Review articles

Breath-hold diving: performance and safety

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Key words
Breath-hold diving, freediving, deep diving, physiology, ascent, hypoxia, safety, review article

Abstract

(Pollock NW. Breath-hold diving: performance and safety. *Diving and Hyperbaric Medicine.* 2008; 38: 79-86.)
Breath-hold diving was probably first conducted shortly after humans ventured into the water realm. Early efforts likely centred on exploration, hunting and gathering. The fundamentals of breath-hold diving have not changed since these earliest efforts but the performance records have undergone an almost unbelievable evolution. Single breath-hold durations (inspiring air) of 9:15 min:s and 8:00 min:s and maximal vertical transits of 214 metres' sea water (msw) and 160 msw for males and females, respectively, are daunting. Competitive performance requires genetic predisposition, motivation and training in both fundamentals and a variety of advanced techniques. The record of safety within the competitive arena is impressive but care must be taken to ensure that the appropriate practices and procedures are communicated to all levels of enthusiast. Current performance records, strategies to optimize performance, and recommendations for safe breath-hold activity are presented.

Introduction

Breath-hold or apnoea diving, increasingly known as freediving, describes in-water activity involving some diving equipment, but no self-contained or surface-supplied breathing gas. Freedivers operate in a wide range of environments and pursue an assortment of goals. Leisure recreation may range from surface snorkelling with little or no voluntary breath-hold to modest surface dives with variable breath-hold effort. Organized sports, typically conducted in swimming pools, include underwater hockey and underwater rugby. Exploration, hunting/spearfishing and other food-gathering activities can vary dramatically with the individual and targets. Formal competition in breath-hold diving has grown rapidly in recent years as an extreme sport. Numerous disciplines are now recognized by the International Association for the Development of Apnoea (AIDA; http://www.aida-international.org/) (Table 1).

Early wisdom held that the safe diving depth during breath-hold was limited by the ratio between total lung capacity and residual volume. The belief was that lungs compressed below residual volume would suffer from barotrauma. For example, a person with a total lung capacity of 6.0 L and a residual volume of 1.2 L would have an approximate maximum safe depth limit of five atmospheres absolute, or 40 metres' sea water (msw). This proposition has been clearly disproved through freediving competition. The accelerated advance of performance records within the past decade reflects the growing enthusiasm for the sport. The greatest depths are achieved in the No-Limits discipline, with a timeline of record depths shown in Figure 1. The current male depth record is 214 metres' sea water (msw), set by Herbert Nitsch of Austria in June 2007. The current female record is 160 msw, set by Tanya Streeter, originally from Britain, in August 2002. The current record for static breath-hold is 9:15 min:s, set by Tom Siestas of Germany in May 2008. The current records in all AIDA-recognized disciplines are shown in Table 1.

Chasing record breath-hold performance

The best breath-hold performance will be achieved through a combination of genetic predisposition, motivation, training and physiological manipulation. The ability to pick your parents may one day be unnecessary as the science of genetic manipulation evolves, but for now the edge still goes to the naturally endowed apnoea athlete. Motivation is an interesting

Figure 1
World record depths in freediving no-limits competition

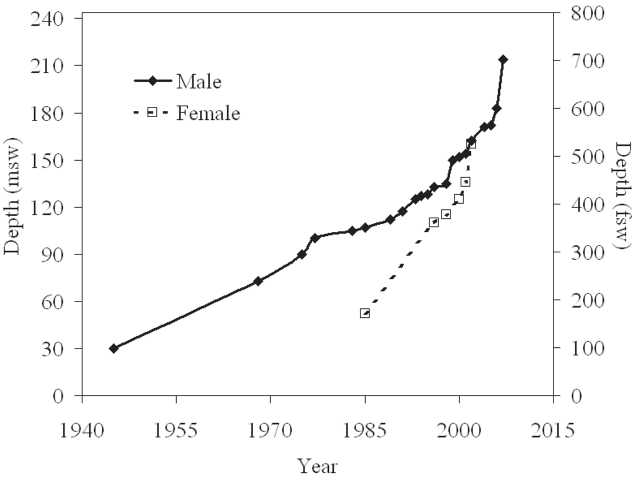


Table 1
AIDA-recognized competitive freediving disciplines and records as of 15 May 2008 (*horizontal swim)

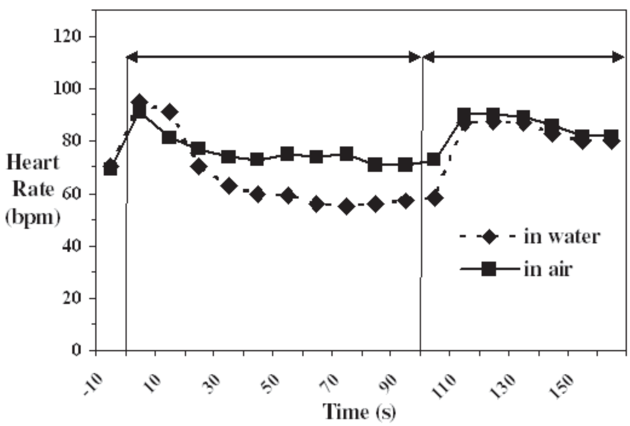
Discipline	Description	Record performance Male	Record performance Female
		Time (min:s)	
Static Apnoea	Resting, immersed breath-hold in controlled water (usually a shallow swimming pool)	9:15	8:00
		Distance/depth (m [ft])	
Dynamic Apnoea – with fins	Horizontal swim in controlled water	244 (801)*	205 (673)*
Dynamic Apnoea – no fins	Horizontal swim in controlled water	186 (610)*	149 (489)*
No-Limits	Vertical descent to a maximum depth on a weighted sled; ascent with a lift bag deployed by the diver	214 (702)	160 (525)
Variable Weight/Ballast	Vertical descent to a maximum depth on weighted sled; ascent by pulling up a line and/or kicking	140 (459)	122 (400)
Constant Weight – with fins	Vertical self-propelled swimming to a maximum depth and back to surface; no line assistance allowed	112 (367)	90 (295)
Constant Weight – no fins	Vertical self-propelled swimming to a maximum depth and back to surface; no line assistance allowed	86 (282)	57 (187)
Free Immersion – no fins	Vertical excursion propelled by pulling on the rope during descent and ascent; no fins	108 (354)	81 (266)

factor when viewed in the context of predisposition. While increasing in popularity, it is still the minority who pursue the challenge of breath-hold competition. The seemingly meteoric rise of some newcomers begs the question about the undiscovered potential outside of the breath-hold community. The factors of training and physiological manipulation are intertwined; training in physiological and, in some cases, anatomical manipulation is a major aspect of the preparation process.

Breath-hold basics

Physiological alterations begin with head-out immersion in water. Intrathoracic blood volume is increased while vital capacity is decreased. Facial immersion, particularly in cool water, initiates the classic diving reflex observed during breath-hold. Heart rate and cardiac output are decreased and peripheral vasoconstriction and blood pressure are increased.[1–3] An example of heart-rate response to facial immersion is depicted in Figure 2.

Figure 2
Heart rate response to resting apnoea in air and during facial immersion (recreated from reference 1)



More recently, the spleen has been appreciated as a source of red blood cells available to the body during breath-hold.[4–6] A modest decrease in splenic volume follows the initiation of breath-hold during facial immersion. One study of repetitive breath-hold performance was conducted with two-minute rest intervals, before splenic volume had recovered. The duration of the second maximal breath-hold was about 20% longer for both trained breath-hold divers and untrained subjects, and only five per cent longer for splenectomized subjects.[6] Greater changes in maximum duration from first to second breath-hold have been seen in other studies using similar procedures but not studying splenic response.[7,8]

A period of apnoea follows every inspiration. It is the duration of the apnoea that sets apart the unconscious breath-hold from the voluntary breath-hold. The partial pressure of carbon dioxide (PCO_2) in the arterial blood is the primary agent driving the respiratory cycle. The PCO_2 in the unconscious control cycles from a low of 40 mm Hg at end-inspiration to approximately 45 mmHg at pre-inspiration. Voluntary breath-hold can allow alveolar PCO_2 to reach into the range of 60 mmHg for the motivated individual. The drive to breathe increases concomitant with the rise in PCO_2 until the breath-hold is broken.

Maximal breath-hold has been described as a two-phase event.[9] The 'easy-going' phase ends at the physiologic breakpoint, with alveolar PCO_2 approximating that at the end of unconscious breath-hold. The 'struggle' phase follows with increasingly active involuntary respiratory muscle contraction until the 'conventional' breakpoint is reached and the breath-hold is broken. Breath-hold time can be increased by either delaying the physiological conditions reaching breakpoint status or increasing the individual tolerance to such conditions.

Extreme breath-hold efforts are not without risk. Injury and death do occur, most commonly amongst less experienced divers. Techniques for improving breath-hold performance are best practised in a controlled setting with responsible oversight.

Manipulative practices

REDUCE METABOLIC DEMAND

One of the simplest ways to extend breath-hold time is to reduce the physical effort involved and thus the metabolic demand. Comparison between the record performances of the no-fin and fin sub-categories of the breath-hold disciplines of Dynamic Apnoea and Constant Weight illustrates the energetic advantage of using fins. The Static Apnoea and No-Limits disciplines demonstrate the extremes of what can be accomplished on a single breath of air if minimal physical effort is involved.

DISTRACTION

Various techniques can be employed to prolong breath-hold time. A simple strategy involves the use of distraction. For example, encouraging relatively inexperienced breath-hold divers to meet the onset of the serious urge to breathe with an attempt to swallow can markedly improve performance. It is difficult to swallow underwater in this condition and the distraction can reduce the sense of urgency. This or other distracting techniques can be used to demonstrate the flexibility in the breathing urge.

HYPERVENTILATION

The most well-known manipulative practice used to increase breath-hold time is hyperventilation. The basis for its effectiveness is the 140–170-fold higher concentration of CO_2 in the arterial blood than that of room air. Increasing the ventilatory exchange beyond that required to meet metabolic needs can produce a rapid drop in arterial PCO_2, potentially reducing it to 20 mmHg or lower with 60 s of aggressive hyperventilation (an arterial PCO_2 of 20 mmHg corresponds to the point at which peripheral symptoms will often be noticed, commonly lightheadedness, possible visual disturbances, and a tingling in the fingertips). A breath-hold commenced after hyperventilation can be prolonged substantially since the hypercapnic drive to breathe will not develop until the normal trigger point is reached. The primary risk of pre-breath-hold hyperventilation is hypoxia. While the arterial PO_2 is slightly increased with hyperventilation, this is nowhere near the magnitude of the decline in arterial PCO_2. Serious hypoxia can develop long before the hypercapnic trigger is reached. A weak hypoxic ventilatory drive could result in unconsciousness before the diver feels any urge to breathe. Hyperventilation can be dangerous, particularly when the potential for unconsciousness without warning is ignored or disbelieved. The likelihood for disaster is increased by the very normal mindset of 'if a little is good, more is better'. A major problem in communicating the risk is the lack of physical evidence left to establish hyperventilation as an agent in breath-hold fatalities. It very likely plays a significant role in a large number of unexplained cases, a problem observed but not overcome in almost 50 years.[10–13]

Apnoeic hypoxia

Loss of consciousness occurring at or near the maximum depth of a dive could follow extreme hyperventilation. This can be described as static blackout. The situation rarely develops during vertical breath-hold dives since the compression effect of descent increases both PCO_2 and PO_2, effectively providing a safety net for the breath-hold diver at depth. The greater hazard develops during surfacing, when the reduction in ambient pressure makes both PCO_2 and PO_2 fall. The PCO_2 will move more slowly towards the breakpoint and the PO_2 will decline faster than explained by metabolic consumption alone. As PO_2 moves beyond the shoulder into the steepest portion of the oxygen dissociation curve, haemoglobin saturation and arterial oxygen content will fall precipitously. A state of problematic hypoxia can develop rapidly, particularly in the final stage of ascent where the relative rate of pressure reduction is the greatest. Even a strong hypoxic drive could be ineffective at this point. Loss of consciousness will commonly occur just before or within 10–15 s of surfacing before the oxygen in the first inspired breath can reach the brain. This condition is best referred to as hypoxia of ascent. While many will know it as 'shallow water blackout', this term is ambiguous (and therefore not recommended) since it was first used to describe unconsciousness in closed-circuit oxygen divers likely caused by high PCO_2.[14]

GLOSSOPHARYNGEAL BREATHING

The use of glossopharyngeal breathing to alter normal pulmonary volumes has gained popularity within the competitive freediving community and as a recent topic of academic study. Glossopharyngeal insufflation (also known as 'lung packing' or 'buccal pumping') is used to increase available gas above normal vital capacity. This involves gulping in a series of small volumes of air after the point of normal full inspiration has been reached, increasing both volume and intrapulmonary gas compression.[15–21] The volume of air held can be increased substantially, in one case by as much as 4.2 L (47%) over measured vital capacity.[20] Most efforts are less extreme, one study documenting a mean (± standard deviation) increase of 1.1±1.3 L (18%) over measured vital capacity.[22] Glossopharyngeal insufflation increases both the available O_2 stores and the depth at which pulmonary compression will become problematic. Aggressive efforts may result in a decreased arterial pressure, increased heart rate, reduced stroke volume, increased transpulmonary pressure and pulmonary vascular resistance, dizziness, tunnel vision, syncope,[23] and possibly pulmonary barotrauma.

Glossopharyngeal exsufflation (also known as 'reverse packing') involves use of the muscles of the glossopharynx to draw air out of the lungs and into the mouth when the lung volume is near or below residual volume.[16,20] This technique can be used to enable middle ear pressure equalisation at depths deeper than would otherwise be possible. Glossopharyngeal exsufflation can allow an additional 0.2–0.4 L to be withdrawn from lungs at residual volume.[24] Breath-hold dives initiated with the lung at residual volume ('empty lung') can also be used to simulate the effects of deeper diving under shallow and more controlled conditions. These effects are even greater if glossopharyngeal exsufflation is employed. A recent observational study followed experienced breath-hold divers conducting repetitive empty lung dives with self-selected amounts of glossopharyngeal exsufflation in a single 20-minute session with dives to depths of 3–6 m. The research team documented reversible changes in voice and compromised pulmonary diffusion capacity, irritation and slight congestion in the larynx and, in some cases, minor bleeding originating somewhere below the vocal folds.[24]

EQUALISATION TECHNIQUES

The need to equalise the sinus and middle ear spaces can be a major challenge of rapid descent through the water column. Equalisation by standard techniques, primarily Valsalva, requires concentration and some degree of muscular effort. As discussed above, glossopharyngeal exsufflation may be used to facilitate this effort with the added cost of removing oxygenated gas from the lungs. An alternative used by some competitors is to fill the spaces with liquid to reduce the effort, time and gas costs associated with equalisation. A recent case report describes the ability of a previous world record holder in the No-Limits category to passively fill his sinuses and auditory tubes with seawater during descent to obviate the need for further equalisation.[25]

MODIFICATION OF SUBSTRATE UTILIZATION

Increased competition means that small advantages can make a critical difference in final ranking. The respiratory exchange ratio (RER) is determined from expired gases to estimate whole body metabolic status, and is most meaningful under steady-state conditions. RER is computed as the volume of CO_2 expired divided by the amount of O_2 consumed. Resting RER normally approximates 0.82–0.85. Lower values indicate a shift towards fat metabolism (with a lower oxygen cost per unit energy provided) and higher values indicate a shift towards carbohydrate metabolism (with a higher oxygen cost per unit energy provided). RER rises as a function of exercise intensity, one reason to encourage minimal effort during breath-hold. Dietary intake and digestive status can also influence RER. The question recently addressed was whether dietary manipulation could affect breath-hold performance. A study of experienced breath-hold divers with depleted carbohydrate stores demonstrated changes in breakpoint CO_2 and O_2 but no change in maximal breath-hold time.[26] Conversely, a study of untrained breath-holders documented maximal breath-hold time increased by fasting and decreased by carbohydrate-rich food intake.[27] It is expected that further work, either academic or training-based, will be conducted in an effort to develop optimal dietary patterns for competitors.

Adaptation to breath-hold

The question of predisposition versus adaptation frequently accompanies extreme performance. For example, vital capacity in breath-hold divers is commonly observed to be, on average, larger than predicted.[16,20,21,28] While some of this may reflect individual predisposition or self-selection, there is also likely a response to training. Five- to six-week training programmes in glossopharyngeal insufflation have been shown to increase vital capacity within groups of normal, healthy females and elite female swimmers.[29,30]

A more open question concerns respiratory drive. One study of competitive underwater hockey players documented a marked difference in response to CO_2 rebreathing (hypercapnic ventilatory response) in comparison with dry-land athletes.[31] The underwater hockey players' responses were on the low end of the normal range, a pattern similar to that seen in a more recent study of two elite breath-hold divers.[32] Another recent study suggested that underwater hockey players might maintain higher resting end-tidal PCO_2 than control subjects,[33] but the low values for the control group suggest that hyperventilation due to an anticipatory response and/or as a response to the mouthpiece could explain the difference.

A small study of sympathetic nerve activity during tests of hypercapnic ventilatory response found similar patterns for nine elite breath-hold divers and a control group comprising individuals who were not regular divers.[34] Another small study of four elite breath-hold divers documented the individual response to air hunger while the subjects were mechanically ventilated with different levels (randomised) of hypercapnic gas. The subjective ratings were within the range of normal air hunger responses for three of the four subjects. The fourth reported no sensations corresponding to air hunger.[32]

The focus on hypercapnic responses in breath-hold research is likely influenced by the common description of the hypoxic drive being weak and therefore a risk factor following hyperventilation. Studies of hypoxic drive and breath-hold, however, indicate that the strength of the two drives may be much more comparable than previously thought.[35,36] An observational study of eight amateur, trained breath-hold divers evaluated respiratory conditions before and after static apnoea dives.[37] Hyperventilation before breath-hold reduced end-tidal PCO_2 to 18.9±2.0 (15.6–21.9) mmHg (mean ± standard deviation with range). Post-breath-hold end-tidal PCO_2 was 38.3±4.7 (29.5–43.4) mmHg and end-tidal PO_2 was 26.9±7.5 (19.6–42.2) mmHg, leading the authors to conclude that the hypoxic drive may have been the dominant factor in terminating breath-hold. The simple expedient of breathing oxygen pre-dive is well known to extend breath-hold time considerably in the laboratory, but is certainly not to be recommended in the open water.

One study of splenic contraction during breath-hold compared trained apnoea divers and untrained controls.[6] The trained divers demonstrated a significantly greater reduction in splenic volume (18% versus 14%), but the difference is small enough to be of limited practical importance.

Breath-hold safety

TRAINING AND SUPERVISION

Safety in breath-hold, as with most endeavors, is best ensured by forethought and a respect for reasonable guidelines. The impressive safety record maintained in competitive events[38] can be replicated at other levels of involvement only if an appreciation for both the techniques and risks is provided. Training programmes should be available to teach appropriate procedures and protocols to ensure safe participation. As a fundamental rule, breath-hold activities should be conducted with a responsible partner or team regardless of the conditions. There are many examples of individuals found dead with no evidence of trauma. While often impossible to confirm, it is likely that manipulative breath-hold practices were involved and that the ready presence of others could have provided timely intervention in many cases.

Effective direct supervision requires an awareness of the diver’s activity during the pre-breath-hold period, close monitoring throughout the breath-hold and 30-second post-breath-hold periods, attention to any sign of compromise, and the ability to provide immediate and effective support in case of a problem. Direct supervision can be applied to many situations. A simple two-person, one-up-one-down buddy team with committed direct supervision can provide effective protection for shallow diving situations. A group of three (one-down, two-up) may be preferable as dive depths increase. Allowing a recovery period of twice the dive duration is a reasonable practice. It also ensures that one of the divers available at the surface for backup is substantially rested. Problems can arise when dive depths approach individual limits. The breath-hold performance of a potential rescuer may be badly compromised by the stress of an emergent situation.

More advanced activities require a more extensive support network, potentially employing counterbalance weighting and/or buoyancy retrieval systems and/or in-water rescue divers.[39] The rarity of serious accidents within breath-hold competitions is a testament to thoughtful planning, proper equipment and monitoring, and emergency protocols.

BUOYANCY

Buoyancy is an important consideration for breath-hold divers. Some choose to wear extra weight to minimize the effort associated with descent. The hazards during ascent, however, far outweigh the benefits of an easy descent. Buoyancy is lost under pressure, more so when a compressible suit is worn, thus a considerable degree of negative buoyancy may exist at depth. This will increase the effort required to ascend (increasing the rate of oxygen consumption and decreasing safe breath-hold time). As discussed, hypoxia of ascent is most likely to develop in the final stage of the ascent. A diver negatively buoyant near the surface will rapidly sink if consciousness is lost. This will make it more difficult, or in some cases impossible, for a timely rescue to be completed. Overweighting is frequently a contributing factor in fatal cases attributed to hypoxic loss of consciousness.[40] For safety reasons, it is recommended that divers are weighted to be neutrally buoyant at a depth of approximately 5 msw.

HYPERVENTILATION

A balance between prolonging breath-hold and mitigating the risk of loss of consciousness is possible by restricting the amount of pre-breath-hold hyperventilation. Limiting hyperventilation to two or three maximal ventilatory exchanges immediately prior to breath-hold will increase breath-hold time but is probably also safe for most leisure circumstances. An early review of non-fatal cases of loss of consciousness attributed to pre-breath-hold hyperventilation described much more extreme efforts.[11]

The competitive freediving community is generally well informed of the hazards of hyperventilation and accepts the attendant risks of more aggressive use of the technique. Most competitors employ some degree of hyperventilation.[22,38] A critical factor, though, is that the risks are mitigated by carefully structured, close monitoring and support protocols.

New terms have entered the lexicon of competitive freediving to describe the range of altered consciousness that may be experienced. “Mooglies” represent language production disturbances occurring at the end of a breath-hold.[41] “Samba” represents a loss of motor control that may include confusion, affected postural control, or muscular spasms.[41] Both loss of consciousness and significant loss of motor control disqualify competitive performance.[42] Minor losses of motor control are considered normal enough in competitive events to not warrant physical examination.[38] It is likely that the relative rarity of serious syncopal events in competition is influenced by strict rules for obligatory delivery of a series of clear and orderly signals by the surfacing diver to avoid disqualification of the attempt.

The natural migration of practices is a concern with hyperventilation. The problem may even be increased by a shift in terminology. It is not uncommon to find breath-hold divers who will deny any use of pre-breath-hold hyperventilation but will then talk about "work-up breathing", which, when described, is effectively hyperventilation. Risk is controlled through realistic appreciation and appropriate responses, not by obfuscation. Care must be taken to ensure that all participants have a realistic appreciation for the effects and risks of practices. The most critical hazard in breath-hold is that of a compromised level of consciousness developing under less than closely monitored conditions. The fear is that the excitement of breath-hold diving can spread faster than an appreciation of the risks. The finding that more than half of recently reported fatal breath-hold incidents were unwitnessed indicates a fundamental problem in practice.[13]

LIMIT BREATH-HOLD TIME

Another way to address the risk of altered consciousness is to arbitrarily limit breath-hold time. One recommendation was proposed for non-competitive breath-hold diving.[43] The point was made that hyperventilation can increase the pleasure of breath-hold dives by reducing symptoms of air hunger. Restricting dive time could reduce the risk of hypoxic compromise developing regardless of the aggressiveness of the pre-breath-hold practices employed. Based on a review of incidents, the author concluded that limiting breath-hold time to 60 s would allow for varying patterns of hyperventilation and physical activity with minimal risk of loss of consciousness. This is a reasonable safety guideline and easy to apply for recreational freedivers, particularly those with minimal experience. The only tool required to make it work is a watch with a countdown function set to beep at 45 or 50 s as appropriate for the depth to remind the diver when to begin to ascend. Freedivers who gain additional experience or progress to more advanced freediving activities will have a safe starting point from which to grow.

FLOTATION VESTS

Flotation vests designed specifically for breath-hold diving represent a potentially important safety tool currently under development. These will automatically inflate if a user-preset (adjustable) time at depth is exceeded. While such devices will not eliminate the risk of static blackout or hypoxia of ascent, or the risk of inspiring water should unconsciousness develop, they will reduce the mortal risk of such events by returning the diver to the surface. The likelihood of being unable to recover a diver in distress would be markedly reduced. This would be an extremely effective aid in many small operations or if the conditions were such that some of the support team might be forced to dive near or beyond their capabilities to perform a rescue.

REPORTING DIVING INCIDENTS

Communication of the details involved in both fatal and non-fatal breath-hold incidents is another tool to improve the safety of current and future freedivers. Learning from the mistakes of others is an important strategy in promoting safety for all. The ability to share salient details about incidents should encourage divers to reflect on and, where appropriate, improve their own practices. Divers Alert Network has included a summary section and brief case reports concerning breath-hold incidents in the annual report on diving safety since 2005.[13,44,45] Primarily focused on fatal incidents, the programme is expanding to capture non-fatal events.[46]

Conclusions

Breath-hold diving is experiencing a growth in popularity that reflects an impressive evolution of record-setting performance. Safe participation in competitive events is fostered by a wide range of carefully developed regulations and protocols. Communication of appropriate safety procedures is critical to ensure that enthusiasts at all levels of involvement are reasonably protected. Elimination of solo freediving and the incautious use of hyperventilation would have the greatest impact on the population at risk. Development of appropriate and accessible training programmes and the regular communication of incident case reports are important strategies to increase awareness of both risks and appropriate practices.

References

1 Strømme SB, Kerem D, Elsner R. Diving bradycardia during rest and exercise and its relation to physical fitness. *J Appl Physiol*. 1970; 28: 614-21.
2 Andersson JPA, Linér MH, Rünow E, Schagatay EKA. Diving response and arterial oxygen saturation during apnea and exercise in breath-hold divers. *J Appl Physiol*. 2002; 93: 882-6.
3 Lindholm P, Nordh J, Gennser M. The heart rate of breath-hold divers during static apnea: effects of competitive stress. *Undersea Hyperb Med*. 2006; 33: 119-24.
4 Hurford WE, Hong SK, Park YS, Ahn DW, Shiraki K, et al. Splenic contraction during breath-hold diving in the Korean ama. *J Appl Physiol*. 1990; 69: 932-6.
5 Espersen K, Frandsen H, Lorentzen T, Kanstrup IL, Christensen NJ. The human spleen as an erythrocyte reservoir in diving-related interventions. *J Appl Physiol*. 2002; 92: 2071-9.
6 Bakovic D, Valic Z, Eterovic D, Vukovic I, Obad A, et

al. Spleen volume and blood flow response to repeated breath-hold apneas. *J Appl Physiol*. 2003; 95: 1460-6.
7 Stewart IB, Bulmer AC, Sharman JE, Ridgway L. Arterial oxygen desaturation kinetics during apnea. *Med Sci Sports Exerc*. 2005; 37: 1871-6.
8 Valic Z, Palada I, Bakovic D, Valic M, Mardesic-Brakus S, Dujic Z. Muscle oxygen supply during cold face immersion in breath-hold divers and controls. *Aviat Space Environ Med*. 2006; 77: 1224-9.
9 Lin YC, Lally DA, Moore TO, Hong SK. Physiological and conventional breath-hold breaking points. *J Appl Physiol*. 1974; 37: 291-6.
10 Craig AB Jr. Causes of loss of consciousness during underwater swimming. *J Appl Physiol*. 1961; 16: 583-6.
11 Craig AB Jr. Underwater swimming and loss of consciousness. *JAMA*. 1961; 176: 255-8.
12 Craig AB Jr. Summary of 58 cases of loss of consciousness during underwater swimming and diving. *Med Sci Sports*. 1976; 8: 171-5.
13 Pollock NW, Vann RD, Denoble PJ, Freiberger JJ, Dovenbarger JA, et al. *Annual diving report*, 2007 edition. Durham, NC: Divers Alert Network; 2007. p. 61-8, 100-3.
14 Donald KW. *Oxygen poisoning in man*. Admiralty Experimental Diving Unit Report number XVI, 1946; 172-81.
15 Simpson G, Ferns J, Murat S. Pulmonary effects of 'lung packing' by buccal pumping in an elite breath-hold diver. *SPUMS J*. 2003; 33:122-6.
16 Lindholm P, Nyren S. Studies on inspiratory and expiratory glossopharyngeal breathing in breath-hold divers employing magnetic resonance imaging and spirometry. *Eur J Appl Physiol*. 2005; 94: 646-51.
17 Jacobson FL, Loring SH, Ferrigno M. Pneumomediastinum after lung packing. *Undersea Hyperb Med*. 2006; 33: 313-6.
18 Overgaard K, Friis S, Pedersen RB, Lykkeboe G. Influence of lung volume, glossopharyngeal inhalation and $P_{ET}O_2$ and $P_{ET}CO_2$ on apnea performance in trained breath-hold divers. *Eur J Appl Physiol*. 2006; 97: 158-64.
19 Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins CR. Features of glossopharyngeal breathing in breath-hold divers. *J Appl Physiol*. 2006; 101: 799-801.
20 Loring SH, O'Donnell CR, Butler JP, Lindholm P, Jacobson F, Ferrigno M. Transpulmonary pressures and lung mechanics with glossopharyngeal insufflation and exsufflation beyond normal lung volumes in competitive breath-hold divers. *J Appl Physiol*. 2007; 102: 841-6.
21 Potkin RT, Cheng V, Siegel R. Effects of glossopharyngeal insufflation on cardiac function: an echocardiographic study in elite breath-hold divers. *J Appl Physiol*. 2007; 103: 823-7.
22 Liner MH, Andersson JPA. Pulmonary edema after competitive breath-hold diving. *J Appl Physiol*. 2008; 104: 986-90.
23 Novalija J, Lindholm P, Loring SH, Diaz E, Fox JA, Ferrigno M. Cardiovascular aspects of glossopharyngeal insufflation and exsufflation. *Undersea Hyperb Med*. 2007; 34: 415-23.
24 Lindholm P, Ekborn A, Oberg D, Gennser M. Pulmonary edema and hemoptysis after breath-hold diving at residual volume. *J Appl Physiol*. 2008; 104: 912-7.
25 Germonpre P, Balestra C, Musimu P. Passive flooding of paranasal sinuses and middle ears as a method of equalisation in extreme breath-hold diving. *Br J Sports Med*. 2008; Epub ahead of print.
26 Lindholm P, Gennser M. Aggravated hypoxia during breath-holds after prolonged exercise. *Eur J Appl Physiol*. 2005; 93: 701-7.
27 Lindholm P, Conniff M, Gennser M, Pendergast D, Lundgren C. Effects of fasting and carbohydrate consumption on voluntary resting apnea duration. *Eur J Appl Physiol*. 2007; 100: 417-25.
28 Song SH, Kang DH, Kang BS, Hong SK. Lung volumes and ventilatory responses to high CO_2 and low O_2 in the ama. *J Appl Physiol*. 1963; 18: 466-70.
29 Nygren-Bonnier M, Lindholm P, Markstrom A, Skedinger M, Mattsson E, Klefbeck B. Effects of glossopharyngeal pistoning for lung insufflation on vital capacity in healthy women. *Am J Phys Med Rehabil*. 2007; 86: 290-4.
30 Nygren-Bonnier M, Gullstrand L, Klefbeck B, Lindholm P. Effects of glossopharyngeal pistoning for lung insufflation in elite swimmers. *Med Sci Sports Exerc*. 2007; 39: 836-41.
31 Davis FM, Graves MP, Guy HJ, Prisk GK, Tanner TE. Carbon dioxide response and breath-hold times in underwater hockey players. *Undersea Biomed Res*. 1987; 14: 527-34.
32 Binks AP, Vovk A, Ferrigno M, Banzett RB. The air hunger response of four elite breath-hold divers. *Respir Physiol Neurobiol*. 2007; 159: 171-7.
33 Lemaitre F, Polin D, Joulia F, Boutry A, Le Pessot D, et al. Physiological responses to repeated apneas in underwater hockey players and controls. *Undersea Hyperb Med*. 2007; 34: 407-14.
34 Dujic Z, Ivancev V, Heusser K, Dzamonja G, Palada I, et al. Central chemoreflex sensitivity and sympathetic neural outflow in elite breath-hold divers. *J Appl Physiol*. 2008; 104: 205-11.
35 Feiner JR, Bickler PE, Severinghaus JW. Hypoxic ventilatory response predicts the extent of maximal breath-holds in man. *Respir Physiol*. 1995; 100: 213-22.
36 Moosavi SH, Golestanian E, Binks AP, Lansing RW, Brown R, Banzett RB. Hypoxic and hypercapnic drives to breathe generate equivalent levels of air hunger in humans. *J Appl Physiol*. 2003; 94: 141-54.
37 Lindholm P, Lundgren CEG. Alveolar gas composition before and after maximal breath-holds in competitive divers. *Undersea Hyperb Med*. 2006; 33: 463-7.
38 Fitz-Clarke JR. Adverse events in competitive breath-hold diving. *Undersea Hyperb Med*. 2006; 33: 55-62.
39 Krack K, Stepanek M, Cruickshank M. Safety techniques and problem management in recreational and competitive freediving. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving*. Proceedings of the Undersea Hyperbaric Medical Society/Divers Alert Network 2006 June 20-21 Workshop. Durham, NC: Divers Alert Network; 2006. p. 82-95.
40 Edmonds CW, Walker DG. Snorkelling deaths in Australia, 1987-1996. *Med J Aust*. 1999; 171: 591-4.
41 Ridgway L, McFarland K, Stewart IB, Bulmer AC. 'Sambas', 'Mooglies' and other acute effects of apnea. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving*. Proceedings of the Undersea Hyperbaric Medical Society/Divers Alert Network 2006 June 20-21 Workshop. Durham, NC: Divers Alert Network; 2006. p. 39-45.
42 Lindholm P. Loss of motor control and/or loss of consciousness during breath-hold competitions. *Int J Sports Med*. 2007; 28: 295-9.
43 Butler FK. A proposed 60 second limit for breath-hold diving. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving*. Proceedings of the Undersea Hyperbaric Medical Society/Divers Alert Network 2006 June 20-21 Workshop. Durham, NC: Divers Alert Network; 2006. p. 64-74.
44 Vann RD, Freiberger JJ, Caruso JL, Denoble PJ, Pollock NW, et al. *Report on decompression illness, diving fatalities*

and Project Dive Exploration: DAN's annual review of recreational scuba diving injuries and fatalities Based on 2003 data; 2005 edition. Durham, NC: Divers Alert Network; 2005. p. 91-8, 129-31.
45 Vann RD, Freiberger JJ, Caruso JL, Denoble PJ, Pollock NW, et al. *Annual diving report*, 2006 edition. Durham, NC: Divers Alert Network; 2006. p. 59-63, 92-3.
46 Pollock NW. Development of the DAN breath-hold incident database. In: Lindholm P, Pollock NW, Lundgren CEG, eds. *Breath-hold diving*. Proceedings of the Undersea Hyperbaric Medical Society/Divers Alert Network 2006 June 20-21 Workshop. Durham, NC: Divers Alert Network; 2006. p. 46-55.

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Dr Pollock was the Guest Speaker at the 2007 SPUMS Annual Scientific Meeting at Tutukaka, New Zealand.

Oxygen toxicity in recreational and technical diving

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Key words
Oxygen, toxicity, technical diving, mixed gas, review article

Abstract

(Fock A, Millar I. Oxygen toxicity in recreational and technical diving. *Diving and Hyperbaric Medicine*. 2008; 38: 86-90.)
It is increasingly common for recreational scuba divers to use breathing mixtures enriched with additional oxygen ('nitrox' or 'enriched air nitrogen') and for technical divers to be exposed to elevated partial pressures of oxygen for prolonged periods of time. The National Oceanic and Atmospheric Administration oxygen exposure limits have traditionally been used by the recreational diving industry and technical diving communities. Review of the original research into oxygen toxicity brings into question the validity of these limits and would suggest revised limits with a maximum partial pressure of oxygen of 162 kPa (1.6 Ata) and 142 kPa (1.4 Ata) at depth and the use of the repetitive air excursion (REPEX) limits for single and repetitive exposures. Suitable conservatism in case of the need for recompression therapy is recommended.

Introduction

The use of breathing mixtures containing high levels of oxygen ('nitrox', 'enriched air nitrogen' for scuba diving has become routine over the last decade. More recently, the advent of technical diving has seen the use of these mixtures as well as pure oxygen to accelerate decompression. Training agencies for both recreational and technical diving have traditionally used the central nervous system (CNS) limits prescribed by the National Oceanic and Atmospheric Administration (NOAA).[1] These describe a relationship between time and an exposure to a particular partial pressure of oxygen (PPO_2) and are provided for both single exposures and daily exposures. However, with the advent of technical diving, where decompression times may exceed five hours, many divers are routinely exceeding these limits apparently without ill effect. Therefore, it would seem timely to review the origins of these limits as well as newer data on oxygen toxicity more relevant to this style of diving.

Manifestations of oxygen toxicity

At a clinical level, the toxic effects of oxygen are most apparent in the lung, brain and eye. This should not be surprising, given the lung's direct exposure to oxygen, the very high blood flow and vaso-reactivity of the CNS and the unique avascular physico-chemical structure of the lens of the eye. In the lung, oxidative damage results in inflammation, capillary leakiness and ultimately fibrosis. The mechanisms of acute CNS toxicity are extremely complex and incompletely understood. In overview, it is thought that increased reactive oxygen species produced through the metabolism of molecular oxygen cause an imbalance between neurotransmitters, triggering uncoordinated electrical depolarisation (an excellent review is provided by Clark and Thom[2]). This often manifests as loss of consciousness with a grand mal-type convulsion and may commence abruptly, with or without preceding symptoms. The occurrence of such an event whilst diving is likely to be fatal, therefore a good understanding of the CNS oxygen tolerance limits is vital if high PPO_2 is to be