Review article

Predicting performance in competitive apnea diving. Part III: depth

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Abstract

(Schagatay E. Predicting performance in competitive apnea diving. Part III: depth. *Diving Hyperb Med*. 2011 December;41(4):216-228.)

Part I described the physiological factors defining the limits of static apnea, while Part II examined performance in dynamic distance swimming. This paper reviews the factors determining performance in the depth disciplines, where hydrostatic pressure is added to the stressors associated with apnea duration and physical work. Apneic duration is essential for performance in all disciplines, and is prolonged by any means that increases gas storage or tolerance to asphyxia, or that reduces metabolic rate. For underwater distance swimming, the main challenge is to restrict metabolism despite the work of swimming, and to redirect blood flow to allow the most vital functions. Here, work economy, local tissue energy and oxygen stores, anaerobic capacity of the muscles, and possibly technical improvements will be essential for further development. In the depth disciplines, direct pressure effects causing barotrauma, the narcotic effects of gases, decompression sickness (DCS) and possibly air embolism during ascent need to be taken into account, as does the risk of hypoxia when the dive cannot be rapidly interrupted before the surface is reached again. While in most deep divers apneic duration is not the main limitation thus far, greater depths may call for exceptionally long apneas and slower ascents to avoid DCS. Narcotic effects may also affect the ultimate depth limit, which elite divers predict to be around 156 metres' sea water. for constant weight with fins. To reach these depths, serious physiological challenges have to be met, technical developments are likely to be needed and safety procedures must be developed concomitantly.

Key words

Breath-hold diving, hypoxia, exercise, cardiovascular, respiratory, physiology, safety, review article

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Introduction

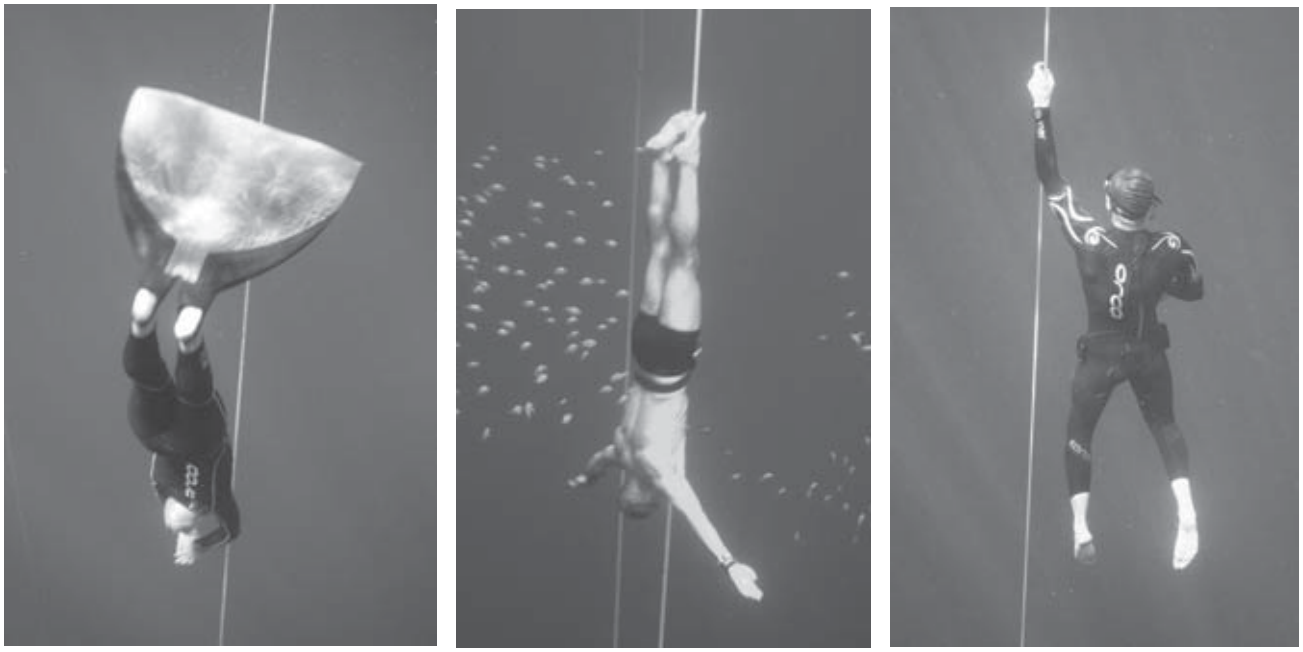
While my two preceding reviews have dealt with the human ability to make long apneas during rest in the competitive discipline of 'static apnea' (STA) and to produce long underwater swims in those of 'dynamic apnea' (DYN and DNF),[1,2] this final review of the pre-requisites for human competitive apnea diving performance will focus on deep diving. The factors associated with apnea duration and physical work are also essential for reaching great depths, but for a detailed review the reader should refer to the previous papers. Here the new factors added with increasing depth will be reviewed.

Several previous papers describing human apneic deep diving have focused on the medical problems and worst-case scenarios that may be associated with such activities.[3–7] However, the fact is that most deep dives by trained athletes are done without any harm to the divers, and the number of divers reaching a depth of over 100 metres' sea water (msw) depth on one breath in unassisted dives is now approaching two dozen, and in assisted dives is even higher. Most physicians and researchers understandably view deep apnea diving largely 'from the outside' and typically describe the threats that the divers are exposed to as events happening somewhat randomly and without the diver's previous awareness or preparedness. After years of study, I believe, on the contrary, that elite divers are well aware of the obstacles they have to overcome to accomplish such performance at an acceptable level of safety, and how to prepare their bodies and minds to sustain such extreme activities. It is correct that there are a series of important limitations to human apneic deep diving, and it can certainly be dangerous if the proper safety measures are not in place. But let us study the diving from the divers' viewpoint to see what can be done to limit risk, to overcome some of the physiological problems and to extend human depth limits. Much of this research is very recent and is only to be found in meeting abstracts and as unpublished observations.

Deep-diving disciplines

The regular competition disciplines of deep diving are 'constant weight with fins' (CWT), 'constant weight without fins' (CNF), in both of which the diver swims down and up without changing their ballast, and 'free immersion' (FIM) in which the diver pulls her/himself down and up along a vertical rope (Figure 1 a–c). There are also two assisted disciplines, which occur only as single events announced by a diver challenging the existing record. These are 'variable weight' (VWT), in which the diver is pulled down by ballast that is left at the bottom and then swims up, and 'no limit' (NLT) where a ballast is used on the way down and lifting bags pull the diver back to the surface. The focus of this

Figure 1
The three unassisted disciplines of deep diving
a. Constant weight with fins (CWT) b. Constant weight without fins (CNF) c. Free immersion (FIM)



review will be on the unassisted competition disciplines, with some examples from the assisted disciplines, mainly because these records can help predict the ultimate limits in unassisted deep diving. Contrary to popular belief, competitions in the three unassisted disciplines have not caused any fatalities; these are, without exception, connected to the assisted disciplines, mainly NLT diving. The male and female record depths in these five disciplines are shown in Table 1. The classic competition discipline is CWT, and we will now follow Annelie Pompe as she completes an 87 msw dive using a monofin, along a rope to which she is attached by a safety line (Figure 1a).

SWIMMING TO 87 msw ON ONE BREATH

"*I have finished three warm-up dives to 14 m on empty lungs and it felt fine. I take calm, deep breaths, exhaling as slowly as possible, and my heart rate falls to a nice low pace. I feel completely relaxed, there are no disturbing thoughts in my mind and I am ready to dive. I take a few last deep breaths, exhale even more slowly and completely, then make a last full inspiration. I lift my upper body over the surface and top my lungs by packing ten packs, and go.*

The first swim stroke is powerful to overcome the buoyancy and I descend rapidly, feeling the pressure rise, and equalize my ears repeatedly using Frenzel – with the back of my tongue, air pressing against the tight nose clip. I try to relax all muscles, except the legs during each kick with the monofin, and in the pauses between kicks also my legs. I equalize based on how my ears feel, using reversed packing to fill my mouth before each Frenzel. The pauses between kicks become longer and longer, I pass what I think is 50 msw, swimming very slowly, and then start free-falling.

I fall head down through the water in my favorite part of the dive in a relaxed but streamlined position. My heart rate falls even further. Feeling the water passing at a more and more rapid rate, I close my eyes, knowing my safety line will keep me close to the rope. The pressure on my chest is increasing and I decide it is time to stop the reversed packing, so I make one last big 'mouth-fill' and then keep my epiglottis closed. This air will be used for the last equalization; blood shift will protect my lungs, but I have to protect my ears. It becomes darker, and I feel the first respiratory contraction, but don't let it disturb my relaxation as I fall toward the bottom plate at 87 m. It becomes even darker and I know I'm almost there. My arms and legs are tingling, everything feels smaller in my head and, being somewhat 'narked', I have to focus to stay alert.

Table 1
AIDA world records in the five depth disciplines as of October 2011 (depths in [ ] awaiting confirmation)

Discipline	Depth (msw)	
	Men	Women
Constant weight without fins	101	62
Constant weight with fins	124	[101]
Free immersion	121	[88]
Variable weight	142	126
No limit	214	160

I touch the bottom plate with my fingers, grab the rope and let my body fall down, and look around. I wish I could stay here longer, but know how far it is back to the surface. I make a single pull upwards on the line and make a first powerful stroke with my legs, now holding my arms close to my chest in order not to stretch the lungs too much. Up, up… I close my eyes again and tell myself my legs are strong and it is not so far left. I feel lactate building up in my legs, but can see it is getting lighter. I feel strong contractions but have to think positive and stay relaxed. I start swimming more slowly as my buoyancy increases, and there is my safety diver at 20 m. When floating the last few metres, I go through in my mind what to do when surfacing. A metre under the surface I exhale half of my lung volume, and as soon as I surface I take a breath. I make one – two – three fast breaths with some resistance on exhale (hook breathing), then the surface protocol: Mask off, OK sign and say "I am OK". I'm more than OK – I'm happy."

Characteristics of the deep-diving disciplines

When studying the 87 msw CWT dive described above, it becomes evident that there are great differences between diving to depth and the other disciplines. The three major differences are:

- the inability to interrupt the dive when at depth;
- the periodic work in depth disciplines;
- the pressure effects.

APNEA DURATION, ASCENT HYPOXIA AND SAFETY

Unlike in the pool disciplines, which are performed at or near the surface, in deep diving the diver cannot resume breathing until reaching the surface again, and must thus estimate the maximal effort at 'mid time' of the performance. In all disciplines, however, the attempted depth has to be pre-announced, and the diver will either reach this depth or turn early and incur penalty points. As with the pool disciplines, divers have to correctly perform the 'surface protocol' to show they are in full control at the end of the dive.[2] Should there be signs of hypoxia the diver is disqualified. Thus, divers cannot propose a dive that is more than they are confident of achieving without risking disqualification. As with the pool disciplines, there are safety divers, who closely follow the competitor during the last 20–30 m of the ascent, depending on depth, where there is a risk of ascent syncope.

Ascent syncope, often somewhat incorrectly termed 'shallow-water blackout',* is a potential risk associated with deep diving and a direct effect of the change in ambient and thereby gas partial pressures. When the diver descends, the air in the lungs is compressed in direct proportion to the ambient (hydrostatic) pressure (Boyle's law) and, as the partial pressure of oxygen (PO_2) in the lungs rises with the total gas pressure (Dalton's law), more O_2 can be transferred from the lungs to the blood (Henry's law). Thus, during the initial half of the dive, there is actually more O_2 available than during a breath hold at the surface. However, during the ascent, ambient (hydrostatic) pressure falls and, as part of the O_2 has now been used, PO_2 will be much lower than before. In fact, it may reach levels lower than the corresponding oxygen pressure in the blood, leading to the reverse transfer of O_2 from the blood back to the lungs.[8] This will rapidly cause a fall in brain oxygenation and potentially a loss of consciousness near the surface. This is because of the non-linearity of the O_2-haemoglobin dissociation curve, where at this point in the dive, a small decrease in PO_2 lead to a large fall in O_2 content of the blood. Thus, as this risk is directly associated with the fall in pressure near the surface, so is the risk for the diver. No such risk of hypoxic loss of consciousness is present at depth where oxygen pressure is higher than normal, as long as the diver is not delayed or prevented from surfacing. This is why it is sufficient to have safety divers present for the last part of the dive in order to prevent an accident should syncope occur during ascent.

The safety divers waiting at depth are also apneic divers, as the transportation to the surface must be very rapid, so that the diver can resume breathing. Divers breathing an air or other gas supply cannot rise to the surface at a sufficiently rapid rate because of a slower swimming speed, and risk decompression sickness (DCS) or arterial gas embolism (AGE) if they try. Using safety free-divers, an hypoxic syncope, if it occurs, happens within a controlled situation in which the athlete begins to breathe spontaneously within seconds of surfacing, or after a 'blow-tap-talk' procedure.[2] It seems that laryngospasm, i.e., automatic closure of the airways, prevents water from entering the lungs as long as the diver is submerged.[9] Although its function in drowning has been debated in the medical literature, it seems clear from all the events in competitive apnea, where unconscious divers brought to the surface start breathing spontaneously without signs of water aspiration, that laryngospasm is involved in protecting the airways in apnea divers (Schagatay E, personal observations and communications with divers).

Should the competitor have problems during earlier parts of the dive, other safety arrangements are present, the major one being a counter-ballast system. The diver is connected with a safety line to the vertical rope, and the rope can be pulled up with the diver attached should the diver not return to the surface at the announced time. Before diving, the diver states the expected dive duration, and the time keeper will feel the pull on the rope when the diver turns and heads for the surface. In major competitions, the diver is often monitored via sonar, and technical divers are standing by on the surface

Editor's footnote: The term '*shallow-water blackout'* is understood to have been coined during World War II by the Royal Navy to describe loss of consciousness (LoC) underwater in attack frogmen breathing oxygen from a closed circuit breathing apparatus. LoC in apnea divers should be termed either '*hypoxic syncope'* or '*ascent syncope'*.

ready to dive should there be any other problems. This has, however, never been necessary in AIDA competitions. Risk of hypoxic syncope is also evident directly after surfacing, due to the circulation time between lungs and brain, and the diver is, therefore, closely observed for 30 s. Divers often use 'hook breathing' to '*re-establish lung function*', likely counteracting hypoxia and pulmonary oedema.[2]

WORK ECONOMY AND THE FOUR PHASES OF A DEEP DIVE

Energy-efficient locomotion is essential for deep dives. As in the dive described above, muscles of the legs and lower abdomen are initially used for propulsion, but the diver will free-fall instead of swimming once negative buoyancy exceeds drag. The level of exertion involved is, therefore, not the same as in the pool disciplines of 'dynamic apnea', although the same muscles are used in these distance disciplines. In dynamic apnea, the work is quite constant, except during the turns against the pool wall. Also, in the most demanding depth discipline from an energetic standpoint, CNF, where the entire body works for propulsion using breaststroke, the total work required is less than in 'dynamic apnea without fins', because, after an initial phase of swimming, the diver reaches a phase of passive, free-falling descent. Also in FIM, involving only upper-body work, the diver free-falls instead of pulling the rope during part of the distance. Thus, despite the differences between the depth disciplines concerning levels of exertion and the muscles used for propulsion, all share a period of free-fall to minimise energy expenditure.

Therefore, diving to depth can be defined in four phases:

- an initial phase of *positive descent* when the diver works to overcome positive buoyancy;
- a second *free-fall* phase when the diver is sufficiently negatively buoyant;
- after the turn, a third phase of *negative ascent* with work to overcome negative buoyancy;
- a final *positive ascent* phase above the point of neutral buoyancy, when the diver may passively rise to the surface.

The same phases have been identified in diving seals and whales, whereby the deep-diving Weddell seal was found to reduce diving energy costs by up to 60%.[10]

The transition points between these phases vary between divers both in duration and depth depending on the individual diver's tissue density (fat/lean body mass ratio), lung volume, dive suit and weighting used. While body tissues will displace the same volume of water and have the same effect on buoyancy at depth, a high ratio of compressible air spaces in the body and dive suit to tissues will lead to a more rapid switch to negative buoyancy during descent (a lean subject with large lungs will thus fall faster compared to someone with low tissue density and small lungs. The first diver can stop swimming and rapidly gain speed in free-fall, while for the latter it may still pay to swim but with a lower stroke frequency. This may be yet another reason large lung volume is beneficial for human diving performance in addition to enhanced oxygen and carbon dioxide storage and increasing surface lung capacity to residual volume (SV/RV) ratio allowing greater compression (see below).[1,2,11]

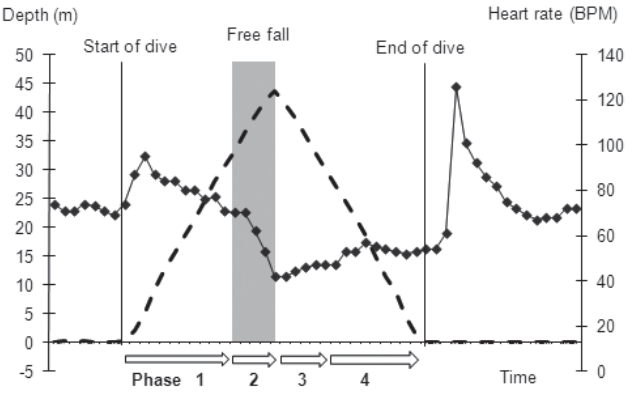
Anthropometric differences may be compensated for by the choice of suit and weighting, as some divers prefer to start to free-fall at an earlier point at the cost of having to work more on the way up, others preferring a deeper point of neutral buoyancy requiring greater initial effort. The passive part of the positive ascent phase may vary greatly in length, which may also reflect individual apneic ability and basal metabolic rate; some divers choose to continue swimming or pulling until surfacing. These transitions between working and resting phases during progressive hypoxia may in turn have effects on cardiovascular regulation.

THE DIVING RESPONSE AT DEPTH

The diving response is a priority system of circulatory responses to apnea that redistributes blood within the body, with a focus on maintaining the oxygen delivery in support of the most vital functions of heart, brain and working muscle.[9,12] The response involves vasoconstriction in organs tolerant of hypoxia and bradycardia, which conserves oxygen both during resting and working dives.[12–16] Its functions have been discussed in detail in the previous reviews.[1,2]

In the depth disciplines, the self-propelled diver must supply working muscle with sufficient energy from some source for the two working phases of the dive. The diver will initially be exposed to an increasing PO_2 caused by the air compression during descent. Thus, in phase 1 of the dive there will be sufficient oxygen and circulation will likely be kept in the working muscles as well as in heart and brain.[12] In phase 2, when the diver stops swimming, muscle circulation no longer needs to be prioritised and during free-fall, the diving

Figure 2
Heart rate (continuous line) during a training CWT dive by Annelie Pompe to 44 msw lasting 2 min; when free-falling starts, HR falls further



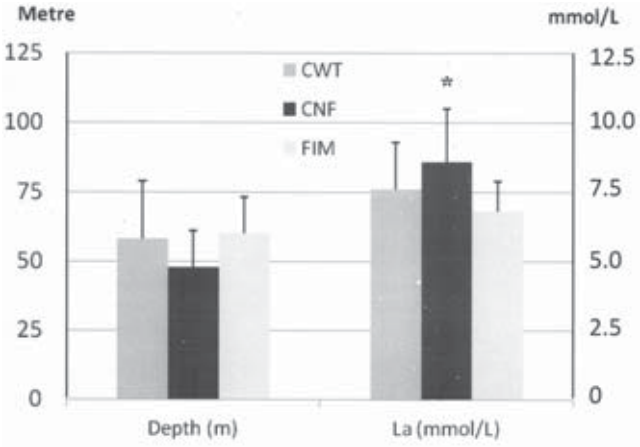
response can develop fully and conserve oxygen for heart and brain. In many divers, this transition between phases can be seen as an additional fall in heart rate (Figure 2). In itself, lung compression at depth may also enhance the diving response as will the colder water at depth, and by the end of the dive the developing asphyxia may contribute.[17–19] These factors all seem to act together to keep O_2 conservation at its maximum during deep diving.

During ascent in phase 3, despite the heavy work needed to overcome the negative buoyancy directly after the turn, heart rate remains low (Figure 2). Despite the urgent need of muscles for O_2, it appears blood flow is not shared between brain and working muscles once it has been redirected. Seals are known to rely on their unusually high levels of muscle myoglobin when this occurs,[20,21] but it is unknown if this factor is important in man. When there is conflict of interest, the brain always takes priority, and oxygen has to be conserved to sustain consciousness during the last part of the dive. Thus, the working muscles may have to rely completely on stored oxygen in the myoglobin and on anaerobic energy pathways, evident as the muscle soreness noted by Annelie in her description of her 87 msw dive, and classically interpreted as lactate accumulation, although its causes are complex and not fully understood.[20–23] In phase 4, the diver will often passively float to the surface and vasoconstriction can prevail.

In all animals relying on anaerobic glycolysis for energy production, the end products lactate and protons may cause metabolic acidosis.[24] It was observed in seals that, during a dive, lactate is sequestered and then released into the general circulation after breathing has been resumed, with a massive surge in the circulating blood on surfacing.[25] In exercise during breathing, where temporary and local limitations in blood flow cause lactate production, other regions may oxidize lactate, with only the net between production and use being detectable in the circulating blood.[26,27] However, the lactate produced during diving probably cannot be metabolised during the apnea, in part because of the massive peripheral vasoconstriction limiting transportation between tissues and in part to the over-all hypoxia; thus there may be no areas with sufficient oxygenation to oxidize lactate.

Therefore, high peaks of lactate can be seen directly after apneic dives, despite a fairly limited level of exertion.[2] To determine these levels and any differences between the depth disciplines, we measured capillary lactate 2–4 min after CWT, CNF and FIM dives during an international depth competition.[28] It seems that CNF, where the whole body is at work, represents the greatest hypoxic stress (Figure 3). However, compared to levels of around 10 mmol L^{-1} found in elite divers after dynamic disciplines,[2] the values after deep dives were lower. Even during static apnea at rest, there will be lactate accumulation, with levels of 5 mmol L^{-1} measured.[2] Reference values before dives were in the range 1–2.5 mmol L^{-1}, while normal resting levels are around 1 mmol L^{-1}. In

Figure 3
Mean (SD) depth and blood lactate concentration after CWT (n = 13), CNF (n = 10) and FIM (n = 10) competition dives
*** indicates $P < 0.05$ between CNF and FIM**



diving mammals, high baseline haemoglobin (Hb) as well as splenic contraction during diving will help buffer the blood and so will the human elevation of circulating Hb via splenic contraction.[29–31] In turtles, lactate can be stored in the shell and skeleton via the formation of calcium lactate, explaining how these air-breathing animals can survive anoxic periods of up to months.[32] Even in humans, asphyxia in critically ill patients has been found to be correlated with hypocalcaemia, suggesting a possible role of this mechanism in extreme conditions.[33]

REACHING MAXIMAL DEPTH – THE EFFECTS OF PRESSURE

While apneic duration and the ability to work efficiently with limited oxygen supplies can be limiting, in deep diving the ultimate challenges may be associated with the effects of increased hydrostatic pressure. The pressure will have direct effects on the air-filled spaces of the body; the increased hydrostatic pressure will cause these to be compressed with a risk of rupture if no equalization takes place, and there will be several effects of pressure changes on gas exchange between air and tissues, one being the risk of hypoxic syncope described above.

Training, preparation and equalization to avoid barotrauma

During increases in ambient pressure, the internal air spaces of the human body will shrink and eventually the negative pressure created may cause damage to surrounding tissues. During ascent, any air trapped in closed spaces will increase in volume and may also cause damage, although the latter case is more typical of compressed gas diving. Deep apnea dives must be preceded by training, and efficient equalization techniques must be employed to avoid barotrauma.

Figure 4
Lotta Ericson, professional free-diving instructor, performing 'reversed packing' and stretching, one of the many training exercises used for deep dive

The depth which can be reached without risk of barotrauma is set by the relation between the inspired lung volume at the surface (SV), and the diver's residual volume (RV). Classically, this surface volume was assumed to be the TLC reached by maximal inspiration.[34] However in elite divers, this is not the case. Divers use 'lung packing' to fill the lungs beyond total lung capacity (TLC) by using the oral cavity as a pump, which means SV can exceed TLC by several litres.[35–37] In addition, by employing long-term training methods involving lung packing and chest stretching, divers state they have increased their TLC by 2 L or more over time. Such training could partially explain why mean vital capacity was 7.3 L in divers, 1.8 L greater than that of a matched control group.[11]

A method called 'reversed packing' in which, after full expiration, the diver will suck air from the lungs can be employed to reduce the RV below normal levels by stretching the diaphragm and chest inwards (Figure 4).[38] Combined with other chest-stretching methods, these training regimes may enable divers to extend their 'lung-safe' depth considerably. Long-term lung training may include dives to moderate depths with empty lungs in order to mimic greater depths. For instance, pool dives to 5 m depth at RV are estimated to mimic dives with full lungs to approximately 90 msw.[39] However, this training may in some cases lead to capillary rupture and pulmonary oedema.[39]

As an example of the effects of such training, we can use a model diver with an original TLC of 4 L and a RV of 1 L. Using Boyle's law, we can calculate that this diver, after a normal TLC inspiration, will reach RV at a pressure of 405 kPa (30 msw). If instead the diver has a TLC of 5 L and an ability to pack an extra litre of air, the 'lung-safe' depth would be 50 msw (608 kPa). However, if the diver also manages to reduce RV by training to 0.5 L, this depth limit will now be 110 msw (1.21 MPa).

'Blood shift'

Another potential lung-protecting mechanism is the central pooling of blood in the thorax, called 'blood shift' which takes place during immersion, and increases progressively as the diver descends.[34,40,41] This redistribution of blood from the periphery to the pulmonary vessels compensates for the diminishing air volume and counteracts the risk of vessel rupture until vessel volume is maximally extended. Training with negative lung volumes may, in fact, increase the possible volume expansion of these vessels as suggested by the notion by divers that haemoptysis occurred only when they had not trained sufficiently (various divers, personal communications, 2010). Thus, specific long-term training methods, combined with warm-up dives, lung packing before descent, and the effects of blood shift may help explain why certain individuals can reach beyond 100 msw, and in extreme cases beyond 200 msw depth, without any detectable damage to the lungs or airways.

Preparation

Just as Annelie before her 87 msw dive, many, but not all, deep divers will use warm-up dives simulating greater depths by diving on either functional or residual lung volume. This may stretch the lung tissue, facilitate blood shift, and protect the lungs from sudden extreme negative pressure at maximal depth. As before performing the pool disciplines, breathe-up techniques may be used before deep dives to lower carbon dioxide storage in fast and slow tissues, and for the diver to reach complete relaxation.[1,2]

Upper airway equalization techniques

To many inexperienced divers, just being able to free-dive to 20 msw seems to present a series of challenges, as the pressure in the middle ear and sinuses generally has to be actively equalized. The most commonly used method is a Valsalva maneouvre, whereby attempted exhalation against a closed nose and mouth elevates airway pressure and forces air via the Eustachian tube into the middle ears. While this method can be used down to the diver's RV depth, after which a negative lung pressure makes it impossible, most trained divers instead use the Frenzel maneouvre. In Frenzel, the back of the tongue is used to elevate pressure in the oral cavity, pushing up against a closed nose. By using this technique, the lungs can be kept separated from the upper airways and pressure differences be allowed to develop.

When the diver descends beyond RV depth, air can still be drawn up into the oro-nasal cavity via reversed packing, the glottis closed, and the air used for equalization of ears and sinuses. However, the deep diver will eventually reach a

level beyond which it is extremely difficult to draw up more air from their lungs and at that point the diver takes the last '*mouth-fill*', after which the glottis must be tightly closed. All the muscles involved, most unfamiliar to the non-diver, can be actively controlled by the trained diver. This last '*mouth-fill'* must enable the diver to equalize all the way to maximal depth, or their eardrums may rupture. A last resort for the deepest divers is to use 'wet equalization', whereby the oro-nasal cavity (including the sinuses) and middle ear are flooded with water thus obviating the need to equalize the ears and sinuses and thereby avoiding aural and sinus barotrauma. This method appears to be used by some divers in extreme dives, but it is unclear if and by what methods the water is removed subsequently or whether there are other problems, such as vertigo when cold water enters the middle ear or infection related to the technique.

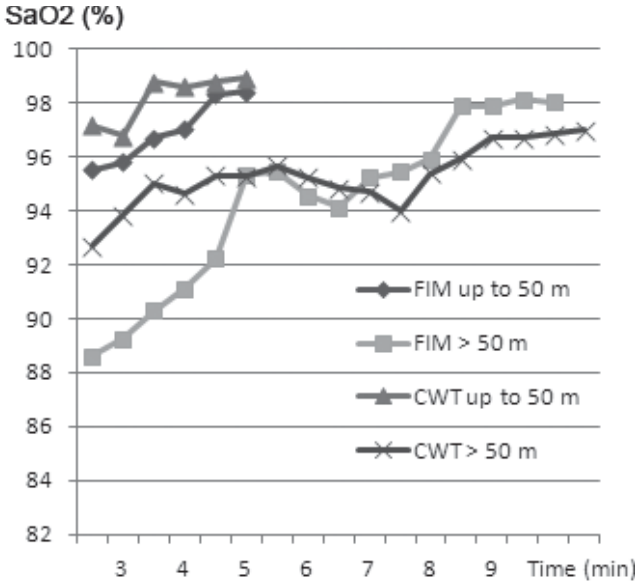
As the diving mask also has to be equalized to avoid eye and soft tissue squeeze, deep divers either use flexible masks with extremely low volumes or goggles filled with water, or avoid a mask entirely. Water-filled goggles offer some protection against abrasion and cold at the cost of good vision, but specialized goggles with lenses compensating for the loss of refractive power in water can also be used.

Despite long- and short-term preparation and excellent equalization skills, squeeze is the single most common problem associated with deep apnea diving. If not limited by other factors, each individual diver will eventually reach a depth where there is competition between ears and lungs for the last volume of air, beyond which damage may occur.

It has been shown that pulmonary oedema, detected as a delay of recovery of arterial oxygen saturation (S_aO_2) is quite common in deeper dives.[42] When we compared the recovery of S_aO_2 after deep CWT and FIM dives using pulse oximetry, we found evidence that not only depth but also the specific discipline may influence lung function. It was clear that, in dives beyond 50 msw, desaturation was greater and recovery slower after FIM dives, possibly because it involves upper body work (Figure 5).[43] This could explain why divers say they have to be careful not to move the chest excessively, as this may cause increased stress in the compressed lung. In the same study, involving a total of 56 dives, S_aO_2 recovery to 97% was found to be delayed beyond 10 min in nine cases (five CWT and four FIM) and cough with haemoptysis was found in three cases. Of these nine cases, eight had been to > 50 msw depth.[43]

This suggests that for divers to reach greater depths, their training and techniques for equalization need further development. However, the record depths reached in the no-limits category show that the lungs of some individuals may tolerate the immense effect of hydrostatic pressure of 2.3 MPa (214 msw), suggesting that the ultimate limiting factor might not be barotrauma. In addition to hypoxia and squeeze, there are other serious physiological challenges

Figure 5
CWT and FIM dives; recovery was the same for dives to ≤ 50 msw, but desaturation was more pronounced and recovery delayed after FIM dives to >50 msw ($P < 0.01$); mean depth for CWT was 53 msw and for FIM 52 msw (NS)



relating to depth, including nitrogen narcosis and DCS that have to be met.

Nitrogen narcosis

While efficient equalization techniques and training extend the tolerance to the effects of mechanical pressure and reduce the risk of barotrauma, the direct effects of hydrostatic pressure on gases cannot easily be avoided. The narcotic effect of nitrogen is fast enough to be evident in current apneic divers, whilst CNS oxygen toxicity may occur given enough partial pressure and time.[44] These effects may limit deep apneic air diving. Oxygen may also give rise to toxic damage to lung tissue, but this is extremely unlikely during the limited exposure time in apneic diving.

Nitrogen narcosis is evident from the anecdotes of many breath-hold divers. Describing her 'no limit' dive to 160 msw, Tanya Streeter reported an inability to perform well-rehearsed maneouvres at depth.[45] Other diving-related problems, including DCS, AGE, alternobaric vertigo, hypoxia and cold exposure could give rise to similar symptoms, but typical of the symptoms associated with narcosis is that they appear regularly and often at a certain depth, and disappear before or with dive termination, as seen by the following accounts (various divers, personal communications, 2009, 2010).

Diver account 1: One diver with a personal best beyond 90 msw, said that all dives beyond 75 msw (about 10 dives) involved some component of narcosis. This could be a lack

of focus as well as a sensation of numbness in the mouth and tingling in the fingers and toes. In some of the dives when he was inexperienced, this resulted in confusion and panic. The confusion was often related to hallucinations: an example was on the ascent from an 85 msw dive, when he believed that he passed the same reef over and over again, and felt he was not moving and was sure he was not going to reach the surface. Suddenly he surfaced without understanding how he had got there. He was now beginning to control these sensations at depth by focusing on something specific prior to the first signs of narcosis beginning.

Diver account 2: A diver with numerous dives beyond 100 msw and best performance beyond 120 msw, stated that narcosis is evident in all her dives beyond 50 msw. The way for her to proceed had been to learn to not give in to it but to cope with it, and focus on the tasks at hand. She has now found ways to perform well despite these problems.

Diver account 3: On his first FIM dive to 82 msw, the diver felt well at 75 msw but when he went on to 82 msw, he suddenly could not focus his mind and experienced blurred vision, making it difficult to see the rope. He found it impossible to think straight. He headed towards the surface with small pulls on the rope so as not to lose it. When he surfaced his mind was clear again. The next day he repeated a dive to the same depth without any problems.

While individual susceptibility and experiences seem to vary, narcosis is a common problem with deeper breath-hold dives and it seems important for divers to train in order to recognise the symptoms and sustain performance despite these effects. In a survey concerning lifetime experience of narcosis-like symptoms among 24 divers during a competition, we found that 12 had experienced symptoms that could be associated with narcosis, including dizziness and confusion (each seven cases), and these were all evident at depths greater than 40 msw (Schagatay et al, unpublished observations).

Decompression sickness

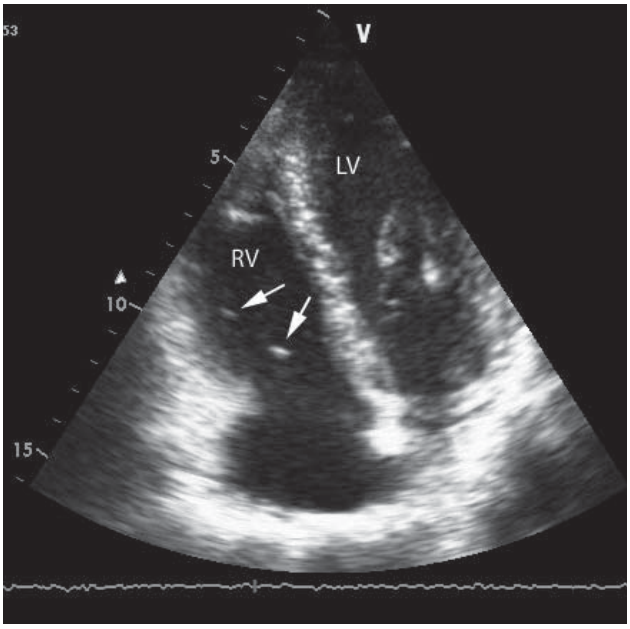
DCS, caused when nitrogen accumulates at depth then forms bubbles in supersaturated tissues during and after ascent, is one of the major limitations of diving breathing compressed gases, but it has long been debated whether breath-hold divers could develop DCS. Early reports of symptoms such as vertigo, nausea, paralysis and unconsciousness were suggestive of neurological DCS after repeated apneic diving in Tuamotu pearl divers.[46] That this could be the case was confirmed when Paulev showed on himself that repeated apnea dives to 20 m depth in a tank resulted in DCS symptoms, which were reversed by recompression in a hyperbaric chamber.[47] It is now generally accepted that repetitive dives even to moderate depths with short intervals may, over time, cause an excessive nitrogen load leading to DCS.[48–50] However, it is usually considered unlikely that competitive divers would be submersed for sufficient time and to sufficient depth to develop DCS, as single or few dives to depth are done in a day. Nevertheless, apnea divers are reaching greater depths, and thereby also increased durations, and DCS-like symptoms are reported after both training and competition, as seen from the following accounts (various, personal communications, 2009).

Diver account 1: An experienced diver was doing six to seven serial dives of 3–3.5 min duration with surface intervals of 3–4 min to 40–45 msw depth, when he suddenly became totally paralysed and unable to speak. His friends took him ashore, and he was taken by ambulance to a specialist hospital with a recompression chamber, but when they heard he had been free-diving, he was transferred to another hospital rather than being recompressed. After about 2 h, the situation spontaneously improved and, after some time, he was able to go home. There were no long-term effects.

Diver account 2: An experienced diver was doing a 'no limit' dive to 90 msw. When she turned on the air for the liftbag, it did not fill up. She tried twice more unsuccessfully and finally the bag filled and the sled started to move, but she had spent much time at depth. Five minutes after surfacing, she developed amnesia which lasted for about 30 min. During the amnesia, she had been "*talking funny*" according to friends who put her on O_2. For one month after this event, she was very weak and thinking and speaking slowly, but her symptoms improved and she could dive again.

We received many similar reports of possible DCS events among elite apnea divers in a 2009 survey (Schagatay E, Lodin-Sundström, unpublished observations). To our

Figure 6
Cardiac ultrasonic image from a diver with occasional bubbles (arrowed) after a competition dive to 70 msw



knowledge, no studies of bubble formation after competitive dives existed, so we also screened for bubbles in divers at two international diving competitions including all three depth disciplines. Mean depths for screened dives were respectively 58, 44 and 56 msw for CWT, CNF and FIM. Divers reported to the laboratory as soon as possible after dive termination, and screening of the right ventricle was done within 10–20 min after dives, using ultrasonic imaging for a single observation of 5–10 min. In about 100 dives, only two low (grade I on the Kisman-Masurel scale[51]) bubble scores were found, one after a CWT dive to 70 msw, the other after a FIM dive to 68 msw (Figure 6).[52] Sixteen of the dives were of depths of 70–90 msw. While bubble formation after deep apnea dives has hereby been confirmed, screening of a greater number of deeper dives over a longer post-dive period is needed to determine the risk for DCS.[53]

These reports and observations illustrate the need for increased awareness among apneic divers of how to prevent nitrogen accumulation and bubble formation, as well as increased awareness of these risks among physicians, so that appropriate treatment is made available. With increasing depths being reached, it becomes relevant to develop advice on free-diving profiles and surface intervals. The ratio between surface interval and diving time determines the nitrogen accumulation and when the ratio is 1 the exposure is roughly equivalent to 50% of the depth for a continuous dive.[54] Some divers, like the Ama and Bajau are capable of a 50/50 ratio over prolonged periods,[55] while competitive apnea divers, despite making fewer dives, reach depths great enough to cause nitrogen accumulation. Whilst the single breath per dive limits nitrogen uptake, the diving response could enhance uptake, by maintaining or increasing circulation to fast tissues such as the brain.

If warm-up dives are kept few and shallow, and spaced by sufficient time and possibly done with empty lungs, their contribution to nitrogen accumulation would be limited. An additional risk factor is that ascent rates are much more rapid in breath-hold diving than those recommended in standard decompression tables. Slowing ascents and including safety stops may help to reduce risk. Normal behaviour patterns may protect diving mammals from decompression injury, but even marine mammals may suffer from DCS in rare events, such as disturbances to their diving pattern from human underwater activity.[56]

The most advanced human divers can make slow ascents if allowed by apneic duration. In his record 'no limit' dive to 214 msw lasting 4 min 24 s, Herbert Nitsch reached target depth within 1 min 45 s, immediately turned and made a fast ascent to 60 msw within 54 s, after which he slowly ascended in 50 s to 10 msw, where he made a 30 s safety stop before slowly surfacing. After an accepted surface protocol, he returned to 10 msw on O_2; a standard method now used after deep competitive dives is for the diver to breathe O_2 at 6 msw depth for a period post-dive.

In deep-diving mammals, some species of which may reach depths of over 1,000 msw, pulmonary shunts and lung collapse may prevent nitrogen uptake and bubble formation on ascent.[50,57,58] For extreme depths in human apnea diving, similar effects may be present, but it is currently unknown when they occur and to what extent the mechanisms could be protective.[50,59] To prevent alveolar gas exchange and thereby be protective against nitrogen accumulation in humans, lung collapse would have to occur at a shallower depth than in most marine species, but modelling in divers after lung packing suggests this occurs at greater depths.[59] Diving with submaximal lung volume would allow collapse at a shallower depth, yet with the great human dependence on lung O_2 stores for apnea, it appears unlikely that this would be beneficial, at least in unassisted diving with great demands on energy for propulsion.

Arterial gas embolism

Another problem related to ascent from depth is AGE. AGE occurs as a direct effect of expansion of trapped air during ascent leading to tissue rupture and, when this occurs in lungs, bubbles may pass into the bloodstream to the left side of the heart.[60] This may lead to bubbles being transported to the brain, where they can block vessels leading to stroke-like symptoms. The development of AGE is increased with rapid ascent, as gas trapping is more likely to occur, especially in regions of the lung injured from pre-dive lung packing or squeeze during the descent in a deep dive. Despite different causes, the symptoms are similar and the treatment of AGE is the same as those for DCS, the most important actions being to give first-aid O_2 and to transport the diver to a recompression chamber.

Psychological requirements

Stress management, 'guts' and self-preservation

Stress management in order to achieve maximal relaxation and minimal metabolic rate is important in all disciplines of competitive apnea, and the mental capacity (stamina) to tolerate the extreme discomfort of an increasing urge to breathe and progressively enforced involuntary breathing movements ('contractions') for performance in static and dynamic apnea should not be neglected. However, in addition to these factors, a new psychological aspect will become crucial in deep diving; the way down is usually easy, but as you cannot start breathing before surfacing, it is an all-or-nothing event, and risk management is essential.

Whilst in deep diving, the *negative ascent* after turning at depth is the most physically demanding part of the dive, the *free-fall* may, in this respect, be the most psychologically demanding. The diver must decide before the dive what depth to aim for, but determine during the easiest course of the dive how well the *negative ascent* will be managed; and decide if an early turn should be performed or not. This is a balance between risk taking and safety awareness. To

Table 2
Factors rated by divers as the main limiting factor per discipline of apnea: CWT – constant weight with fins; DYN – dynamic apnea distance swimming, and STA – static apnea

	Pressure effects	O_2/CO_2 storage	Relaxation	Training capacity	Equipment	Other	Total no. answers
CWT	8	5	-	1	-	1 (science)	15
DYN	-	10	-	2	2	1 (diet)	15
STA	-	9	6	-	-	1 (diet)	16

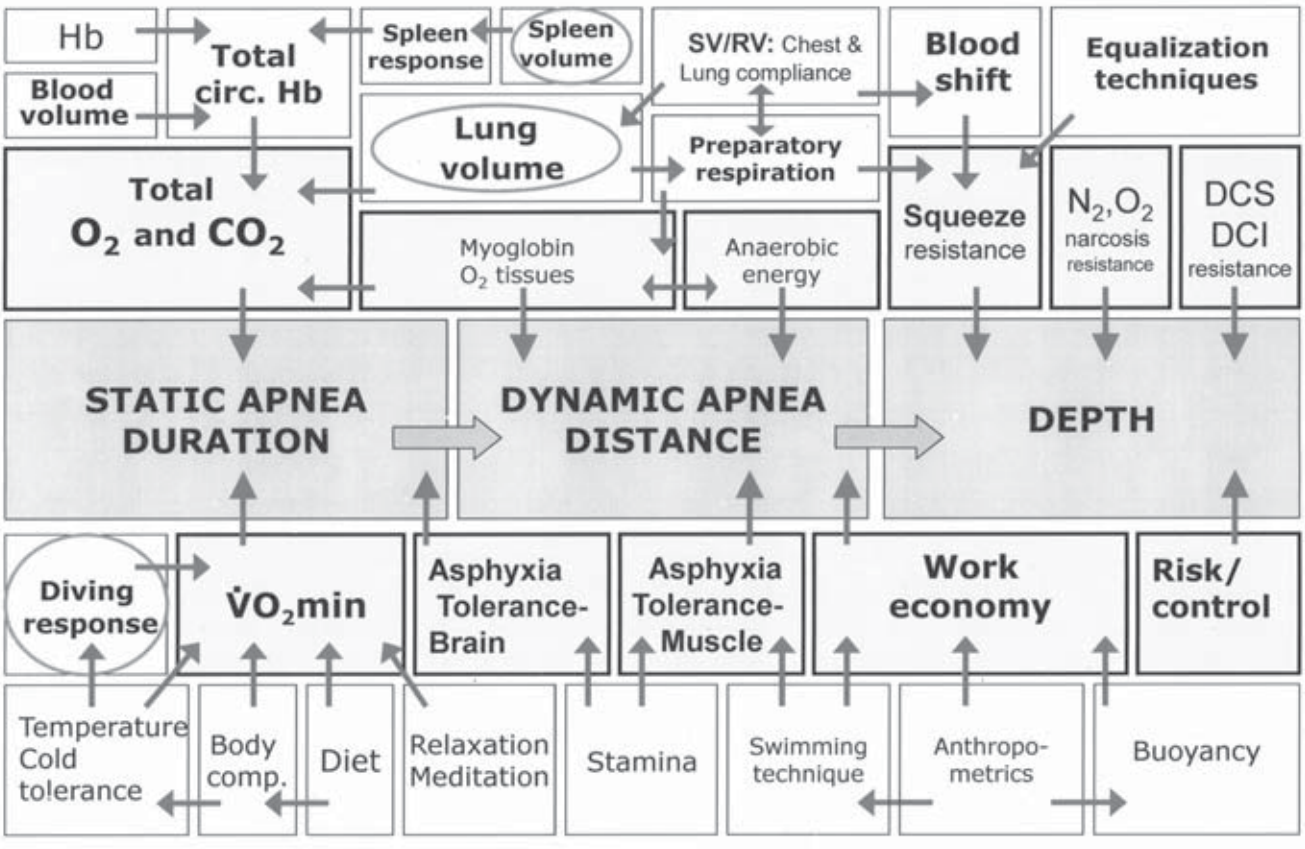
be deprived of the possibility to breathe during hard work, when the urge becomes overwhelming, is truly frightening. To make decisions about how to manage the last part of the dive and reach the surface without blacking out, before being at the midpoint is an unusual challenge. It is essential to keep this focus even under the influence of narcosis.

The commonly held view that these attempts are made by suicidal dare-devils is incorrect; breath-hold divers are generally highly concerned with safety and do not want to take unnecessary risks. Yet the balance between a safe and easy dive and an extremely uncomfortable event leading to a record has a component of 'pure guts' and determination to it. This is paralleled in very few sports except, perhaps, extreme skiing and climbing.

Where are the limits?

Predicting the limits in deep apneic diving is not easy, as a new set of factors related to depth are added to all the ones determining resting apnea duration and shallow diving working capacity. Essential in the development of records is to balance faster speed with reduction in energy cost; just faster speed will not help, as the energy requirements may increase and duration shorten. While apneic duration appears not to have been the major limitation thus far, greater depths may call for exceptionally long apneas and slower ascents with safety stops aiming at avoiding DCS. Thus, only descent speed may be increased; but that puts great pressure on the diver's equalization techniques and the body's resistance to barotrauma. Increases in body oxygen storage as well as

Figure 7
Factors influencing performance in the various apnea disciplines; factors affecting static apnea are transfered right to dynamic apnea, which then all combine with new factors added for depth (for explanations see text)



enhanced tolerance to asphyxia will allow deeper dives even at the same speed and metabolic rate.[1,2]

It is clear that individual athletes may be limited by different factors, but in order to get further, all eventually have to deal with the enhanced effects of hydrostatic pressure. Physiological features involved may include lung volume, where both TLC and the SV/RV ratio are important, blood shift and resistance to squeeze, haemoglobin concentration and total haemoglobin, splenic volume and maximal contraction, muscle myoglobin concentration, the diving response, work economy, cold tolerance, asphyxia tolerance, anaerobic capacity and minimum metabolic rate, special techniques for respiration, inspiration and equalization, and also psychological factors involving stress control, risk/caution and to some extent 'pure guts' (Figure 7). As in all sports, maximal performance will be determined by the maximum capacity of individuals, achieved by genetic predisposition, long-term training, and use of proper techniques for preparation and performance. The ultimate limits will be reached only if participation in the sport of apnea diving approaches that of more widespread sports.

Input from elite divers

Physiologists are historically known to be utterly wrong in predictions of maximal performance, partly because these are often based on laboratory-derived data from apnea experiments collected from non-divers or moderately trained divers. Even after several studies of the physiology of elite divers, predicting future performance is difficult, as new divers enter the field and training methods improve. My prediction in the first review of a likely maximum static apnea time of slightly over 11 minutes, based on current data from active divers, was redundant when a new record of 11 min 35 s was set the same year![1]

We asked elite apneic divers what they thought would set the limits of free-diving. Seventeen divers (10 males and seven females) participating in the 2008 apnea world championship were asked to predict the ultimate performance in the CWT, DYN and STA disciplines and to list the main limiting factor per discipline, without suggestions given as to what these factors might be. Their mean (SD) personal bests were 63 (15) msw in CWT, 136 (30) msw in DYN and for STA 6 min 4 s (13 s). Predicted ultimate limits in the three disciplines were for CWT 156 (13) msw depth, for DYN 323 (31) metres' distance and for STA a time of 14 min 54 s (3 min 17 s). Different limiting factors were stated for the different disciplines of competitive apnea (Table 2). In CWT, the most frequent 'number one limitation' stated was 'pressure effects' followed by five first ratings for gas storage (Schagatay E and Lodin-Sundström A, unpublished observations, 2008).

Based on physiological measurements and observations in elite divers, and the fact that the current record development does not seem to be levelling off, I would consider these predictions to be realistic, with the pressure effects related to nitrogen saturation most likely setting the ultimate limits for the depth disciplines.

Conclusions

Currently, deep diving problems are dominated by hypoxic syncope on surfacing and barotraumas which, even if not life-threatening, may stop many individual divers from reaching further. However, the 214 msw performance in the 'no limit' discipline suggests that certain individuals may tolerate nearly complete compression of the lungs and airways. As DCS and nitrogen narcosis cannot likely be avoided in man by diving on empty lungs allowing early lung collapse, because of human dependence on lung oxygen stores, I would hold it likely that the ultimate limits for deep diving will be set by these factors, when training techniques have been developed further to prevent barotraumas, hypoxic syncope and AGE. Further technical developments may also be needed, and safety arrangements must develop concomitantly with the increase in depth.

Acknowledgements

I wish to thank all the elite divers who participated in our studies and those who shared their experiences and views concerning competitive apnea diving techniques and limits, especially Dr Natalia Molchanova, Sara Campbell, Annelie Pompe, Lotta Ericson, Stephane Mifsud, Dr Stig Severinsen, Herbert Nitsch, and Sebastian Näslund for discussions of special techniques. A special thanks also to the many people helping us with experiments around the competition dive sites, to my co-workers in the Environmental Physiology Group and other laboratories for their scientific contributions and to Christian Ernest for reviewing the manuscript, and finally to the still-going-strong Enzo Maiorca, for inspiration.

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Submitted: 24 October 2011
Accepted: 02 November 2011

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