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Adverse Events in Competitive Freediving – Clinical Presentation, Management, and Prevention

Zwischenfälle beim Wettkampfapnoetauchen – Symptome, Management und Prävention

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Summary

› **Freediving** (synonyms: breath-hold diving, apnea diving) has become a popular leisure activity and continues to gain popularity and recognition as both a competitive and recreational sport. Formal freediving competitions are sanctioned by the international Confédération Mondiale des Activités Subaquatiques (CMAS) or the Association Internationale pour le Développement de l'Apnée (AIDA). Both organizations have set up rules and guidelines for competitive pool disciplines where athletes compete for maximum submerged breath-hold time or underwater swimming distance with or without fins.

› **In freshwater disciplines** athletes strive for maximum depth using different freediving techniques. These aquatic breath-holding activities carry unique medical risks that are related to environmental factors eliciting extreme physiological challenges. Pool disciplines carry an increased risk of hypoxemia and, consequently, loss of consciousness, particularly in untrained individuals. Hypoxic complications are reported to occur in up to 10% of dives during freediving competitions. Shallow water blackout following hyperventilation to extend breath-hold capability is a serious risk applying to all aquatic breath-hold activities, however, is more common during recreational freediving.

› **Deep freediving** poses the athlete to further risks such as barotrauma of ear drums or lungs, immersion pulmonary edema, nitrogen narcosis, and decompression sickness when reaching great depths. While serious complications in competitive freediving are rare, however, the risk clearly rises with increasing depth. Special breathing techniques to increase lung volumes such as glossopharyngeal insufflation carry additional risks.

› **This article reviews** possible complications and injury that may occur in competitive freedivers and discusses strategies for management and prevention of possible injury.

KEY WORDS:
Breath-Holding, Hypoxia, Barotrauma, Decompression Sickness, Immersion Pulmonary Edema

Introduction

Freediving (synonyms: breath-hold diving, apnea diving) continues to gain popularity and recognition as both a competitive and recreational sport. The Confédération Mondiale des Activités Subaquatiques (English: World Underwater Federation, CMAS), founded in 1959, ratified most of the early achievements made by recreational freedivers. In 1976 French diver Jacques Mayol was the first to reach the 100m depth mark. In 1992 the Association Internationale pour le Développement de l'Apnée (International Association for the Development of Apnea, AIDA) was created and formulated rules and guidelines for competitions and record attempts, while CMAS resumed ratifying records in 1995 due to an ever-increasing popularity of recreational freediving. While prevalence data on overall recreational freediving are not available, data from participation in AIDA competitions in 2023 revealed an all-time high of 2979 athletes.

The continued progression of world records since the inaugural competitions is remarkable, particularly given the extreme hypoxia and hydrostatic pressures these athletes endure. The current world record in static breath-holding, where the subject is floating motionless on the water surface with the face submerged, has been set at 11min 35sec in 2009 (ratified by AIDA). In dynamic disciplines, however, where athletes compete for distance in underwater swimming on a single breath, the records continue to improve, with the current world records using fins or no fins set at 321.43m in 2022 and 238m in 2024, respectively. By contrast depth disciplines request athletes to dive as deep as possible using constant weights with or without fins; there are also sled-assisted disciplines that allow athletes to descend more quickly. As the latter disciplines involve greater achievable depths and, therefore, risks, sled dives are not allowed in competitions. The former sled disci-

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pline “No limits”, with the diver being pulled down by a sled and ascending with the assistance of a balloon, has been abandoned in 2019 due to an increasing number of divers suffering serious injury or death during record attempts. The depth record in this discipline of 214m seawater had been set in 2007 by Austrian Herbert Nitsch (43), who suffered a serious stroke-like syndrome in 2012 when attempting to surpass his own depth record.

Competitive freediving carries unique medical risks that are due to the extreme environmental conditions when the body is submerged under water. Lack of oxygen, hydrostatic pressure, and altered physical properties of water will stress the submersed athlete, be it a competitive freediver, underwater hockey or rugby player, or synchronized swimmer. Competitive freedivers, however, face extreme hypoxia and hypercapnia when competing for maximum breath-hold duration or maximum distance swimming under water. Deep competitive freedives will pose additional risks that are associated with rapidly changing ambient pressure.

Accordingly, medical complications may arise from preexisting health conditions, pressure equalization issues, hypoxic blackout, thoracic blood pooling and lung squeeze, nitrogen narcosis or decompression illness. Hazards may also arise from certain techniques and maladaptations to extreme freediving, such as glossopharyngeal breathing, and rapid descent to and ascent from great depths. This article will focus on epidemiology and clinical presentation of medical complications in competitive freediving and will discuss strategies for management and prevention of possible injury.

Epidemiology of Freediving Incidents

The exact incidence of adverse events in competitive freediving is unknown. Divers Alert Network (DAN), a nonprofit organization providing expert medical information for the benefit of the diving public, collects incidents reported during breath-hold diving activity (36). However, most of those incidents are fatalities with the majority having been reported during recreational snorkeling. Likewise, data on breath-hold diving fatalities from Queensland, Australia, show most cases related to recreational snorkeling or breath-holding activities (25). Remarkably, a substantial increase in snorkeling fatalities in Queensland was seen over the past two decades which has been attributed to an increased number of participants, their higher ages and poorer health. Estimates of the incidence of adverse events in depth competitions (in most cases, mild blackout and mild pulmonary barotrauma) range between 3-4%, indicating that three to four out of 100 dives may result in freediving incidents (27).

More detailed data are available from systematic observations of freediving competitions. However, methods of adverse event reporting differ greatly between publications and reported incidences may not accurately reflect competitive freediving in general. Observations were made at the 4th AIDA World Free Diving Championship held in Vancouver, Canada in August 2006 (12). A total of 57 divers participated and adverse events occurring after training dives or during competition dives and were observed by a physician. There was a total of 35 adverse events with episodes of loss of motor control due to hypoxia being most common. Three divers lost consciousness under water during ascents from depths between 38 and 75 meters. Four divers presented with hemoptysis and chest discomfort and cough, and one subject showed signs of pulmonary edema upon auscultation that recurred during another competition dive three days later. Another study reported data on loss of motor control and/or loss of consciousness from collections of the official results of the major international competitions for national teams organized by AIDA in between 1998 and 2004 (22). This study revealed that an average of 9.7-11.1% of performances in international freediving competitions were disqualified due to signs attributable to severe brain hypoxia. For the competitions in 2002-2004, a distinction was made in the rules between loss of consciousness and loss of motor control, demonstrating an 1.1% and 9.6% incidence in loss of consciousness and loss of motor control, respectively, during static apnea performances. In the constant weight discipline where athletes compete for maximum depth, frequencies of loss of consciousness and loss of motor control were 6.1% and 6.1%, respectively. Another questionnaire study found a high self-reported prevalence of respiratory symptoms in 212 freediving instructors (10). Fifty-six subjects (26.4%) reported previous events such as cough, thoracic constraint, and hemoptysis, associated with various degrees of dyspnea.

Shallow Water Blackout

Clinical signs of hypoxemia during freediving activities range from temporary confusion, speech problems, spasms, transient non-responsiveness to cognitive deficits and impairment (12, 22). Due to strict safety rules and standards applied during competitions held by AIDA or CMAS, in most cases symptoms are mild and transient, leading to disqualification of the record attempt. However, risk of hypoxemia will dramatically increase without supervision and safety measures in place and can obviously become life-threatening in the underwater environment where unconsciousness eventually results in drowning. Development of critical hypoxemia during underwater freediving usually is prevented by an urgency to breathe, the breath-hold breaking point, where arterial partial pressure of CO_2 (P_aCO_2) exceeds approximately 45-55 mmHg and involuntary inspiratory efforts such as contractions of the thorax and the diaphragm occur. However, hyperventilation before the dive will manipulate the time-course of CO_2 increase during the breath-hold and, thus, prolong breath-hold time. Shallow-water blackout may result from cerebral hypoxia towards the end of a breath-hold dive in shallow water when arterial partial pressure of O_2 (P_aO_2) falls below the critical level necessary to maintain consciousness, as P_aCO_2 rises from a substantially lower than normal level, and the onset of central unconsciousness prevents a response to the respiratory stimulus (figure 1).

Critical hypoxemia with loss of consciousness may likewise occur during ascent from a deep breath-hold dive. Subjective assessment of time needed to return safely to surface may be confounded by a high P_aO_2 at depth, and relatively low P_aCO_2, especially when P_aCO_2 rose from a lower than normal level following hyperventilation. Upon ascent P_aO_2 will decline progressively and fall below the critical level necessary to maintain consciousness. It should be noted that blackout can occur upon ascent while the diver is surfacing or even after surfacing and can still cause drowning if not immediately attended by safety divers.

Barotrauma During Descent

Due to the inverse relationship between ambient pressure and gas volume at constant temperature, air filled body cavities are subject to decreasing air volume during descent as hydrostatic pressure increases. This may concern the eyes if wearing goggles or a face mask, the middle ear, the outer ear when wearing a hood, and paranasal sinuses. Risk of barotrauma is not ›

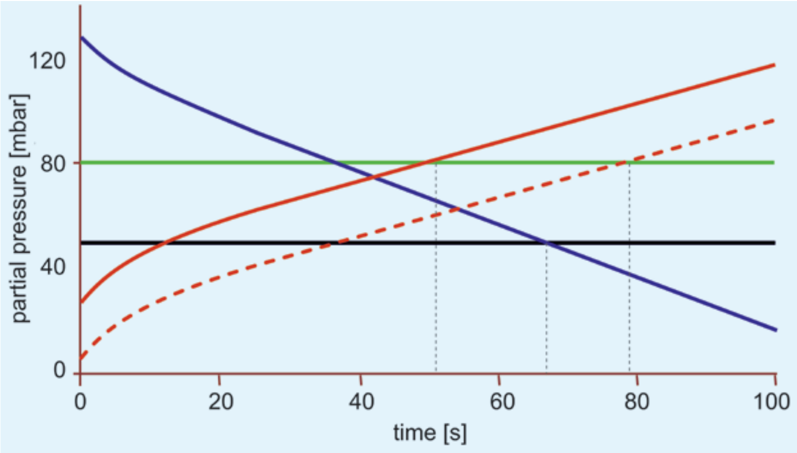

Figure 1

Blood gases during breath-holding with and without preceeding hyperventilation. When breath-holding following tidal breathing arterial oxygen pressure will drop continuously over time (solid blue line; O_2), while arterial carbon dioxide pressure steadily increases (solid red line; CO_2) from baseline. Hyperventilation will lower carbon dioxide baseline and, thus, postpone increase in arterial carbon dioxide pressure (dashed red line; CO_2 after hyperventilation). Blackout may occur when hypoxia leads to cerebral unconsciousness (solid black line; blackout level) before an increasing carbon dioxide level will ultimately trigger an urge to breathe (solid green line; respiratory stimulus threshold).

unique for freediving and applies to all diving. However, due to speed of descent and breath-holding, frequent and effective pressure equalization is mandatory when performing competitive deep freedives.

When the volume of air in the space between the conjunctivae and mask or goggles shrinks, eye barotrauma may occur if no air is put into the mask during descent, resulting in trauma of capillaries in the conjunctiva. This can be avoided by remembering to equalize pressure in the mask frequently. Decreasing air volume in the middle ear will bulge the eardrum inwards; leakage of fluid and bleeding of ruptured vessels may occur if pressure is not equalized frequently during descent. The Eustachian tubes connecting the throat with the tympanic cavity normally provide passage for air when pressure equalization is needed. Maneuvers such as swallowing or yawning can facilitate opening of the tube. Of note, forceful equalization under these conditions can increase the pressure differential between the inner ear and the middle ear, resulting in round window rupture with perilymph leakage and inner ear damage. Hood squeeze may occur if there is air trapped between the outer ear and the hood and decreasing air volume may pull on the ear drum. Putting water inside the hood can prevent outer ear barotrauma. Finally, congestion in the paranasal sinuses may hinder air to travel freely through them because of their relatively narrow connecting passageways. With small changes in depth, symptoms are usually mild and subacute but can be exacerbated upon continued descent. Larger pressure changes can be more injurious, especially with forceful attempts at equilibration exposing the diver at risk of sinus barotrauma causing pain and bleeding.

Pulmonary barotrauma may occur during descent when total intrapulmonary gas volume is compressed below residual volume and a critical negative intraalveolar pressure is reached, causing capillary rupture and fluid exudation into the alveolar space. It was previously assumed that pulmonary barotrauma of descent is a rare event and may only occur when reaching great depths (40); however, an increasing number of reports of respiratory symptoms with hemorrhage occurring also at shallower depths led to an acknowledgement of pulmonary edema with or without barotrauma as a more frequent risk in freediving (28).

Pulmonary Edema / Hemoptysis

Immersion in water induces an increase in intrathoracic blood volume and pulmonary capillary pressure that may be further enhanced by peripheral vasoconstriction induced by the diving response and a cold environment. Additional stressors may be present during deep freedives such as exercise, negative intrapulmonary pressure that is caused by involuntary breathing movements during the late phase of a breath-hold dive just when reaching the breath-hold breaking point, and/or negative pressure precipitated by lung volume compression (figure 2).

Accordingly, cases of hemoptysis have been reported during underwater breath-hold dives ranging between 25-35 meters of seawater (6, 16, 17, 32). Common symptoms include dyspnea, cough, expectoration of frothy sputum and hypoxemia. Auscultation may reveal bibasilar rales, and imaging may show patchy infiltrates on chest x-ray of CT. Other symptoms, e.g., presyncope may occur simultaneously during ascent as P_aO_2 falls rapidly with the decline in ambient pressure (33). It has been suggested that voluntary diaphragmatic contractions could be the main contributing factor for the alveolar hemorrhage occurring in freediving spearfishers at shallow depths, in addition to relatively low water temperature, exercise, and immersion (17). Interestingly, cases of hemoptysis have also been reported following underwater hockey, and it was postulated that combination of the hemodynamic effects of strenuous exertion, submersion, and diaphragmatic contractions on the pulmonary capillaries was contributory to capillary stress failure (2). All these mechanisms occur simultaneously in underwater hockey, as this is the case in freediving spearfishers. In an experimental study 11 competitive freedivers performed breath-hold dives to a depth of six meters after complete exspiration to residual volume, precipitating negative pressure stress on pulmonary capillaries (21). Dynamic ventilatory volumes were found to be reduced after the dive, and laryngoscopy revealed fresh blood in two divers, indicating thoracic squeeze trauma. Another study measured spirometry in 19 competitive athletes competing in dynamic apnea and deep dives during an international freediving competition (23). After deep dives (25-75 m), 12 of the divers had signs of pulmonary edema. None had any symptoms or signs after shallow pool dives. Six freedivers experienced significant respiratory symptoms after diving to depths between 41 and 75m (mean 61m). In this subgroup with typical symptoms and signs of pulmonary edema, changes in the physiological measurements were aggravated compared with in the whole group of divers. From these studies it can be concluded that competitive freedivers, when diving to deep depths, are at risk of developing a depth-dependent pulmonary edema or even frank alveolar hemorrhage.

However, subclinical interstitial pulmonary edema may be more commonly associated with under water breath-holding activity than previously believed, since extravascular lung water has been observed using ultrasound B-line counts (35) in a variety of freediving activities (7, 18). B-lines indicating extravascular lung water have been described to increase after breath-hold diving and disappear within 24 hours (13). Ultrasound lung B-lines correlate with diving depth, confirming that extra-vascular lung water increases with deeper dives (34), however, may also be present in some subjects after shallow water dynamic freedives or shallow water spearfishing activities (7). Thus, the presence of extravascular lung water may depend on several factors, with depth and effort possibly being the most prominent (30, 34). In fact, non-maximal shallow water freedives were not related to an increase in ultrasonic evidence of extravascular lung water (30).

Nitrogen Narcosis

Symptoms of inert gas narcosis such as fatigue, impaired cognition and dexterity, and amnesia have been reported after deep freedives, similar to nitrogen narcosis when breathing compressed air at depth (33). Reports from competitive athletes and monitored freedives indicate that signs of nitrogen narcosis are correlated with depth and time at depth, and symptoms including amnesia are reversible within 30 min after surfacing (33). It is believed that narcosis is mainly due to diffusion of nitrogen molecules into body tissues altering the equilibrium between open and closed states of various neurotransmitter receptors. In contrast of breathing air at depth, however, the amount of nitrogen molecules is limited to the volume of air in the lungs before submersion. It has been speculated that narcosis in freedivers differs from classical nitrogen narcosis in compressed air divers and that raised carbon dioxide production, placing the freediver into a state of hypercapnic hypoxia may play a distinct role impairing cerebral function by increasing blood flow (20). Of note, increasing total lung volume by glossopharyngeal breathing will enhance the total amount of nitrogen and eventually increase risk of narcosis.

Decompression Illness

Deep freediving is associated with risk of decompression illness, an umbrella term encompassing diseases caused by gas bubbles in blood or tissue during or after a reduction in ambient pressure. Gas may evolve from nitrogen gas microbubbles that are formed when critical tissue supersaturation is exceeded during rapid ascent from depth and cause tissue and vascular injury (decompression sickness). Gas may also result from pulmonary barotrauma and creep into tissue or blood, depending on the location of lung tissue rupture. If introduced into the arterial circulation, gas may cause arterial gas embolism with multifocal ischemia (29).

Decompression Sickness

Occurrence of decompression sickness (DCS) while freediving is uncommon. Unlike when breathing compressed air at depth, nitrogen load associated with single breath-hold dives usually is insufficient to provoke DCS. However, repeated deep breath-hold dives with short surface intervals between dives may accumulate nitrogen in body tissues leading to supersaturation and cause symptoms of DCS (21, 39). A recent systematic literature review concluded that breath-hold divers can suffer neurological symptoms after diving; however, to what extent this is attributable to DCS or arterial gas embolism is not completely understood (5). Evidence of classical DCS symptoms such as pain, skin rash, numbness, tingling, loss of consciousness, fatigue, headaches, paralysis, visual disturbances, mood swings, nausea, vertigo, tinnitus, and loss of hearing in competitive freedivers after single deep dives is mostly anecdotal (see diary excerpt from a world-cham- ›

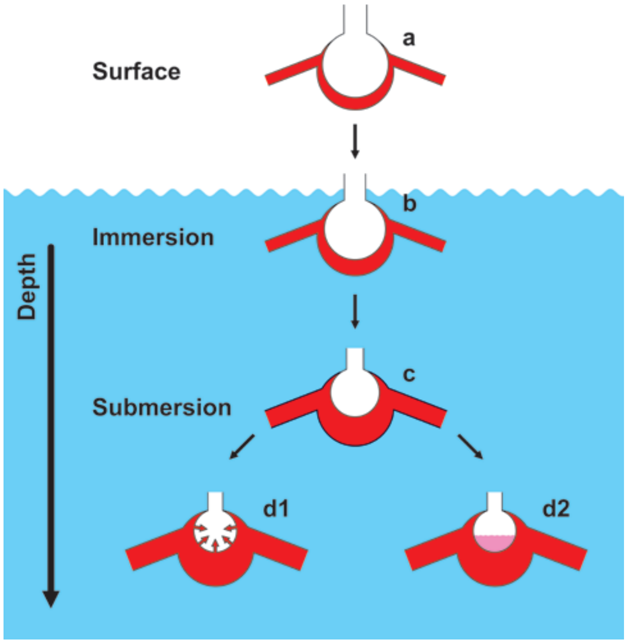

Figure 2

Immersion-related blood shift and possible pulmonary complications. Physiology of the alveoli of the lung and the pulmonary blood vessels at surface (a) will be altered by immersion (b) to the neck generating negative intrathoracic pressure and, thus, increased venous return to the pulmonary vessels. Submersion while breath-holding (c) will lead to compression of alveoli due to Boyle's law and a further redistribution of blood into the thoracic vessels, leading to a engorgement of these vessels. At greater depth this mechanism may lead either to a rupture of blood vessels due to overdistension (d1), or to an intraalveolar edema (d2) due to high hydrostatic pressure.

Box 1

Diary excerpt from a world-champion competitive freediver during training, 2022.

„Yesterday was our last deep session at home before leaving for Cas, Turkey. North wind brought a 2 degree drop in water temperature. But the same wind brought a flat sea, comfy for dives. I set a modestly challenging for me depth, a 100 m with monofin. Tania insisted on bringing the oxygen tank from inside the boat to the deck. I was viewing it as an over'precaution, but she was right and I was wrong.

So I did 99.9 meters (yep, nice precision in setting the bottom plate), and again, only 3 minutes after surfacing, swiftly appeared heavy symptoms of DCS: right half of the face paralyzed and speech impaired.

This time I went into the decom pression dive quickly and, I may say, routinely. Five minutes into the dive all symptoms faded away. I was rather bewildered. It seems we learned all the lessons from the previous DCS case, and here it comes again. So the same day I went to the clinic for blood tests.

(note: tests ended up being ok) I felt sorry for not having done it after the first DCS episode. And it completely changes my attitude towards oxygen. We are thinking about buying an oxygen boosting compressor to increase the carbon tank pressure to 300 atm."

pion freediver in the box (box 1)) and only few case reports have been published (5). In contrast to DCS associated with compressed air diving, however, DCS in freedivers involves frequently the central nervous system featuring stroke-like symptoms (26, 38).

Risk of DCS can be minimized, e.g., by avoiding deep breath-hold dives (>100 meters) and observing adequate surface intervals between dives when performing repeated dives. Oxygen should be breathed after deep freedives for at least ten minutes to help eliminate nitrogen from body tissues. Adequate hydration before and after diving is as crucial as it is for compressed air diving.

Pulmonary Barotrauma of Ascent

Pulmonary barotrauma can be a serious complication of compressed air diving if the diver does not exhale properly and lung volume increases during ascent, and overdistention of alveoli and bronchi may lead to tissue rupture. Pulmonary barotrauma may also occur during freediving, although the risk of lung over-inflation is negligible since the diver does not add any gas to the intrapulmonary gas volume at depth. If the freediver is breathing from a compressed air tank at depth, however, risk of pulmonary barotrauma clearly increases (3). There have been several reports of pneumomediastinum with retrosternal pain and coughing, pneumothorax, and/or neurological symptoms that occurred after breath-hold dives without use of any breathing equipment (1, 4, 14, 36, 40), indicating that freediving is associated with risk of pulmonary barotrauma. It is assumed that during ascent, in particular during deep freedives that involve lung compression and atelectasis formation, overexpansion of regional lung segments during ascent may cause trauma when other parts of the lung do not open up simultaneously. Experimental simulation of lung volumes below residual volume demonstrated regional bronchial collapse in competitive freedivers in magnetic resonance imaging studies with hyperpolarized gas (31).

Complications of Glossopharyngeal Inhalation

Competitive freedivers use glossopharyngeal breathing (commonly called “lung packing”) to increase their performance with regards to duration of breath-holding, attenuation of the consequences of increasing pressure on the chest, and facilitation of pressure equalization in the ear during descent toward the bottom. Glossopharyngeal inhalation increases the volume of air in the lungs above the total lung capacity and increases intra- and transpulmonary pressures (42). Thus, lung injury may occur in competitive freedivers following glossopharyngeal inhalation. In fact, cases of clinically asymptomatic radiologically detectable pneumomediastinum have been reported and indicated that lung barotrauma may be a common occurrence in freedivers (9, 15). Even neurological complications due to arterial gas embolism may follow lung injury after glossopharyngeal inhalation (24, 38). Substantial hemodynamic changes with dramatic decreases in blood pressure induced by lung packing have been described that were related to the amount of additional gas insufflated (8, 11).

Conclusions

Competitive freediving elicits extreme cardiovascular and respiratory responses to both exercise and asphyxia during submerged breath-holding, which alone or in combination with a rapid sequence of compression and decompression challenge human physiology. Serious injury may result when physiologic limits are being surpassed. Hypoxic loss of motor control and unconsciousness are major risks during shallow water competitions, while risks of pulmonary edema and barotrauma, nitrogen narcosis, and decompression sickness rise with increasing depth. To minimize risks during shallow water freediving competitions, application of and adherence to strict safety measures as mandated by CMAS or AIDA is paramount. Special breathing techniques to increase breath-hold performance, such as hyperventilation to induce hypocapnia, or glossopharyngeal insufflation to increase oxygen storage, should be strictly avoided. Because of the inherent risks that will increase with depth, deep freedives beyond 30m can only be supported from a medical perspective in experienced and healthy athletes.

Conflict of Interest
The authors have no conflict of interest.

Summary Box

- Competitive freediving has gained popularity as an extreme aquatic sport. Athletes strive for maximum underwater distance or depth while holding their breath. Complications during competitive performance and training may arise from the extreme physiological challenges elicited by submersion, apnea, exercise, and changing pressure when diving deep.

- This article reviews current epidemiology and clinical presentation of freediving complications, including more recent data on hypoxic blackout and pulmonary edema. An emphasis is provided on the increase in risk for decompression issues and narcosis when going to great depths, which has only been recognized more recently in breath-hold diving.

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