Adverse events in competitive breath-hold diving.

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Fitz-Clarke J. Adverse events in competitive breath-hold diving. Undersea Hyperb Med 2006; 33(1):55-62. Medical observations are reported from an eight-day world championship breath-hold diving competition involving 57 participants. The deepest dive was to 75 metres, and the longest breath-hold time exceeded 9 minutes. There were 35 diving-related adverse events witnessed or reported, including transient loss of motor control due to hypoxia, syncope during ascent, hemoptysis, and pulmonary edema. All events occurred in healthy individuals, and resolved without apparent sequelae. There was no relationship between symptoms and depth. The medical implications of these adverse events are discussed. Despite the inherent risks of the sport, established organizational procedures for competitive breath-hold diving maintain a high degree of safety.

INTRODUCTION

Breath-hold diving as a recreational sport has grown rapidly world-wide, particularly over the past decade. Participants have developed procedures and training techniques that have advanced breath-hold dives to depths over 100 metres, and underwater times over eight minutes (1, 2). Many dedicated individuals attend national and international competitions that are held each year. The competitive side of the sport emphasizes prolonging breath-hold time through relaxation, lung hyperinflation, and economy of swimming technique to achieve a maximum depth, time, or distance underwater.

Breath-hold diving is associated with a variety of physiological responses induced by immersion, apnea, and lung compression (3-5). The most prominent responses are bradycardia, peripheral vasoconstriction, and splenic contraction, all of which help to conserve oxygen during dives (6), and delay the effects of hypoxia and hypercapnia. Effectiveness of these diving reflexes can be enhanced with appropriate training and experience (7).

Any sport that challenges the limits of human performance has inherent risks that are accepted by its participants. Most serious competitive breath-hold divers have had episodes of symptomatic hypoxia or underwater syncope. There is a large body of anecdotal information circulating among enthusiasts that has not caught the attention of the scientific community. Although there has been considerable research into the physiology of breath-hold diving (8-10), there have been no previous descriptions of adverse effects and sequelae of diving competitions, particularly in light of the most recent evolution of the sport.

This report discusses medical issues and observations made at a major international breath-hold diving championship. This information may help organizers anticipate and prepare for adverse events likely to be seen at championship level, and may serve as a resource to physicians responsible for examining divers and assessing fitness for participation.

METHODS

Observations were made at the Fourth AIDA World Free Diving Championship held in Vancouver, Canada over 8 days in August

of 2004. A total of 57 divers from 13 countries participated and were grouped into teams for the purpose of scoring. Ages ranged from 20 to 52 years, with 33 males and 24 females, including several world record holders in various categories. The author was present as event physician. All subjects were in good health according to medical forms completed by home physicians, and confirmed by interviews at registration. Several competitors had documented spirometric tests performed in their home countries. Reports available on four subjects indicated total lung capacities in the range of 7.1 to 10.0 litres, including those of two previously reported individuals (11). Medications being taken on arrival included nasal decongestants, antihistamines, oral contraceptives, thyroid supplement, antidepressants, acetaminophen, and ibuprofen. Medications were restricted prior to competition according to WADA anti-doping regulations (12).

Competition events are summarized in Table 1. All breath-holds were conducted with air. Pre-dive meditation and ritual slow, moderately deep breathing exercises were routinely practised. Although excessive hyperventilation was avoided, many divers engaged in multiple deep exhalations, commonly called "purges", to reduce lung carbon dioxide prior to submergence. The constant-weight event involved two days of unlimited practice off an ocean barge anchored in deep water, followed by two days of formal competition, during which each diver had one unaided swimming attempt to retrieve a tag from the bottom of a cable set to a predetermined depth. Judges monitored competitors for any outward signs of hypoxia occurring within the first 60 seconds after surfacing. Video analysis was available for resolving disputes, as some hypoxic episodes were unrecognized by the divers themselves. Safety breath-hold divers and mixed-gas scuba divers were stationed at intervals in the water column, and stayed close to all competitors underwater. Ocean temperature was 20^{o} C on the surface, with calm conditions. All competitors wore wet suits for ocean dives and many of the pool dives, and used an assortment of dual fins and monofins according to personal preference. Static and dynamic apnea events were held in a 50-metre outdoor pool.

Table 1. Competition Event Categories

Constant Weight	Diver wearing a weight belt and fins swims down to maximum ***depth*** and back on a single breath of air under his or her own power, without any type of assistance.
Dynamic Apnea	Diver with fins swims maximum horizontal ***distance*** underwater on a single breath of air in a swimming pool.
Static Apnea	Diver lies face down in a swimming pool, holding his or her breath for as long a ***time*** as possible. Diver must periodically indicate conscious state by signal to a spotter.

Athletes were examined after all adverse events, except for some cases of brief minor losses of motor control, which did not warrant examination. Symptoms and mental status were assessed, followed by chest auscultation. Twelve subjects were chosen for urine drug tests at the conclusion, in accordance with anti-doping regulations (12).

RESULTS

Although many of the divers had previously achieved greater depths and durations, initial performances were generally more conservative than world record marks, so as to minimize the risk of disqualification due to potential hypoxic symptoms on overly aggressive dives. Almost all athletes employed "lung packing" in the water prior to

submersion. This is an inspiratory technique for hyperinflating the lungs using the pharyngeal and glottic muscles in a repetitive manoeuvre resembling gulping or swallowing (13, 14). Only one competitor did not pack due to a past tendency to develop hypotension from increased intrathoracic pressure impeding venous return, and a self-reported perceived lack of benefit from the technique.

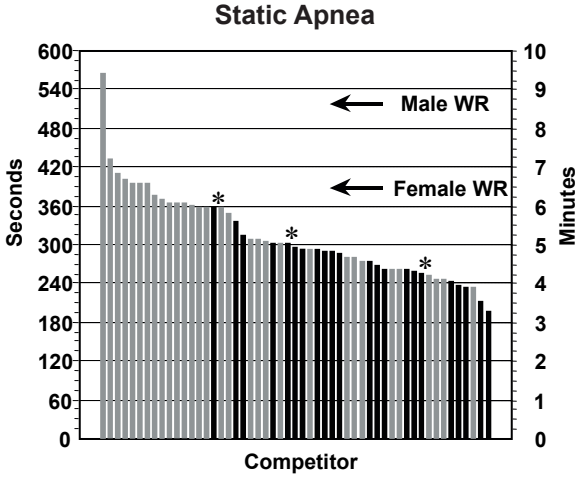

Fig. 1. Breath-hold times in the static apnea event for 53 attempts by males (grey) and females (black). The three attempts denoted by asterisks were associated with hypoxic loss of motor control, resulting in disqualification. World record (WR) times are shown.

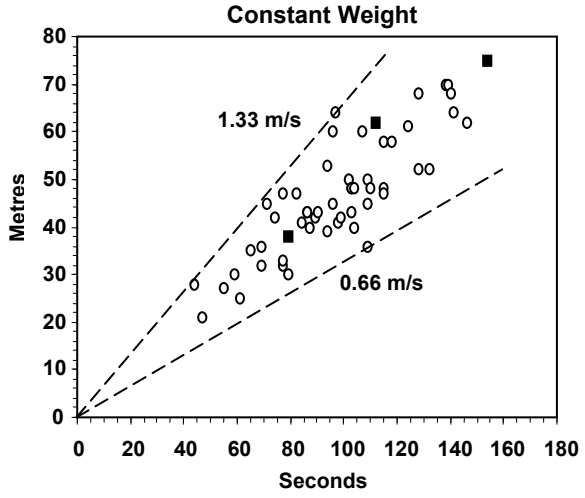

Fig. 2. Total dive times, including descent and ascent, and maximum depths achieved in the constant weight event for 55 divers. Episodes of hypoxic syncope are denoted by the three solid squares. Dashed lines indicate the range of swimming velocities.

Breath-hold times of 4 to 7 minutes were common in the static apnea event, as summarized in Figure 1. The longest was an immersed breath-hold in the pool for 9 minutes and 24 seconds, an unofficial world record (WR). Underwater times were much shorter in the constant weight and dynamic apnea events due to the exertion of fin kicking. These times ranged from 1 to 3 minutes, as shown in Figure 2. The slope of a line from the origin to any point equals one half of the average swimming velocity for that dive because the time includes both descent and the ascent. Velocities ranged from 0.66 to 1.33 metres per second, as indicated by the dashed lines.

Maximum depth achieved in the constant weight event was 75 metres, which resulted in loss of consciousness during the ascent. The deepest successful dive was 70 metres, considerably short of the most recent world record of 102 metres. The longest dynamic apnea was a 180-metre horizontal swim underwater.

Table 2. Adverse Events on Each of Eight Days

	Ocean	Pool	Total
Hemoptysis	3, 1, 0, 0	0, 0, 0, 0	4
Pulmonary Edema	1, 0, 0, 1	0, 0, 0, 0	2
Ascent Syncope	0, 1, 0, 2	0, 0, 0, 0	3
Laryngospasm	0, 0, 0, 0	0, 0, 0, 1	1
LMC	2, 2, 1, 1	2, 3, 4, 10	25

All divers exited the water unassisted, and those experiencing symptoms were promptly assessed. Adverse events are summarized in Table 2, including incidents during both practice and competition dives. It is not possible to express risks as percentage statistics because the total number of practice dives is not known. There were no serious or persistent adverse events among athletes, and no incidents affecting safety divers. Episodes of loss of motor control (LMC) due to hypoxia,

commonly called "sambas", were frequent, with a total of 25 occurring during both official and practice dives. LMC is defined as the presence of hypoxic signs first appearing after surfacing, without complete loss of consciousness. It is recognized by judges as a failure to demonstrate full control and awareness. Most episodes consisted of illegal head movements, partial facial immersion, abnormal eye movement or closure, or blank staring. There were no cases of myoclonus or seizures. All divers with LMCs recovered rapidly, usually within seconds on the surface. Three divers lost consciousness underwater during ascents from depths between 38 and 75 metres. All three cases of ascent syncope occurred near the surface, and were managed by safety divers who protected the airways. Divers experiencing syncope or prolonged LMCs were given oxygen by mask for 5 minutes as a precaution. Full recovery was rapid in all cases, however no further diving was allowed for that day. There was no relationship between incidence of syncope and depth or duration of dives.

Four divers experienced mild hemoptysis, characterized by expectoration of one or more boluses of bright red blood. Three episodes occurred on the first ocean day, and were seen after dives to depths between 45 and 60 metres. Hemoptysis did not occur in subjects who achieved the deepest dives. Three other subjects reported a past history of similar episodes. All four cases were associated with upper anterior chest or neck discomfort, mild transient cough, with no wheezing or dyspnea. It was ascertained that hemoptysis was not due to nosebleed, respiratory infection, or sinus barotrauma. All affected individuals felt certain that the blood was from the respiratory tract. Each had employed lung packing prior to submersion. Lungs were clear to auscultation.

Another subject, very fit and 26 years of age, developed a sensation of deep wet chest congestion immediately after a dive to 60 metres, and felt it continue to develop on the surface and on the barge, without coughing or hemoptysis. Auscultation confirmed bilateral basal rales, suggestive of pulmonary edema. Blood pressure was normal. This episode resolved after about an hour, but recurred three days later in competition to a greater depth. There was no on-site access to radiography to determine the nature of the rales or hemoptysis in these divers. Further investigations were not deemed practical, since symptoms resolved completely. One case of aspiration-induced laryngospasm occurred in the static apnea event, characterized by inspiratory stridor with no expiratory wheeze. This resolved completely over about 10 minutes, without sequelae.

DISCUSSION

Although much of the basic physiology of breath-hold diving is understood in terms of cardiovascular and respiratory responses (8-10), reports of adverse events in competitive diving have not previously been published. The competition was run with excellent attention to safety, and there were no incidents that could not be managed on site. A total of 35 adverse events were witnessed, all of which recovered completely with no apparent sequelae. The number of adverse events may have been higher during practice dives if some individuals were reluctant to report complications. All competition dives, however, were carefully observed by judges for one minute after surfacing, so non-witnessed events were unlikely to have occurred on those days. Adverse events, as presented above, are considered by the athletes to be within the acceptable risk of the sport at the competitive level.

Cases of hemoptysis after breath-hold dives have been reported (15), some of which have been associated with aspirin ingestion (16), and involuntary diaphragmatic

contractions brought on by prolonged apnea (17). Most divers interviewed commonly experience diaphragmatic contractions beginning a few minutes into static apnea. The latency of contraction onset has been shown to correlate with breath-holding ability (18). Some competitors, who had previously experienced hemoptysis after free dives, had been investigated in the past with chest radiographs, computed tomography, and bronchoscopy with no findings. These episodes are generally assumed to be minor, since there is complete resolution, but no information is available on possible long-term effects. Investigation of isolated cases of minor post-dive hemoptysis is probably not warranted in otherwise healthy non-smokers.

The etiology of post-dive hemoptysis remains unclear, since it has yet to be directly observed with bronchoscopy. Using direct laryngoscopy, Lindholm et. al. (19) observed blood below the vocal cords in 2 of 11 divers after simulating deep dives by exhaling to near residual volume, and descending to 6 metres. Hemoptysis might be a form of pulmonary capillary stress failure (20, 21), but given the location of discomfort in the upper chest and neck regions, brightness of expectorated blood, and absence of froth, at least some cases likely originated from the tracheal or bronchial circulation (22), rather than from the alveoli. There has been only one report in the literature of diving-related thoracic squeeze associated conclusively with hemorrhagic lungs at the alveolar level (23). In one other case, radiographic changes were seen that were suggestive of alveolar blood following a breath-hold dive (16). The depth at which lung damage might occur was once believed to be determined by the ratio of total lung capacity (TLC) to residual volume (RV). Experience does not support this assumption, as many breath-hold divers have been much deeper than this theoretical prediction, without any apparent problems. Lung air spaces can be compressed far below RV owing to chest and diaphragm elasticity, and the high distensibility of the pulmonary vasculature, which engorges to compensate for air volume changes from compression (24).

It is not clear why some divers can reach great depths without complications, while others develop hemoptysis after relatively shallow dives. Anecdotally, many divers believe that experience and gradual depth progression of training dives are important in preventing hemoptysis. Exertion and rapid rate of descent may be predisposing factors, but have not been specifically studied. It is conceivable that rapid descents might damage capillary endothelium, while slower descents might promote leakage of plasma and blood-derived proteins into the alveolar space, which could dilute the surfactant layer and increase surface tension. This might aid capillary transmural reinforcement, thus reducing the likelihood of capillary rupture (25). Anatomical variants in the bronchial circulation might play a role if specific vessels are comparatively weak (26). Airway mucosal blood flow increases with negative intrathoracic pressure during a Müller manoeuvre (27), similar to that occurring at depth, where inadequate chest compression creates an intrathoracic pressure below that of the rest of the body, redistributing blood from the periphery into the pulmonary circulation (28). Cold water may have contributed to hemoptysis through a more pronounced early pressor effect in unacclimatized individuals from warmer regions. Peripheral vasoconstriction contributes to a larger thoracic blood shift from the lower body (29), which is likely associated with greater distension and stress of capillaries in the submucosal plexus of the tracheal and bronchial circulations. Co-existing high arterial pressures might additionally contribute to capillary failure. Systolic blood pressures up to 290 mm Hg have been documented in

two healthy subjects during simulated breath-hold dives in a hyperbaric chamber (30). Based on reported symptoms, auscultation of rales, and absence of hemoptysis, it is reasonable to assume that the subject experiencing post-dive chest congestion had mild pulmonary edema. Radiography was not available at the time for more objective confirmation. Pulmonary edema has been known to occur in cold water scuba divers (31-33) and surface swimmers (34), but has rarely been reported in breath-hold divers.

Breath-hold times of 5 to 9 minutes on air can be achieved without harm so long as oxygen from the lungs continues to circulate to the brain. This situation differs from that of cardiac arrest, where tissues are disconnected from lung oxygen stores, and brain anoxia causes damage after a few minutes. Long periods of voluntary apnea require great tolerance to carbon dioxide and suppression of the urge to breathe, despite progressive hypercapnia, can be trained. The time to hypoxic syncope in apnea is primarily a function of initial lung volume and the rate of oxygen consumption (24), the latter of which is influenced by exertion and minimized by relaxation. Progressive hypercapnia increases cerebral blood flow and oxygen delivery, which may help prolong the time to loss of consciousness (35).

While episodes of symptomatic hypoxia resulted in disqualification for the specific event, they were not an apparent health concern to the athletes. Many competitive breath-hold divers have experienced loss of consciousness or other 'sambas' in the past, and consider them to be a normal acceptable part of the sport's risk. The acute effects of hypoxic blackout in diving appear to be similar to those of other forms of transient syncope that are followed by rapid spontaneous recovery, including vasovagal and orthostatic hypotension (36), acute high-altitude exposure (37), and G-induced loss of consciousness in aviators (38). While there is presently no evidence that brief or even recurrent episodes of syncope are associated with any persistent neurological or cognitive deficits (39-42), this possibility cannot be ruled out, particularly given the frequency with which some individuals intentionally expose themselves to hypoxia during training. Neurological manifestations of decompression illness (43, 44) and MRI documented cerebral infarction (45) have also been reported following repetitive breath-hold dives. Magno et al. reported a case of hemiplegia occurring after a single assisted dive to 120 metres (46). Drowning is the greatest concern with syncope in unsupervised aquatic settings, but is easily averted by trained and attentive safety divers.

In summary, competitive breath-hold diving is associated with some unique alterations of human physiology. Episodes of symptomatic hypoxia are common in competition, but usually resolve quickly without apparent sequelae. Brief hypoxic loss of motor control on the surface was the most common adverse event, followed by minor hemoptysis and underwater syncope during ascent. There was no prior indication as to which individuals would be most susceptible to hypoxia or any relationship between symptoms and diving depth. Established regulations and organizational procedures appear to ensure a high degree of safety in diving competition.

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