasting diver will likely produce shorter apnoeas than a fed one. However, fasting also involves decreased $CO_2$ production (by 30%) which will postpone the respiratory stimulus.[63] In inexperienced divers, this could prolong apnoeas but lead to an increased risk due to a later warning signal to end the apnoea.[78] In experienced divers, the $P_aCO_2$ at a given time is largely determined by pre-apnoeic respiration, and effects of hypoxia may be more important for ending the apnoea. Extended physical work causes the same shift towards increased fat metabolism when glycogen is depleted.[100] However, exercise is carefully avoided by the divers before competitions and fasting is preferred.

There may be several reasons why experienced divers fast during competition. Caloric restriction has been shown to reduce resting metabolic rate by up to 17%.[101,102] Thus fasting may switch the body to a starvation mode restricting energy expenditure and oxygen consumption, irrespective of the substrate, with conceivably positive net effects for apnoeic performance. With a full stomach, the diving response may be compromised by ongoing digestive processes requiring considerable circulatory resources (unpublished observations). Restricted energy supply leading to hypoglycaemia may also delay the onset of shivering thermogenesis (discussed above).[103] While the best results in STA may be produced during fasting, this factor is not likely to enhance performance much further as most divers already exploit this strategy during both training and competitive performance.

In contrast to this, some divers take carbohydrate supplements just before competitions to enhance performance, but the net effect is questionable. As in most sports, a well-composed diet with sufficient iron intake will, in the long run, most likely be relevant to achieving the best result from training but competition results could depend on dietary modifications. Strikingly, while many divers are extremely conscious of what they eat, other top divers disregard this factor altogether.

**Figure 6**
**Diver meditating just before starting a dynamic apnoea**

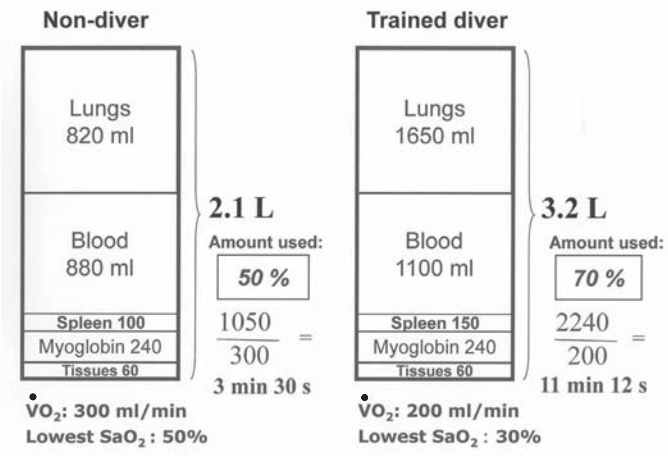
*Relaxation techniques*

In a sport where $\dot{V}O_{2\,min}$ is more important than $\dot{V}O_{2\,max}$, it is obvious that relaxation techniques have a significant impact on results. This is specifically evident in static apnoea performance, but used in all disciplines. Specialized relaxation borrowed from Yoga meditation, with breathing techniques adapted to apnoeic performance, is used by nearly all elite divers before competition (Figure 6). Cyclic yoga relaxation techniques have been reported to lower $O_2$ consumption by 32 per cent.[104] This will likely be developed further and yield longer static apnoeas. Slow yoga breathing may also affect slow-tissue gas stores, and, by lowering their $CO_2$ content, there will be an increased storage space made available for accumulation during apnoea.[52]

**Calculations of duration of static apnoea – where are the limits?**

The total $O_2$ store in a 70 kg person with TLC of 5.5 L, was calculated by Rahn to be 1,996 ml, with 820 ml of the storage in the lungs, 880 ml in the blood, 240 ml in the myoglobin, and 56 ml in physical solution in tissues.[105] With a consumption rate of 300 ml.min$^{-1}$, and if the total amount could be consumed, this would allow for roughly 6 min 40

**Figure 7**
**Calculations of maximal voluntary apnoeic duration in a standardized non-diver and in an elite diver with maximized gas storage and asphyxia tolerance and reduced metabolic rate. Values for divers are based on data measured in individual divers and for non-divers on values adopted from Rahn.[105]**




s of apnoea. Only about half of this amount, however, can be consumed before the normal person loses consciousness, thus a more realistic figure for the allowed voluntary apnoeic duration with these $O_2$ stores is 3 min 20 s. Based on roughly the same figures, but adding the amount of $O_2$ possibly provided by a normal-sized spleen emptying by two thirds during apnoea[33] (100 ml) the total amount would be 2,100 ml and the time with 50% usage about 3 min 30 s (Figure 7). This may well describe the situation in non-divers, but obviously does not fit with the current record dive beyond 10 min or the fact that the majority of world-class competitive free divers can produce static apnoeas in the five to seven minute range.

In a hypothetical diver with a TLC after lung packing of a good 11 L (1,650 ml $O_2$), a blood $O_2$ supply of 1,100 ml due mainly to elevated [Hb] and blood volume, a spleen volume of 600 ml (150 ml $O_2$) and with a similar myoglobin concentration and physical solution in tissue (60 ml), the total stores would instead amount to 3,200 ml (Figure 7). With a low metabolic rate of 200 ml.min$^{-1}$ allowed by specific relaxation techniques and a powerful diving response, and the lowest possible $S_aO_2$ in conscious divers set to 30%, the maximum duration would be 11 min 12 s, one minute beyond the current record (Figure 7).

These anthropometric values represent the upper range of values measured on elite divers in our laboratory (partly unpublished), but we have not identified all these factors in the same individual at any one time. It should be noted that not all factors evident in marine mammals, with the potential to increase $O_2$ storage, e.g., increased muscle myoglobin, have been included in the model. Also not included are the effect of lung packing on lung oxygen concentration and pressure, thus the duration is likely underestimated. Discussing the limits of static apnoea duration with elite free divers revealed that they expect the limit to be extended to around 15 minutes before record setting will level off. It seems likely that the addition of some $O_2$ stores, a further lowering of metabolic rate below resting levels and anaerobic metabolism in vasoconstricted areas could prolong apnoeas even further.

## Conclusions

Gas storage and tolerance to asphyxia can be significantly increased in divers compared to non-divers and metabolism can be reduced by specific meditation techniques. We can explain the current record of 10 min 12 s by including values of gas storage and asphyxia tolerance measured in elite divers. Several of these factors can be enhanced by training and possibly further developed by increased discipline specialization. However myoglobin increases or tissue gas storage modifications have not been included in these calculations. Current knowledge about human apnoeic capacity is incomplete and several potentially contributing factors known from marine mammals have yet to be studied in human divers.

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